



The Rionero's special type of Lyapunov function and its application to a diffusive epidemic model with information

Bruno Buonomo¹ · Alberto d'Onofrio²

Received: 21 June 2023 / Accepted: 19 July 2023 / Published online: 22 August 2023
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Abstract

We consider a SIR-like reaction-diffusion epidemic model which embeds opinion-driven human behavioural changes. We assume that the contagion rate is theoretically saturated with respect to the density of the disease prevalence. The model extends the general reaction-diffusion epidemic model proposed in 1993 by Capasso and Di Liddo. We study the nonlinear attractivity of the endemic steady state solution by employing a special Lyapunov function introduced in 2006 by S. Rionero. Sufficient conditions for the conditional nonlinear stability of the endemic equilibrium are derived.

Keywords Epidemic model · diffusion · Human behaviour · Information · Lyapunov function

Mathematics Subject Classification 92D30 · 34C60

1 Introduction

The stability analysis of equilibria of dynamical systems modelling biological phenomena is always mapped in a major issue: the assessment of the robustness of the biological equilibrium. The semantic of such stability sometime is positive (e.g., Is the pest *eradication* stable w.r.t. possible pest importation?) other times it is nega-

This paper is dedicated to the memory of professor Salvatore Rionero, Scientist and Master of great passion and humanity.

✉ Bruno Buonomo
buonomo@unina.it

Alberto d'Onofrio
alberto.donofrio@units.it

¹ Department of Mathematics and Applications, University of Naples Federico II, via Cintia, 80126 Naples, Italy

² Department of Mathematics and Geosciences, University of Trieste, Via Alfonso Valerio 12/1, 34127 Trieste, Italy

time (e.g., Is the *invasion* equilibrium robust?). An important biological application of dynamical systems is the area of modelling the spread and the control of infectious diseases, i.e. the *Mathematical Epidemiology* (ME) [7, 15]. Although the vast majority of mathematical studies in the area of ME focuses on purely temporal models, a number of significant studies have been devoted to spatio-temporal models [1–3, 7, 14, 15, 20, 23, 30]. A relevant example is the general reaction–diffusion epidemic model proposed in 1993 by Capasso and Di Liddo to study the role of cross–diffusion [8]. They primarily focused on global attractivity of the uniform steady state solutions and indicated several specific diseases whose transmission may be described in such general settings, including host–vector diseases (as malaria and schistosomiasis) and carrier-borne diseases (as typhoid fever). The work [8] is of interest because later studies showed that in other important contexts in theoretical population dynamics the presence of cross–diffusion induces instability and patterning [16, 19]. This makes the results of [8] even more important, since the global stability of the endemic equilibrium was shown there. Moreover, and more closely related to the present work, the study by Capasso and Di Liddo stimulated some interesting works on the application of nonlinear Lyapunov Stability to Mathematical Biology [17, 22].

However, classical models of ME suffer of a major drawback: they are based on the analogy between contagion and chemical reactions, thus modelling the infections by means of the mass action law and the recovery as chemical degradation. In other words, classical models of ME essentially see human beings as molecules and therefore neglect the human behavioural changes. This approximation is not always adequate to represent real scenarios since the two major determinants of the epidemic spread, i.e. the degree of enacted social distancing and the degree of adherence to vaccination programs, fully depend on human decisions. The same could be said for the adoption of antiviral drugs, when available. This consideration has led to the birth of a new field, the Behavioural Epidemiology of Infectious Diseases (BEID) [21, 29]. Remarkably, the first pioneering example of BEID has probably been the paper on modelling social distancing published in 1978 by Capasso and Serio [9]. In such a paper, the contact rate was considered as a dynamical parameter that ‘adapts’ to the current state of the disease spread during an epidemic outbreak. Inexplicably, the paper [9] had a limited impact for more than a quarter of century but the basic concepts contained therein are nowadays fundamental in contemporary BEID and are at the basis of many works [21]. For example, in the paper [13] the adaptation depends on the state of the present and past information on the spread of epidemic and endemic diseases. As far as spatio-temporal effects in the framework of BEID are concerned, the body of research on this important aspect remained limited so far [4, 12, 18].

Our aim in this paper is twofold. From one hand, starting from the Capasso and Di Liddo general setting, we aim at providing a further relevant case, i.e. we propose a SIR-like reaction-diffusion epidemic model which embeds opinion-driven human behavioural changes. The model extends along the line of d’Onofrio et al. [12] the above-mentioned model by Capasso and Serio. On the other hand, we intend to give a new application of a special Lyapunov function introduced in 2006 by S. Rionero to study the nonlinear stability of equilibria of both spatially homogeneous and spatially non-homogeneous systems of differential equations [24–27]. Such a function has been successfully employed in a number of problems in theoretical population biol-

ogy. For example, it has been applied to diffusive population models like predator–prey interactions [10], pioneer and climax species [5], Lotka–Volterra with special functional response [11]. It has been also used to study ODE systems like SIR and SEIR classical epidemic models [6, 27]. In our case, we aim at using this approach to investigate the degree of the robustness of the endemic equilibrium induced by the human behaviour. At the best of our knowledge, this is its first application of the approach due to S.Rionero to models of BEID.

The rest of the paper is organized as follows: in Sect. 2 we introduce the general family of epidemic models and we discuss the existence of equilibria; in Sect. 3, we derive the perturbation system and rewrite it in an sort of ‘optimized form’ amenable to its analysis by means of the Rionero’s function. The problem of the nonlinear L^2 -stability of the endemic equilibrium is discussed in Sect. 4. Some conclusions end this work.

2 The model

Capasso and Di Liddo [8] considered the following general system of reaction–diffusion equations:

$$\partial w/\partial t = D\Delta w + f(w) \quad \text{in } \Omega \times \mathbf{R}_+, \tag{1}$$

with boundary conditions

$$\beta D\partial w/\partial \nu = \alpha (w^* - w) \quad \text{in } \partial\Omega \times \mathbf{R}_+, \tag{2}$$

where $\partial w/\partial \nu$ is the outward normal derivative and w^* is a constant vector in \mathbf{R}^m . The following assumptions are also considered:

- (i) The function $f : G \subseteq \mathbf{R}^m \rightarrow \mathbf{R}^m$, $m \in \mathbf{N} \setminus \{0\}$, is a locally Lipschitz function;
- (ii) The constant vector w^* is a spatially uniform steady state of (1–2), i.e. $f(w^*) = 0$;
- (iii) The domain Ω is a bounded open connected set of \mathbf{R}^n , $n \in \mathbf{N} \setminus \{0\}$. If $n > 1$, then Ω is assumed to be uniformly of class $C^{1+\alpha}$, with boundary $\partial\Omega$ uniformly of class $C^{2+\alpha}$, for some $\alpha > 0$;
- (iv) The constants α and β in (2) are nonnegative and such that $\alpha + \beta > 0$;
- (v) The term D is a linear operator on \mathbf{R}^m such that:

$$\langle z, Dz \rangle = \sum_{i,j=1}^m d_{ij} z_i z_j > 0,$$

where d_{ij} are the coefficients of the matrix D . In other words, the matrix D is *positive definite*.

d’Onofrio et al. [12] consider the following spatially structured SIR model with opinion-driven human behavioural changes in $\Omega \times (0, T)$:

$$\begin{aligned} \partial_t S &= D_S \Delta S + \hat{\mu} (1 - S) - \Psi(S, I) \\ \partial_t I &= D_I \Delta I + \Psi(S, I) - (\hat{\nu} + \hat{\mu})I \end{aligned} \tag{3}$$

where S is the spatial density of Susceptible subjects and I is the spatial density of infectious subjects; D_S and D_I are the diffusion rates of, respectively, susceptible and Infectious subjects; $\hat{\mu}$ is the death rate due to natural causes (for the sake of the simplicity, the recruitment rate is assumed to be equal to the death rate).

The function $\Psi(S, I)$ models the contagion rate. The dependence by S and I mimics the opinion-driven human behavioural changes in contact rate (in other words, the opinion-driven adoption of social distancing). In particular, it is assumed that $\Psi(S, I)$ can be decomposed as follows

$$\Psi(S, I) = \Phi(I) C(S, I),$$

where $C(S, I)$ is the contagion rate in absence of human behavioural response and $\Phi(I)$ models the impact of social distancing on the contagion rate. These assumptions implies that:

- (i) $0 \leq \Phi(I) \leq 1, \Phi(0) = 1, \Phi'(I) < 0$;
- (ii) $C(S, 0) = C(0, I) = 0, \partial_I C(S, I) > 0, \partial_S C(S, I) > 0$.

According to the general setting proposed by Capasso and Di Liddo [8] we append boundary conditions (2). Note that $\alpha = 0$ means Neumann homogeneous boundary conditions and $\beta = 0$ means Dirichlet boundary conditions. However, in order to apply the Lyapunov function proposed by S. Rionero, we will assume that both α and β are strictly positive constants.

Model (3) admits the *Disease-Free equilibrium* $DFE = (1, 0)$. Denote by $E = (\tilde{S}, \tilde{I})$ a spatially homogeneous equilibrium with $\tilde{I} > 0$ (*Endemic equilibrium*). Then:

$$\hat{\mu} (1 - \tilde{S}) = \Psi(\tilde{S}, \tilde{I}) = (\hat{\nu} + \hat{\mu}) \tilde{I}$$

and

$$\hat{\mu} - (\hat{\nu} + \hat{\mu}) \tilde{I} = \hat{\mu} \tilde{S}.$$

Therefore,

$$\tilde{S} = 1 - \frac{(\hat{\mu} + \hat{\nu})}{\hat{\mu}} \tilde{I} = 1 - (1 + \rho) \tilde{I}, \tag{4}$$

where

$$\rho = \frac{\hat{\nu}}{\hat{\mu}} \gg 1,$$

and

$$\Psi \left[1 - (1 + \rho) \tilde{I}, \tilde{I} \right] = (\hat{v} + \hat{\mu}) \tilde{I}.$$

Now, we first assume that the behavioural reaction of individuals influences the contact rate by making it no more constant. Namely, we assume that the effective contact rate is a decreasing function of the information on the disease prevalence: $\beta_{eff}(I)$, with $\beta'_{eff}(I) < 0$. Second, we also assume that the contagion rate $\beta_{eff}(I)IS$ remains an increasing function of the disease prevalence. This biologically realistic constraint can be fulfilled by many functions. However, following [9, 13], we choose:

$$\beta_{eff}(I) = \frac{\hat{\beta}}{1 + \hat{\alpha}I}. \tag{5}$$

In other words, the contagion rate is theoretically saturated with respect to the density of the disease prevalence:

$$\Psi(\tilde{S}, \tilde{I}) = \hat{\beta} \frac{SI}{1 + \hat{\alpha}I}. \tag{6}$$

Under such assumption, there exists an unique endemic equilibrium

$$E = (\tilde{S}, \tilde{I}), \tag{7}$$

where \tilde{S} is given by (4) and:

$$\tilde{I} = \frac{\hat{\mu}(R_0 - 1)}{\hat{\alpha}\hat{\mu} + \hat{\beta}},$$

where

$$R_0 = \frac{\hat{\beta}}{\hat{\mu} + \hat{v}}.$$

3 Perturbation system

3.1 Preliminaries

Using (6), model (3) reads:

$$\begin{cases} \partial_t S = D_S \Delta S + \hat{\mu}(1 - S) - \hat{\beta} \frac{SI}{1 + \hat{\alpha}I} \\ \partial_t I = D_I \Delta I + \hat{\beta} \frac{SI}{1 + \hat{\alpha}I} - (\hat{v} + \hat{\mu})I. \end{cases} \tag{8}$$

Consider the perturbations u, v such that:

$$S = \tilde{S} + k_1 u, \quad I = \tilde{I} + k_2 v.$$

where k_1 and k_2 are two positive quantities. We get:

$$\begin{cases} \partial_t(k_1u) = D_S\Delta(k_1u) + \mu(1 - k_1u - \tilde{S}) - \hat{\beta} \frac{(\tilde{S} + k_1u)(\tilde{I} + k_2v)}{1 + \hat{\alpha}(\tilde{I} + k_2v)} \\ \partial_t(k_2v) = D_I\Delta(k_2v) + \hat{\beta} \frac{(\tilde{S} + k_1u)(\tilde{I} + k_2v)}{1 + \hat{\alpha}(\tilde{I} + k_2v)} - (\hat{v} + \hat{\mu})(\tilde{I} + k_2v) \end{cases} \tag{9}$$

In the next subsection we will manipulate the kinetics terms of this system to write them a form that can be convenient for the application of the Rionero’s approach to stability.

3.2 Reformulation of the first equation

From the first equation, set:

$$F(u, v) = \hat{\mu}(1 - k_1u - \tilde{S}) - \hat{\beta} \frac{(\tilde{S} + k_1u)(\tilde{I} + k_2v)}{1 + \hat{\alpha}(\tilde{I} + k_2v)}.$$

We have:

$$\begin{aligned} F(u, v) &= \frac{\hat{\mu}(1-\tilde{S})\hat{\alpha}k_2v - \hat{\mu}k_1u(1+\hat{\alpha}\tilde{I}+\hat{\alpha}k_2v) - \hat{\beta}\tilde{S}k_2v - \hat{\beta}\tilde{I}k_1u - \hat{\beta}k_1k_2uv}{1+\hat{\alpha}\tilde{I}+\hat{\alpha}k_2v} \\ &= \frac{[\hat{\mu}(1-\tilde{S})\hat{\alpha}k_2 - \hat{\beta}\tilde{S}k_2]v + [-\hat{\mu}k_1(1+\hat{\alpha}\tilde{I}) - \hat{\beta}\tilde{I}k_1]u + [-\hat{\mu}k_1\hat{\alpha}k_2 - \hat{\beta}k_1k_2]uv}{1+\hat{\alpha}\tilde{I}+\hat{\alpha}k_2v}. \end{aligned}$$

Introduce some unknown quantities, a_{11} , a_{12} , A , and impose that $F(u, v)$ takes the form:

$$F(u, v) = \frac{a_{11}k_1u [1 + \hat{\alpha}(\tilde{I} + k_2v)] + a_{12}k_2v [1 + \hat{\alpha}(\tilde{I} + k_2v)] + Auv}{1 + \hat{\alpha}(\tilde{I} + k_2v)}.$$

One has:

$$a_{11} = -\frac{[\hat{\mu}(1 + \hat{\alpha}\tilde{I}) + \hat{\beta}\tilde{I}]}{1 + \hat{\alpha}\tilde{I}}, \tag{10}$$

$$a_{12} = \frac{\hat{\mu}(1 - \tilde{S})\hat{\alpha} - \hat{\beta}\tilde{S}}{1 + \hat{\alpha}\tilde{I}}, \tag{11}$$

and

$$A = -(\hat{\mu}\hat{\alpha} + \hat{\beta})k_1k_2 - \hat{\alpha}k_2a_{11}k_1.$$

The first equation of (9) becomes

$$\partial_t u = D_S \Delta u + a_{11}u + a_{12} \frac{k_2}{k_1} v + \frac{k_1^{-1} A uv + k_1^{-1} a_{12} \hat{\alpha} k_2^2 v^2}{[1 + \hat{\alpha}(\tilde{I} + k_2 v)]}.$$

3.3 Reformulation of the second equation

Set

$$G(u, v) = \hat{\beta} \frac{(\tilde{S} + k_1 u)(\tilde{I} + k_2 v)}{1 + \hat{\alpha}(\tilde{I} + k_2 v)} - (\hat{v} + \hat{\mu})(\tilde{I} + k_2 v),$$

that is

$$\begin{aligned} G(u, v) &= \frac{\hat{\beta} \tilde{S} \tilde{I} + \hat{\beta} \tilde{S} k_2 v + \hat{\beta} \tilde{I} k_1 u + \hat{\beta} k_1 k_2 uv - (\hat{\mu} + \hat{v})(\tilde{I} + k_2 v)(1 + \hat{\alpha} \tilde{I} + \hat{\alpha} k_2 v)}{1 + \hat{\alpha} \tilde{I} + \hat{\alpha} k_2 v} \\ &= \frac{[-(\hat{\mu} + \hat{v}) \tilde{I} \hat{\alpha} k_2] v + \hat{\beta} \tilde{I} k_1 u + \hat{\beta} k_1 k_2 uv - (\hat{\mu} + \hat{v}) \hat{\alpha} k_2^2 v^2}{1 + \hat{\alpha} \tilde{I} + \hat{\alpha} k_2 v} \end{aligned}$$

Introduce the quantities a_{21} , a_{22} , B , C , and impose that $G(u, v)$ takes the form:

$$G(u, v) = \frac{a_{21} k_1 u (1 + \hat{\alpha} \tilde{I} + \hat{\alpha} k_2 v) + a_{22} k_2 v (1 + \hat{\alpha} \tilde{I} + \hat{\alpha} k_2 v) + B uv + C v^2}{1 + \hat{\alpha} \tilde{I} + \hat{\alpha} k_2 v}.$$

It follows:

$$a_{21} = \frac{\hat{\beta} \tilde{I}}{1 + \hat{\alpha} \tilde{I}}, \tag{12}$$

and

$$a_{22} = -\frac{(\hat{\mu} + \hat{v}) \hat{\alpha} \tilde{I}}{1 + \hat{\alpha} \tilde{I}} = \left[\frac{\hat{\beta} \tilde{S}}{(1 + \hat{\alpha} \tilde{I})^2} - (\hat{\mu} + \hat{v}) \right] = -\frac{\hat{\alpha} \hat{\beta} \tilde{S} \tilde{I}}{(1 + \hat{\alpha} \tilde{I})^2}, \tag{13}$$

where we have used the equilibrium condition $(\hat{\mu} + \hat{v})(1 + \hat{\alpha} \tilde{I}) = \hat{\beta} \tilde{S}$. Furthermore,

$$\begin{aligned} B &= (\hat{\beta} - a_{21} \hat{\alpha}) k_1 k_2 \\ C &= -[(\hat{\mu} + \hat{v}) - a_{22}] \hat{\alpha} k_2^2 \end{aligned}$$

The second equation of (9) becomes

$$\partial_t v = D_I \Delta v + \frac{k_1}{k_2} a_{21} u + a_{22} v + \frac{k_2^{-1} B uv + k_2^{-1} C v^2}{[1 + \hat{\alpha}(\tilde{I} + k_2 v)]}.$$

In conclusion, the perturbation system is:

$$\begin{cases} \partial_t u = D_S \Delta u + a_{11}u + a_{12} \frac{k_2}{k_1} v + \frac{k_1^{-1} A u v + k_1^{-1} a_{12} \hat{\alpha} k_2^2 v^2}{[1 + \hat{\alpha}(\tilde{I} + k_2 v)]} \\ \partial_t v = D_I \Delta v + \frac{k_1}{k_2} a_{21}u + a_{22}v + \frac{k_2^{-1} B u v + k_2^{-1} C v^2}{[1 + \hat{\alpha}(\tilde{I} + k_2 v)]}. \end{cases} \tag{14}$$

which must be studied with the following boundary conditions in $\partial\Omega \times \mathbf{R}_+$:

$$\begin{cases} \beta D_S \partial u / \partial v = -\alpha u \\ \beta D_I \partial v / \partial v = -\alpha v. \end{cases} \tag{15}$$

4 Nonlinear L^2 -stability

In this section, we will apply the method developed in a series of papers by Rionero [24–26] to get the nonlinear L^2 stability of the equilibrium E given in (7). The method is based on the analysis of a suitable linear planar system of ordinary differential equations. The stability will be studied with respect to the perturbations (u, v) belonging, for all $t \in \mathbf{R}_+$, to $[W^{1,2}(\Omega, \alpha, \beta, D)]$, where $W^{1,2}(\Omega)$ is the functional space such that

$$\varphi \in W^{1,2}(\Omega, \alpha, \beta, D) \rightarrow \left\{ \varphi \in W^{1,2}(\Omega) : \text{the b. condit. (2) hold with } w^* = 0 \right\}. \tag{16}$$

In the space $W^{1,2}(\Omega, \alpha, \beta, D)$ the following inequality holds¹

$$\|\nabla\varphi\|^2 + \frac{\alpha}{D\beta} \|\varphi\|_{\partial\Omega}^2 \geq \hat{\lambda} \|\varphi\|^2, \tag{17}$$

where $\|\cdot\|$ denotes the L^2 -norm, $\|\cdot\|_{\partial\Omega}$ denotes the $L^2(\partial\Omega)$ -norm and the positive constant $\hat{\lambda}(\Omega, \alpha, \beta, D)$ is the lowest eigenvalue of the problem

$$\Delta\varphi + \lambda\varphi = 0, \tag{18}$$

in $W^{1,2}(\Omega, \alpha, \beta, D)$, that is the principal eigenvalue of $-\Delta$.

Now, take

$$\mu = \frac{k_1}{k_2},$$

¹ To obtain the inequality (17) starts from (18) to get $\varphi \Delta\varphi + \lambda\varphi^2 = 0$. Then integrating over Ω and applying the divergence theorem one gets $\int_{\partial\Omega} \varphi \nabla\varphi \cdot \mathbf{n} d\sigma - \int_{\Omega} (\nabla\varphi)^2 d\Omega + \lambda \int_{\Omega} \varphi^2 d\Omega = 0$. Taking into account of (15) one has $-\frac{\alpha}{D\beta} \int_{\partial\Omega} \varphi^2 d\sigma - \int_{\Omega} (\nabla\varphi)^2 d\Omega + \lambda \int_{\Omega} \varphi^2 d\Omega = 0$, and therefore (17), where $\hat{\lambda} \leq \lambda$.

add and subtract the quantities

$$(D_S - \bar{\epsilon}) \hat{\lambda} u; \quad (D_I - \bar{\epsilon}) \hat{\lambda} v, \tag{19}$$

where $\bar{\epsilon}$ is a constant to be chosen later, in the first and the second equation of (14), respectively. Take also into account of (18). System (14) now reads

$$\begin{cases} \partial_t u = b_{11} u + \frac{a_{12}}{\mu} v + \hat{F} + F^* \\ \partial_t v = \mu a_{21} u + b_{22} v + \hat{G} + G^*, \end{cases} \tag{20}$$

where,

$$b_{11} = a_{11} - (D_S - \bar{\epsilon}) \hat{\lambda}; \quad b_{22} = a_{22} - (D_I - \bar{\epsilon}) \hat{\lambda}, \tag{21}$$

and

$$\hat{F} = (D_S + \bar{\epsilon}) \Delta u + D_S \hat{\lambda} u; \quad \hat{G} = (D_I + \bar{\epsilon}) \Delta v + D_I \hat{\lambda} v, \tag{22}$$

and

$$F^* = \frac{k_1^{-1} A u v + k_1^{-1} a_{12} \hat{\alpha} k_2^2 v^2}{[1 + \hat{\alpha}(\tilde{I} + k_2 v)]}; \quad G^* = \frac{k_2^{-1} B u v + k_2^{-1} C v^2}{[1 + \hat{\alpha}(\tilde{I} + k_2 v)]}. \tag{23}$$

Now take the constant $\bar{\epsilon}$ in (19) such that:

$$0 < \bar{\epsilon} < \inf \left\{ D_S, D_I, \frac{|a_{11} - D_S \hat{\lambda}|}{\hat{\lambda}}, \frac{(a_{11} - D_S \hat{\lambda})(a_{22} - D_I \hat{\lambda}) - a_{12} a_{21}}{\hat{\lambda}(a_{11} - D_S \hat{\lambda} + a_{22} - D_I \hat{\lambda})}, \frac{|a_{11} - D_S \hat{\lambda} + a_{22} - D_I \hat{\lambda}|}{2 \hat{\lambda}} \right\}. \tag{24}$$

In this way, it follows that $b_{22} < 0$ (since $a_{22} < 0$) and $I < 0, \hat{A} > 0$ where

$$I = \text{tr } J = b_{11} + b_{22}; \quad \hat{A} = \det J = b_{11} b_{22} - a_{12} a_{21}, \tag{25}$$

are the principal invariants of the matrix J associated to the linear terms in (20).

Now, consider the Rionero's special type of Lyapunov functions [24–26]:

$$W = \frac{1}{2} \left[\hat{A} (\|u\|^2 + \|v\|^2) + \|b_{11} v - \mu a_{21} u\|^2 + \left\| \frac{a_{12}}{\mu} v - b_{22} u \right\|^2 \right]. \tag{26}$$

It immediately follows that the time derivative along the solution of (20) is given by

$$\dot{W} = \hat{A} I (\|u\|^2 + \|v\|^2) + \hat{\Psi} + \Psi^*, \tag{27}$$

where

$$\hat{\Psi} = \langle \bar{\alpha}_1 u - \bar{\alpha}_3 v, \hat{F} \rangle + \langle \bar{\alpha}_2 v - \bar{\alpha}_3 u, \hat{G} \rangle, \tag{28}$$

and

$$\Psi^* = \langle \bar{\alpha}_1 u - \bar{\alpha}_3 v, F^* \rangle + \langle \bar{\alpha}_2 v - \bar{\alpha}_3 u, G^* \rangle, \tag{29}$$

where $\langle \cdot \rangle$ denotes the scalar product in $L^2(\Omega)$, and

$$\bar{\alpha}_1 = \hat{A} + \mu^2 a_{21}^2 + b_{22}^2; \quad \bar{\alpha}_2 = \hat{A} + b_{11}^2 + \frac{a_{12}^2}{\mu^2}; \quad \bar{\alpha}_3 = \mu b_{11} a_{21} + \frac{a_{12} b_{22}}{\mu}. \tag{30}$$

Our aim now is to estimate the terms $\hat{\Psi}$ and Ψ^* in (27). Let us begin by observing that from (10), (11), (12), (13), (21) it follows that $a_{12} b_{22} > 0$ and $a_{21} b_{11} < 0$. Therefore it can be taken

$$\mu = \sqrt{\left| \frac{a_{12} b_{22}}{a_{21} b_{11}} \right|},$$

to get: $\bar{\alpha}_3 = 0$. As a consequence, taking into account the estimate (17) we obtain

$$\hat{\Psi} = \langle \bar{\alpha}_1 u, \hat{F} \rangle + \langle \bar{\alpha}_2 v, \hat{G} \rangle. \tag{31}$$

From (22) first term may be written

$$\langle \bar{\alpha}_1 u, \hat{F} \rangle = \bar{\alpha}_1 \int_{\Omega} \left[(D_s + \bar{\epsilon}) u \Delta u + D_s \hat{\lambda} u^2 \right] d\Omega.$$

Rearranging and using the divergence theorem we have

$$\langle \bar{\alpha}_1 u, \hat{F} \rangle = \bar{\alpha}_1 \left[(D_s + \bar{\epsilon}) \left(\int_{\partial\Omega} u \frac{\partial u}{\partial \nu} d\sigma - \int_{\Omega} (\nabla u)^2 d\Omega \right) + D_s \hat{\lambda} \int_{\Omega} u^2 d\Omega \right].$$

Therefore, taking into account the boundary conditions (15), we can write

$$\langle \bar{\alpha}_1 u, \hat{F} \rangle = \bar{\alpha}_1 D_s \left(-\frac{\alpha}{\beta D_s} \|u\|_{\partial\Omega}^2 - \|\nabla u\|^2 + \hat{\lambda} \|u\|^2 \right) - \bar{\alpha}_1 \bar{\epsilon} \left(\|u\|_{\partial\Omega}^2 + \|\nabla u\|^2 \right).$$

Using the inequality (17) and $-\bar{\alpha}_1 \bar{\epsilon} \|u\|_{\partial\Omega}^2 < 0$, it follows

$$\langle \bar{\alpha}_1 u, \hat{F} \rangle < -\bar{\alpha}_1 \bar{\epsilon} \|\nabla u\|^2.$$

Reasoning in the same way when managing the term $\langle \bar{\alpha}_2 v, G^* \rangle$ in (31), one gets:

$$\hat{\Psi} < -\bar{\alpha} \bar{\epsilon} \left(\|\nabla u\|^2 + \|\nabla v\|^2 \right), \tag{32}$$

where $\bar{\alpha} = \min \{ \bar{\alpha}_1, \bar{\alpha}_2 \} > 0$.

Now we need to estimate the term Ψ^* in (27) where $\bar{\alpha}_3 = 0$, i.e.:

$$\Psi^* = \langle \bar{\alpha}_1 u, F^* \rangle + \langle \bar{\alpha}_2 v, G^* \rangle. \tag{33}$$

By considering perturbations such that $k_2 v > -\tilde{I}$, from (23) one gets:

$$|F^*| \leq \frac{1}{k_1} [c_1 |uv| + c_2 |v|^2],$$

and

$$|G^*| \leq \frac{1}{k_2} [c_3 |uv| + c_4 |v|^2],$$

where $c_i, i = 1, \dots, 4$ are suitable positive constants. Therefore we get:

$$|\Psi^*| \leq \frac{1}{k_1} |\bar{\alpha}_1| [c_1 \langle |u|^2 |v| \rangle + c_2 \langle |u| |v|^2 \rangle] + \frac{1}{k_2} |\bar{\alpha}_2| [c_3 \langle |u| |v|^2 \rangle + c_4 \langle |v|^3 \rangle]. \tag{34}$$

Using the Hölder inequality it follows

$$|\Psi^*| \leq \hat{h} \left(\|u\|^2 + \|v\|^2 \right)^{1/2} \left[c_1 \|u\|_4^2 + (c_2 + c_3 + c_4) \|v\|_4^2, \right] \tag{35}$$

where $\hat{h} = \min \{k_1^{-1} |\bar{\alpha}_1|, k_2^{-1} |\bar{\alpha}_2|\}$. Using the inequality [28]:

$$\|f\|_4^2 \leq \kappa \left(\|\nabla f\|^2 + \|f\|^2 \right),$$

where $\kappa = \kappa(\Omega)$ is a positive constant, we have

$$|\Psi^*| \leq \kappa \hat{h} M \left(\|u\|^2 + \|v\|^2 \right)^{1/2} \left(\|u\|^2 + \|v\|^2 + \|\nabla u\|^2 + \|\nabla v\|^2 \right), \tag{36}$$

where $M = \max \{c_1, c_2 + c_3 + c_4\}$. Now, from the (26), being $\hat{A} > 0$, it follows that W is positive definite and equivalent to the $L^2(\Omega)$ -norm. In particular, there exist two constants $\tilde{\kappa}_1$ and $\tilde{\kappa}_2$, such that

$$\frac{\kappa_1}{2} \left(\|u\|^2 + \|v\|^2 \right) \leq W \leq \frac{\kappa_2}{2} \left(\|u\|^2 + \|v\|^2 \right). \tag{37}$$

Recalling that $I < 0$, from (27), (31) and (36), (37) one gets

$$\begin{aligned} \dot{W} &= \hat{A}I \left(\|u\|^2 + \|v\|^2 \right) + \hat{\Psi} + \Psi^* \\ &\leq -\frac{2\hat{A}|I|}{\kappa_2} W - \bar{\alpha} \bar{\epsilon} \left(\|\nabla u\|^2 + \|\nabla v\|^2 \right) + \frac{2\sqrt{2}\kappa\hat{h}M}{\sqrt{\kappa_1^3}} W^{3/2} \end{aligned}$$

$$\begin{aligned}
 & + \frac{\sqrt{2\kappa\hat{h}M}}{\sqrt{\kappa_1}} W^{1/2} \left(\|\nabla u\|^2 + \|\nabla v\|^2 \right) \\
 = & - \left(\frac{2\hat{A}|I|}{\kappa_2} - \frac{2\sqrt{2\kappa\hat{h}M}}{\sqrt{\kappa_1^3}} W^{1/2} \right) W - \left(\bar{\alpha}\bar{\epsilon} \right. \\
 & \left. - \frac{\sqrt{2\kappa\hat{h}M}}{\sqrt{\kappa_1}} W^{1/2} \right) \left(\|\nabla u\|^2 + \|\nabla v\|^2 \right) \\
 = & - \left(\frac{2\hat{A}|I|}{\kappa_2} - \frac{2\sqrt{2\kappa\hat{h}M}}{\sqrt{\kappa_1^3}} W^{1/2} \right) W - \frac{k_1}{2} \left(\frac{2\bar{\alpha}\bar{\epsilon}}{k_1} \right. \\
 & \left. - \frac{2\sqrt{2\kappa\hat{h}M}}{\sqrt{\kappa_1^3}} W^{1/2} \right) \left(\|\nabla u\|^2 + \|\nabla v\|^2 \right).
 \end{aligned}$$

Now take the initial value of W , say W_0 , such that:

$$\frac{2\sqrt{2\kappa\hat{h}M}}{\sqrt{\kappa_1^3}} W_0^{1/2} < \hat{w} = \inf \left\{ \frac{2\hat{A}|I|}{\kappa_2}, \frac{2\bar{\alpha}\bar{\epsilon}}{k_1} \right\}. \tag{38}$$

Then, $\dot{W}(0) < 0$, and $\dot{W}(\tilde{t}) < 0$ when

$$\frac{2\sqrt{2\kappa\hat{h}M}}{\sqrt{\kappa_1^3}} (W(t))^{1/2} = \hat{w}, \quad \text{for some } t = \tilde{t}.$$

Therefore

$$\frac{2\sqrt{2\kappa\hat{h}M}}{\sqrt{\kappa_1^3}} (W(t))^{1/2} < \hat{w}, \quad \text{for all } t > 0.$$

We conclude that:

$$\dot{W} \leq - \left(\frac{2\hat{A}|I|}{\kappa_2} - \hat{w} \right) W,$$

so that:

$$W(t) \leq W_0 e^{-\left(\frac{2\hat{A}|I|}{\kappa_2} - \hat{w}\right)t}.$$

This estimates indicates that the endemic equilibrium is nonlinearly stable in the W -norm, and therefore in the L^2 -norm, although the restriction (38) on the initial data

makes the stability not verified for all initial data, i.e. it is only *conditional* stability [28].

5 Conclusions

In this work, we focus on the problem of nonlinear stability of an uniform endemic equilibrium induced by human behaviour in a spatio-temporal SIR epidemic model. The impact of human behaviour on the contact rate is assumed to be of inverse linear affine type, which means that as result of opinion-driven adoption of social distancing the contagion rate is theoretically saturated with respect to the density of the disease prevalence. We use the Rionero's special type of Lyapunov function, which has been previously fruitfully employed in a number of problems in theoretical population biology. The task has been non-trivial despite of the simple form that we use to represent the human behavioural response. For the proposed model, we obtain a stability result which is subject to a restriction on the initial perturbation. Assessing the global stability of the endemic equilibrium remains an open problem.

Acknowledgements The authors thank Florinda Capone (University of Naples Federico II) for her valuable suggestions and discussions. This work has been performed under the auspices of the Italian National Group for Mathematical Physics (GNFM) of the National Institute for Advanced Mathematics (INdAM). B.B. acknowledges EU funding within the NextGenerationEU—MUR PNRR Extended Partnership initiative on Emerging Infectious Diseases (Project no. PE00000007, INF-ACT) and PRIN 2020 project (No. 2020JLWP23) “Integrated Mathematical Approaches to Socio–Epidemiological Dynamics”.

Funding Open access funding provided by Università degli Studi di Napoli Federico II within the CRUI-CARE Agreement.

Data availability statement Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

Declarations

Conflict of interest The author states that there is no conflict of interest.

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