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GLOBAL STABILITY OF INFECTIOUS DISEASE MODELS WITH CONTACT RATE AS A FUNCTION OF PREVALENCE INDEX

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Abstract. In this paper, we consider a $SEIR$ epidemiological model with information–related changes in contact patterns. One of the main features of the model is that it includes an information variable, a negative feedback on the behavior of susceptible subjects, and a function that describes the role played by the infectious size in the information dynamics. Here we focus in the case of delayed information. By using suitable assumptions, we analyze the global stability of the endemic equilibrium point and disease–free equilibrium point. Our approach is applicable to global stability of the endemic equilibrium of the previously defined SIR and SIS models with feedback on behavior of susceptible subjects.

1. **Introduction.** Although mathematical models which describe the spread of infectious diseases are among the most successful application of mathematics in biology [14], they were classically derived by using methods of mean field theories in statistical mechanics. In other words, the agents, who are persons or animals, were approximated by means of particles. This constitutes maybe the main limit of classical approach in mathematical epidemiology: agents involved in the infectious spread are not particles, and their behavior, including the psychological aspects are important in shaping the population dynamics. It was the first stressed by Capasso and Serio [13] in seventies, but only in recent years it increasingly became clear that the role of human behavior and also misconducts (as the pseudo-rational exemption) ought thus to be included in some manner in the modeling of infectious disease

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spreading, which is triggering a large corpus of scientific research (see, just to name a few of contributions, [1, 2, 3, 5, 6, 19, 20, 21, 23, 24, 43] and the collective book [39]). Although there is a wide range of approaches [39], all these works explicitly include the feedback (FB) that the information about an infectious disease has on the agents' behavior and thus on the spreading of the target disease [2, 15, 18, 43, 45].

A first type of FB is the one given by the influence of the information on the behavior of healthy subjects [9, 18].

A second type of FB is the *pseudo-rational exemption* which is defined as the family's decision to not vaccinate children because of a pseudo-rational comparison between the perceived risk of infection and the perceived risk of side effects caused by the vaccine [7, 8, 15, 16, 17].

We shall focus on the first kind of feedback, which has first been introduced in the above mentioned paper [13] in a SIR epidemic model, where the force of infection was modeled as a decreasing function of the fraction of infectious subjects. In [18], the pioneering work by Capasso and Serio was extended to take into the account that the behavior inducing the reduction of the contact rate is in reality influenced by the information on the spread. As previously stressed in [18] the information does not only reflect the current state of the spread but also past states, both due to delays and to memory of past epidemics. In case of *exponentially fading memory kernel* in [18] it was shown that there is a unique endemic equilibrium point (EEP), and that it is locally stable. Some sharp conditions for the global stability of EEP were given in [9]. They use the so-called geometrical approach to global stability problem, originally developed by Li and Muldowney [34, 35], which has gained some popularity in recent years (see, e.g., [10, 11, 12, 36, 37, 40]).

The investigation of the global stability of EEP has not only an intrinsic mathematical interest, but also a practical one, since verifying the global behavior of an EEP allows avoiding simulation for each specific set of parameters and initial conditions.

We note that the idea of a population changing its behaviour in response to external stimuli has been explored by other research groups [41, 42]. This developed formalism has been applied to a model SIR type epidemics [41] and a predator-prey model [42]. In [41], it is shown how one can model the response of the susceptible agents to the stimuli such as information about the epidemics as switches, and, thus, the authors obtained a model similar to the considered in [9, 18] for simple switches, and also obtained a model with hysteresis and permanent memory of past epidemics using bistable switches.

Here, we will introduce a $SEIR$ model and define the effects of the information-related behavior on the force of infection (FoI) of a disease. We will investigate its global behavior by means of appropriate Lyapunov's functions. In addition, we will adopt similar methods to briefly assess the global behavior of the previously defined SIR and SIS models with FB on behavior of susceptible subjects [9, 18].

The paper is organized as follows. In the next section, the general $SEIR$ epidemic model with contact rate as a function of the available information on the past disease prevalence is introduced. In Section 3 some preliminary properties of the $SEIR$ model are presented. Section 4 is concerned with the global stability properties of the equilibria by means of Lyapunov functions. In Section 5, we shall extend the method of Lyapunov functions to SIR and SIS models with negative feedback. Further comments on the biological relevance of our results and on the particulars

of the chosen approach are stated in Section 6, together with a few concluding remarks.

2. **Modeling the influence of the behavior on the contact rate.** In this section, we consider the following family of $SEIR$ epidemic models for a non fatal disease in a constant population, with information–related changes in contact patterns:

$$\begin{aligned} \frac{dS}{dt} &= \mu(1-S) - \beta(M)SI, \\ \frac{dE}{dt} &= \beta(M)SI - (\mu+\sigma)E, \\ \frac{dI}{dt} &= \sigma E - (\mu+\nu)I, \end{aligned} \tag{1}$$

and

$$\frac{dR}{dt} = \nu I - \mu R,$$

where $S(t)$, $E(t)$, $I(t)$ and $R(t)$ denote the subpopulations of susceptible, exposed, infectious and recovered with permanent immunity, respectively. There is no disease–related death. The natural death rate and birth rate are assumed to be equal, denoted by $\mu > 0$, and thus $S(t) + E(t) + I(t) + R(t) = 1$ for all time (the constant population size). The parameter $\sigma > 0$ denote the transfer rate between the exposed subpopulation and the infectious subpopulation. The parameter $\nu > 0$ describes the rate that the infectious subpopulation becomes recovered. The contact rate is a function of some information index $M(t)$ summarizing the current and the past history of the disease prevalence: $FoI(M(t)) = \beta(M(t))I(t)$, where $M(t)$ is related to the past prevalence through a suitable function F_a^n as follows [8, 9, 18]:

$$M(t) = \int_{-\infty}^{t} g(I(\tau))F_a^n(t-\tau)d\tau.$$

The term F_a^n is a delaying kernel. Generally, F_a^n is the density function for a gamma distribution:

$$F_a^n(u) = \frac{a^{n+1}u^n}{n!}e^{-au},$$

where $a > 0$ is a constant and $n \geq 0$ is an integer. The average delay is defined by $\tau = (n+1)/a$, and n is called the order of the delay kernel.
Throughout this paper, we use the kernel with $n = 0$, that is,

$$F_a^0(u) = ae^{-au}.$$

This kernel is called the weak exponential delay kernel or the exponentially fading memory kernel because it pays a declining weight to the past. The parameter "a" assumes the biological meaning of inverse of the average delay of the collected information on the disease, as well as the average length of the historical memory concerning the disease in study. Such kernel was also used in another infectious disease models with negative feedback [7, 8, 9, 16, 17, 18]. In this case we have,

$$\frac{dM}{dt} = ag(I) - aM. \tag{2}$$

The function $g(I)$ describes the role played by the infectious size in the information dynamics.

From the latter equality and the equations of system (1) we obtain the $SEIR$ model with information-dependent contact rate:

$$\begin{aligned} \frac{dS}{dt} &= \mu(1-S) - \beta(M)SI, \\ \frac{dE}{dt} &= \beta(M)SI - (\mu+\sigma)E, \\ \frac{dI}{dt} &= \sigma E - (\mu+\nu)I, \\ \frac{dM}{dt} &= ag(I) - aM. \end{aligned} \tag{3}$$

Since $R(t)$ does not appear in the equations of system (1), it is enough to consider only the equations for $S(t)$, $E(t)$ and $I(t)$.

The initial condition of ordinary differential equations (3) is given as

$$S(0) > 0,\ E(0) \geq 0,\quad I(0) > 0,\quad M(0) \geq 0. \tag{4}$$

Finally, we shall make the following assumptions on the functions $\beta(M)$ and $g(I)$.

(H1): $\beta(0) > 0$; $\beta(M) > 0$ for $M > 0$.
(H2): $\beta'(M) < 0$ for $M > 0$.
(H3): $g(0) = 0$; $g(I) > 0$ for $I > 0$.
(H4): $g'(I) > 0$ for $I > 0$.

It is clear from assumptions (**H1**) and (**H2**) that system (3) is an epidemic system with negative feedback. The following choice of a negative feedback is proposed in [9, 18]: as a rational function $\beta(M) = \beta_0/(1+pM)$, where β_0 and p are positive constants.

As for $g(I)$, the following choice is proposed in [9, 18]: as a function of prevalence of infection $g(I) = wI$, where w is a parameter subsuming aspects such as pathogenicity [3]; or as a saturating function $g(I) = wI/(1+qI)$, where w and q are positive constants.

3. Preliminaries.

The dynamics of infectious disease crucially depend on the basic reproductive number R_0. Following the definition of the basic reproductive number given by van den Driessche and Watmough [44], the basic reproductive number for system (3) is presented as

$$R_0 = \frac{\sigma\beta(0)}{(\mu+\sigma)(\mu+\nu)}. \tag{5}$$

Direct calculation shows that system (3) has two possible equilibrium points in the non-negative orthant $\mathbb{R}^4_+ = \{(S,E,I,M) \in \mathbb{R}^4 : S \geq 0, E \geq 0, I \geq 0, M \geq 0\}$: the disease-free equilibrium point $P_0 = (1,0,0,0)$, and a endemic equilibrium point $P^* = (S^*, E^*, I^*, M^*)$ where $S^* = \mu/(\mu + I^*\beta(g(I^*)))$, $E^* = (\mu+\nu)I^*/\sigma$, $M^* = g(I^*)$ and I^* is the solution of

$$\frac{\beta(g(I^*))\mu}{\mu + I^*\beta(g(I^*))} - \frac{(\mu+\nu)(\mu+\sigma)}{\sigma} = 0. \tag{6}$$

The number of solutions of equation (6) can be analyzed geometrically through determining the points of intersection of the graphs of functions $F_1(I)$ and $F_2(I)$ in

the first quadrant. The functions $F_1(I)$ and $F_2(I)$ are defined as

$$\begin{aligned} F_1(I) &= \frac{\mu\sigma}{(\mu+\nu)(\mu+\sigma)}\frac{\beta(g(I))}{(\mu+I\cdot\beta(g(I)))}, \\ F_2(I) &= 1. \end{aligned}$$

Using assumptions (**H1**) and (**H3**), and the expression of R_0 in (5), we obtain

$$F_1(0) = \frac{\sigma\beta(0)}{(\mu+\sigma)(\mu+\nu)} = R_0. \tag{7}$$

We calculate the derivative of $F_1(I)$

$$F_1'(I) = \frac{\mu\sigma}{(\mu+\nu)(\mu+\sigma)}\frac{\mu\beta'(g(I))\cdot g'(I)-\beta^2(g(I))}{(\mu+I\cdot\beta(g(I)))^2}.$$

By (**H2**) and (**H4**) holds, it is easy to see that $F_1(I)\to 0$ as $I\to+\infty$, and that the function $F_1(I)$ is decreasing ($F_1'(I)<0$). Note that if $R_0 = F_1(0) > 1$, then the graphs of functions $F_1(I)$ and $F_2(I)$ intersect at a single point in the first quadrant. This result indicates that if an EEP exists and it is unique. Note that an epidemiologically meaningful P^* does not exist if $R_0 = F_1(0) < 1$, and it becomes disease-free equilibrium point P_0 when $R_0 = F_1(0) = 1$.

We summarize the results for the existence of equilibrium points in the following theorem.

Theorem 3.1. *Suppose that the functions $\beta(M)$ and $g(I)$ satisfy the conditions (**H1**), (**H2**), (**H3**) and (**H4**). System (3) always has the disease-free equilibrium point $P_0 = (1,0,0,0)$. If $R_0 > 1$, there is a unique endemic equilibrium point $P^* = (S^*, E^*, I^*, M^*)$.*

Finally, we shall show that the system (3) is bounded.

Theorem 3.2. *Let $(S(t), E(t), I(t), M(t))$ be the solution of system (3) satisfying initial conditions (4). Then $S(t)$, $E(t)$, $I(t)$, and $M(t)$ are all bounded for all $t > 0$ at which the solution exists.*

Proof. From the first equation of (3), we obtain

$$\frac{dS}{dt} \le \mu(1-S),$$

and thus $\limsup_{t\to\infty} S \le 1$. Adding the first three equations of (3), we get

$$\frac{d}{dt}(S+E+I) = \mu(1-S-E-I) - \nu I \le \mu(1-S-E-I).$$

By a standard comparison theorem, we can conclude that $\limsup_{t\to\infty}(S+E+I) \le 1$. This relation and the fourth equation of (3) imply

$$\frac{dM}{dt} = ag(I) - aM \le ag(1) - aM,$$

and thus $\limsup_{t\to\infty} M \le g(1)$. Therefore, $S(t)$, $E(t)$, $I(t)$, and $M(t)$ are all bounded for all $t > 0$. This completes the proof. □

The dynamics of system (3) can be analyzed in the following bounded feasible region:

$$\Gamma = \left\{(S,E,I,M)\in\mathbb{R}^4_+ : S,\ E,\ I\ge 0,\ S\le 1,\ S+E+I\le 1,\ 0\le M\le g(1)\right\}.$$

Furthermore, the region Γ is positively invariant with respect to model (3).

4. **Global stability of equilibrium points.** In this section, we shall use the following Lyapunov function for systems with negative feedback:

$$U(M) \quad = \quad M - M^* - \int_{M^*}^{M} \frac{\beta(\eta)}{\beta(M^*)} d\eta. \tag{8}$$

Using assumptions (**H1**) and (**H2**), it is easy to verify that the function $U(M)$ has a global minimum at $M = M^*$ and satisfies $U(M) \geq U(M^*)$ with equal sign taken when $M = M^*$.

The function $U(M)$ is introduced to prove the global stability of the positive equilibrium in virus dynamics models with nonlytic immune response [46, 48].

We shall use the family of Volterra–type Lyapunov function

$$V(x) = x - 1 - \ln x. \tag{9}$$

Thus, the function $V(x)$ has a global minimum at $x = 1$ and satisfies $V(x) \geq V(1)$ the equality case being $x = 1$.

The Volterra–type function $V(x)$ is extensively used to demonstrate the global stability of the equilibria of Lotka-Volterra systems [22], infectious disease models [4, 14, 25, 26] and virus dynamics models [27, 46, 47, 48]. The function $V(x)$ was originally discovered by Vito Volterra as the first integral of classic predator–prey model.

We inspired by the Lyapunov function techniques that was developed during last decade [28, 29, 30, 31, 32] and particularly by the recent works [46, 48], we will determine the conditions for the global stability of the endemic equilibrium point of the epidemic system (3).

Remark 1. The functions U and V can be generalized to the form

$$H(x, f) = \int_{x^*}^{x} \left(1 - \frac{f(x^*)}{f(\eta)}\right) d\eta.$$

Volterra-type function is $H(x, x)$ and the function U is $H\left(M, \frac{1}{\beta}\right)$.

In Section 2, we assume that $\beta(M)$ satisfies assumptions (**H1**) and (**H2**). We also make the following assumption about the negative feedback on behavior of healthy subjects $\beta(M)$.

(H5): $(M\beta(M))' > 0$ for $M > 0$.

This assumption is a technical one, required to prove Lemma 4.1 (and the Theorems 4.3 and 5.1).

The following lemmas are used in the proof of the global stability of the EEP.

Lemma 4.1. *(See [48]) Let the hypotheses (**H2**) and (**H5**) hold, then*

$$\left(\frac{\beta(M)}{\beta(M^*)} - 1\right)\left(\frac{M\beta(M)}{M^*\beta(M^*)} - 1\right) < 0$$

for all $M > 0$ and $M \neq M^*$.

4.1. **Disease-free equilibrium point.** In the absence of the infectious disease, the system has a unique disease-free equilibrium point P_0. By constructing a Lyapunov function, we can prove the global stability of the disease-free equilibrium point P_0 when the basic reproductive number is less than or equal to unity.

Theorem 4.2. *Suppose that conditions (**H1**)–(**H4**) are satisfied. If $R_0 \leq 1$, then the disease-free equilibrium point P_0 of (3) is globally asymptotically stable in Γ.*

Proof. Let $W_s = V(S)$. Calculating the time derivative of $W_s(S)$, we obtain

$$\begin{aligned}\frac{d}{dt}\left[V(S)\right] &= \left(1-\frac{1}{S}\right)\left[\mu(1-S)-\beta(M)SI\right],\\ &= \mu\left(2-S-\frac{1}{S}\right)-\beta(M)SI+\beta(M)I. \end{aligned} \tag{10}$$

Let $W_{ei} = E + \frac{(\mu+\sigma)}{\sigma}I$. Next, we obtain

$$\begin{aligned}\frac{d}{dt}\left[E+\frac{(\mu+\sigma)}{\sigma}I\right] &= \beta(M)SI-(\mu+\sigma)E+\frac{(\mu+\sigma)}{\sigma}\left[\sigma E-(\mu+\nu)I\right],\\ &= \beta(M)SI-\frac{(\mu+\sigma)(\mu+\nu)}{\sigma}I. \end{aligned} \tag{11}$$

Now, define the Lyapunov function $W:\{(S,E,I,M)\in\Gamma : S>0\}\to\mathbb{R}$ by

$$W(S,E,I,M) = cW_s + cW_{ei}, \tag{12}$$

where c is a positive constant. Finally, adding (10) and (11), we obtain the derivative of W along the solutions of system (3):

$$\begin{aligned}\frac{dW}{dt} &= c\frac{dW_s}{dt}+c\frac{dW_{ei}}{dt},\\ &= c\mu\left(2-S-\frac{1}{S}\right)+c\beta(M)I-c\frac{(\mu+\sigma)(\mu+\nu)}{\sigma}I.\end{aligned}$$

Using assumptions (**H2**), we obtain $\beta(M)<\beta(0)$

$$\begin{aligned}\frac{dW}{dt} &< c\mu\left(2-S-\frac{1}{S}\right)-c\frac{(\mu+\sigma)(\mu+\nu)}{\sigma}\left[1-\frac{\sigma\beta(0)}{(\mu+\sigma)(\mu+\nu)}\right]I,\\ &< -c\mu\left[V(S)+V\left(\frac{1}{S}\right)\right]-c\frac{(\mu+\sigma)(\mu+\nu)}{\sigma}\left[1-R_0\right]I.\end{aligned}$$

$V(S)$ and $V\left(\frac{1}{S}\right)$ are Volterra–type functions. These functions are positive definite. Thus, $R_0\le 1$ implies that $dW/dt\le 0$. If $dW/dt=0$ then $S=1$ and $I=0$. Hence, W is a Lyapunov function on Γ. Thus, $(S,E,I)\to(1,0,0)$ as $t\to\infty$. Using $I=0$ in the last equation of (3) shows that $M\to 0$ as $t\to\infty$. Therefore, it follows from the LaSalle's Invariance Principle [33], that every solution of the equations in the model (3), with initial conditions in Γ, approaches P_0 as $t\to\infty$. This completes the proof. □

4.2. **Endemic equilibrium point.** We get the global stability of the EEP for the special case $g(I)=wI$.

Theorem 4.3. *Suppose that conditions (**H1**)–(**H5**) are satisfied. Assume that $g(I)=wI$. If $R_0>1$ then the unique endemic equilibrium point P^* of system (3) is globally asymptotically stable in the interior of the feasible region Γ.*

Proof. At endemic equilibrium point, we have

$$\mu = \mu S^* + \beta(M^*)S^*I^*, \tag{13}$$

$$\mu+\sigma = \frac{\beta(M^*)S^*I^*}{E^*}, \tag{14}$$

$$\mu+\nu = \frac{\sigma E^*}{I^*}, \tag{15}$$

$$M^* = wI^*. \tag{16}$$

Let $L_s = S^*V\left(\frac{S}{S^*}\right)$. By using (13), we have

$$\begin{aligned}\frac{d}{dt}\left[S^*V\left(\frac{S}{S^*}\right)\right] &= \left(1-\frac{S^*}{S}\right)\left[\mu - \mu S - \beta(M)SI\right],\\ &= \left(1-\frac{S^*}{S}\right)\left[\mu S^*\left(1-\frac{S}{S^*}\right)\right.\\ &\quad \left.+\beta(M^*)S^*I^*\left(1-\frac{\beta(M)}{\beta(M^*)}\frac{SI}{S^*I^*}\right)\right],\\ &= \mu S^*\left(2-\frac{S^*}{S}-\frac{S}{S^*}\right)\\ &+ \beta(M^*)S^*I^*\left(1-\frac{\beta(M)}{\beta(M^*)}\frac{SI}{S^*I^*}-\frac{S^*}{S}+\frac{\beta(M)}{\beta(M^*)}\frac{I}{I^*}\right). \quad (17)\end{aligned}$$

Define $L_e = E^*V\left(\frac{E}{E^*}\right)$. Using (14), we have

$$\begin{aligned}\frac{d}{dt}\left[E^*V\left(\frac{E}{E^*}\right)\right] &= \left(1-\frac{E^*}{E}\right)\left[\beta(M)SI-(\mu+\sigma)E\right]\\ &= \beta(M^*)S^*I^*\left(1-\frac{E^*}{E}\right)\left[\frac{\beta(M)}{\beta(M^*)}\frac{SI}{S^*I^*}-\frac{E}{E^*}\right]\\ &= \beta(M^*)S^*I^*\left[\frac{\beta(M)}{\beta(M^*)}\frac{SI}{S^*I^*}-\frac{E}{E^*}\right.\\ &\quad \left.-\frac{\beta(M)}{\beta(M^*)}\frac{SIE^*}{S^*I^*E}+1\right]. \quad (18)\end{aligned}$$

Let $L_i = I^*V\left(\frac{I}{I^*}\right)$. By using (15), we have

$$\begin{aligned}\frac{d}{dt}\left[I^*V\left(\frac{I}{I^*}\right)\right] &= \left(1-\frac{I^*}{I}\right)\left[\sigma E-(\mu+\nu)I\right],\\ &= \sigma E^*\left(1-\frac{I^*}{I}\right)\left[\frac{E}{E^*}-\frac{I}{I^*}\right],\\ &= \sigma E^*\left[\frac{E}{E^*}-\frac{I}{I^*}-\frac{I^*E}{IE^*}+1\right]. \quad (19)\end{aligned}$$

Let $L_m = U(M)$. Here we used (16).

$$\begin{aligned}\frac{d}{dt}\left[U(M)\right] &= \left(1-\frac{\beta(M)}{\beta(M^*)}\right)\left[awI-aM\right],\\ &= awI^*\left(\frac{I}{I^*}-\frac{M}{M^*}-\frac{\beta(M)}{\beta(M^*)}\frac{I}{I^*}+\frac{\beta(M)}{\beta(M^*)}\frac{M}{M^*}\right). \quad (20)\end{aligned}$$

Let us consider the Lyapunov function

$$L(S,E,I,M) = kL_s + kL_e + k\frac{\beta(M^*)S^*I^*}{\sigma E^*}L_i + k\frac{\beta(M^*)S^*}{aw}L_m, \quad (21)$$

where k is a positive constant. Computing the derivative of (21) along the solutions of system (3), we obtain

$$\frac{dL}{dt} = k\frac{dL_s}{dt} + k\frac{dL_e}{dt} + k\frac{\beta(M^*)S^*I^*}{\sigma E^*}\frac{dL_i}{dt} + k\frac{\beta(M^*)S^*}{aw}\frac{dL_m}{dt}. \quad (22)$$

Substituting (17)–(20) in (22), we obtain

$$\begin{aligned}
\frac{dL}{dt} &= k\mu S^*\left(2-\frac{S^*}{S}-\frac{S}{S^*}\right)\\
&+ k\beta(M^*)S^*I^*\left(3-\frac{S^*}{S}-\frac{\beta(M)}{\beta(M^*)}\frac{SIE^*}{S^*I^*E}-\frac{I^*E}{IE^*}\right)\\
&+ k\beta(M^*)S^*I^*\left(-\frac{M}{M^*}+\frac{\beta(M)}{\beta(M^*)}\frac{M}{M^*}\right),\\
&= k\mu S^*\left(2-\frac{S^*}{S}-\frac{S}{S^*}\right)\\
&+ k\beta(M^*)S^*I^*\left(4-\frac{S^*}{S}-\frac{\beta(M)}{\beta(M^*)}\frac{SIE^*}{S^*I^*E}-\frac{I^*E}{IE^*}-\frac{\beta(M^*)}{\beta(M)}\right)\\
&+ k\beta(M^*)S^*I^*\left(\frac{\beta(M^*)}{\beta(M)}-\frac{M}{M^*}+\frac{\beta(M)}{\beta(M^*)}\frac{M}{M^*}-1\right),\\
&= -k\mu S^*\left[V\left(\frac{S}{S^*}\right)+V\left(\frac{S^*}{S}\right)\right]\\
&- k\beta(M^*)S^*I^*\left[V\left(\frac{S^*}{S}\right)+V\left(\frac{\beta(M)}{\beta(M^*)}\frac{SIE^*}{S^*I^*E}\right)\right.\\
&\quad \left.+V\left(\frac{I^*E}{IE^*}\right)+V\left(\frac{\beta(M^*)}{\beta(M)}\right)\right]\\
&+ k\beta(M^*)S^*I^*\left(\frac{\beta(M)}{\beta(M^*)}-1\right)\left(\frac{M\beta(M)}{M^*\beta(M^*)}-1\right)\frac{\beta(M^*)}{\beta(M)}. \qquad (23)
\end{aligned}$$

The terms between the brackets, in the expression (23), are Volterra–type functions. These functions are positive definite.

By Lemma 4.1,

$$\left(\frac{\beta(M)}{\beta(M^*)}-1\right)\left(\frac{M\beta(M)}{M^*\beta(M^*)}-1\right)<0$$

holds for all $M>0$.

It is easy to see that dL/dt is negative in the interior of Γ. We have $dL/dt=0$ if and only if $\frac{S}{S^*}=1$, $\frac{I^*E}{IE^*}=1$ and $\frac{M}{M^*}=1$ holds. The largest compact invariant set in $\{(S,E,I,M)\in\Gamma : dL/dt=0\}$ is the singleton $\{P^*\}$, where P^* is the EEP. By LaSalle's invariance principle [33] then implies that P^* is globally asymptotically stable in the interior of Γ. This completes the proof. □

5. **SIR and SIS epidemic models.** Our approach is applicable for SIR and SIS epidemic models with an information-dependent contact rate that have been studied in [9, 18]. We give conditions for global stability of the endemic equilibrium point, whenever it exists.

First, SIR model is given by the following system of ordinary differential equations [9, 18]:

$$\begin{aligned}
\frac{dS}{dt} &= \mu-\mu S-\beta(M)SI,\\
\frac{dI}{dt} &= \beta(M)SI-(\mu+\nu)I, \qquad (24)\\
\frac{dM}{dt} &= ag(I)-aM,
\end{aligned}$$

and the equation of the recovered subpopulation is given by $dR(t)/dt = \nu I - \mu R$. The states variables and parameters are the same as in the $SEIR$ model. For this model, the feasible region is given by

$$\Omega = \left\{(S, I, M) \in \mathbb{R}^3_+ : 0 \leq S + I \leq 1,\ 0 \leq M \leq g(1)\right\},$$

and the basic reproductive number is still given by

$$R_0^{SIR} = \frac{\beta(0)}{\mu + \nu}. \tag{25}$$

For SIR model with special function $g(I) = wI$, we prove the global stability of EEP in the following theorem.

Theorem 5.1. *Suppose that conditions (**H1**)–(**H6**) are satisfied. Assume that $g(I) = wI$. If $R_0^{SIR} > 1$ then a unique endemic equilibrium $P^* = (S^*, I^*, M^*)$ of system (24) is globally asymptotically stable in the interior of Ω.*

Proof. The proof is similar to the proof of *Theorem 4.3*, but with the following Lyapunov function of the form

$$L(S, I, M) = kL_s + kL_i + k\frac{\beta(M^*)S^*}{aw}L_m, \tag{26}$$

where $k > 0$. The functions L_s, L_i and L_m are previously defined in subsection 4.2.

Define $L_i = I^*V\left(\frac{I}{I^*}\right)$. By using $(\mu + \nu) = \beta(M^*)S^*$, we have

$$\begin{aligned}
\frac{d}{dt}\left[I^*V\left(\frac{I}{I^*}\right)\right] &= \left(1 - \frac{I^*}{I}\right)[\beta(M)SI - (\mu + \nu)I] \\
&= \beta(M^*)S^*I^*\left(1 - \frac{I^*}{I}\right)\left[\frac{\beta(M)}{\beta(M^*)}\frac{SI}{S^*I^*} - \frac{I}{I^*}\right] \\
&= \beta(M^*)S^*I^*\left[\frac{\beta(M)}{\beta(M^*)}\frac{SI}{S^*I^*} - \frac{I}{I^*} - \frac{\beta(M)}{\beta(M^*)}\frac{S}{S^*} + 1\right]. \qquad (27)
\end{aligned}$$

The derivative of (26) along solution of (24) is given by

$$\frac{dL}{dt} = k\frac{dL_s}{dt} + k\frac{dL_i}{dt} + k\frac{\beta(M^*)S^*}{aw}\frac{dL_m}{dt}.$$

By using (17), (27) and (20), we obtain

$$\begin{aligned}
\frac{dL}{dt} &= k\mu S^*\left(2 - \frac{S^*}{S} - \frac{S}{S^*}\right) + k\beta(M^*)S^*I^*\left(3 - \frac{S^*}{S} - \frac{\beta(M)}{\beta(M^*)}\frac{S}{S^*} - \frac{\beta(M^*)}{\beta(M)}\right) \\
&\quad + k\beta(M^*)S^*I^*\left(\frac{\beta(M)}{\beta(M^*)} - 1\right)\left(\frac{M}{M^*} - \frac{\beta(M^*)}{\beta(M)}\right), \\
&= -k\mu S^*\left[V\left(\frac{S}{S^*}\right) + V\left(\frac{S^*}{S}\right)\right] \\
&\quad - k\beta(M^*)S^*I^*\left[V\left(\frac{S^*}{S}\right) + V\left(\frac{\beta(M)}{\beta(M^*)}\frac{S}{S^*}\right) + V\left(\frac{\beta(M^*)}{\beta(M)}\right)\right] \\
&\quad + k\beta(M^*)S^*I^*\left(\frac{\beta(M)}{\beta(M^*)} - 1\right)\left(\frac{M\beta(M)}{M^*\beta(M^*)} - 1\right)\frac{\beta(M^*)}{\beta(M)} \leq 0.
\end{aligned}$$

Clearly, $dL/dt \leq 0$, the conclusions are similar to the proof of *Theorem 4.3*. □

Second, the differential equations for the SIS model are [9]:

$$\begin{aligned} \frac{dS}{dt} &= \mu - \mu S - \beta(M)SI + \delta I, \\ \frac{dI}{dt} &= \beta(M)SI - (\mu+\delta)I, \\ \frac{dM}{dt} &= ag(I) - aM. \end{aligned} \tag{28}$$

Here the parameter $\delta > 0$ is the recovery rate. The other parameters and variables are the same as in the previous models.

To analyze the global stability of the EEP, first of all, we reduce the model to a two-dimensional model as follows. The system (28) is subject to the restriction $S(t) + I(t) = 1$, and using $S(t) = 1 - I(t)$ in the model, we can eliminate $S(t)$ from the equations. This substitution gives the simpler model:

$$\begin{aligned} \frac{dI}{dt} &= I\left(\beta(M)(1-I) - (\mu+\delta)\right), \\ \frac{dM}{dt} &= ag(I) - aM. \end{aligned} \tag{29}$$

The feasible region of system (29) is given by

$$\Sigma = \left\{(I, M) \in \mathbb{R}^2_+ : 0 \le I \le 1,\ 0 \le M \le g(1)\right\},$$

and the corresponding basic reproductive number is given by

$$R_0^{SIS} = \frac{\beta(0)}{\mu+\delta}. \tag{30}$$

For SIS model, we prove the global stability of EEP, in the following theorem.

Theorem 5.2. *Suppose that conditions (**H1**)–(**H4**) are satisfied. If $R_0^{SIS} > 1$ then a unique endemic equilibrium $P^* = (I^*, M^*)$ of system (29) is globally asymptotically stable in the interior of Σ.*

Proof. Consider the Lyapunov function

$$L(I, M) = k\int_{I^*}^{I} \frac{g(\eta) - g(I^*)}{\eta} d\eta + k\frac{(\mu+\delta)}{a} L_m, \tag{31}$$

where k is a positive constant. The function L_m is previously defined in subsection 4.2.

By using $1 = I^* + \frac{(\mu+\delta)}{\beta(M^*)}$, we have

$$\begin{aligned} \frac{d}{dt}\left[\int_{I^*}^{I} \frac{g(\eta) - g(I^*)}{\eta} d\eta\right] &= I\left(\frac{g(I) - g(I^*)}{I}\right)\left[(\mu+\delta)\left(\frac{\beta(M)}{\beta(M^*)} - 1\right)\right. \\ &\quad \left. + \beta(M)I^*\left(1 - \frac{I}{I^*}\right)\right], \\ &= (\mu+\delta)\left(g(I) - g(I^*)\right)\left(\frac{\beta(M)}{\beta(M^*)} - 1\right) \\ &+ \beta(M)I^*\left(g(I) - g(I^*)\right)\left(1 - \frac{I}{I^*}\right). \end{aligned} \tag{32}$$

Let $L_m = U(M)$. We have

$$\begin{aligned}\frac{d}{dt}\left[U(M)\right] &= \left(1-\frac{\beta(M)}{\beta(M^*)}\right)\left[ag(I)-aM\right],\\ &= \left(1-\frac{\beta(M)}{\beta(M^*)}\right)\left[a\left(g(I)-g(I^*)\right)-a(M-M^*)\right]. \qquad (33)\end{aligned}$$

The time derivative of (31) along the solutions of system (29),

$$\frac{dL}{dt} = k\frac{d}{dt}\left[\int_{I^*}^{I}\frac{g(\eta)-g(I^*)}{\eta}d\eta\right] + k\frac{(\mu+\delta)}{a}\frac{dL_m}{dt}.$$

By using (32) and (33), we obtain

$$\begin{aligned}\frac{dL}{dt} &= k(\mu+\delta)\left(g(I)-g(I^*)\right)\left(\frac{\beta(M)}{\beta(M^*)}-1\right)\\ &+ k\beta(M)I^*\left(g(I)-g(I^*)\right)\left(1-\frac{I}{I^*}\right)\\ &+ k(\mu+\delta)\left(1-\frac{\beta(M)}{\beta(M^*)}\right)\left(g(I)-g(I^*)\right)\\ &- k(\mu+\delta)\left(1-\frac{\beta(M)}{\beta(M^*)}\right)(M-M^*),\\ &= k\beta(M)I^*g(I)\left(1-\frac{I}{I^*}\right)\left(1-\frac{g(I^*)}{g(I)}\right)\\ &+ k(\mu+\delta)M^*\left(1-\frac{\beta(M)}{\beta(M^*)}\right)\left(1-\frac{M}{M^*}\right).\end{aligned}$$

Furthermore,

$$\left(1-\frac{I}{I^*}\right)\left(1-\frac{g(I^*)}{g(I)}\right)\leq 0,$$

since for an increasing function $g(I)$, $g(I)\geq g(I^*)$ when $I\geq I^*$ and $g(I)\leq g(I^*)$ when $I\leq I^*$. Also, for a decreasing function $\beta(M)$ ensures that

$$\left(1-\frac{\beta(M)}{\beta(M^*)}\right)\left(1-\frac{M}{M^*}\right)\leq 0,$$

with equality iff $M=M^*$.

Hence dL/dt is negative definite. By the [38], then implies that E^* is globally asymptotically stable in the interior of Σ. □

Remark 2. When $g(I)=wI$ the Lyapunov function $\int_{I^*}^{I}\frac{g(\eta)-g(I^*)}{\eta}d\eta$ is Volterra-type function.

6. **Concluding remarks.** We extended in this work the research of the dynamic implications of information-related changes in contact patterns for $SEIR$ diseases.

First, we study a $SEIR$ model (3) with an information variable M, a negative feedback on the behavior of susceptible subjects $\beta(M)$, and a function that describes the role played by the infectious size in the information dynamics $g(I)$. This system is the case of the *exponentially fading memory kernel* with $T_{delay}=a^{-1}$, which is the average delay of the collected information on the disease. We have identified the basic reproductive number, and we analyzed the global stability of both endemic equilibrium point P^* and the disease-free equilibrium point P_0. For the special

function $g(I) = wI$, we have shown the global asymptotic stability of the EEP. The results in this paper show the case of the exponentially fading memory kernel that does not affect the global asymptotic properties of the $SEIR$ model (3).

Second, an epidemiologically important consequence of the existence and uniqueness of an endemic equilibrium of system (3) is the analysis of the inhibitory effects of the information-related behavior on the force of infection. The effects of the information are to (i) increase the equilibrium number of susceptible, and (ii) reduce the equilibrium numbers of infected and exposed. We showed that the coordinates of EEP can be controlled.

Third, we extended our technique of Lyapunov functions to SIR and SIS models with contact rate as a function of prevalence index developed in [9, 18]. Our global stability conditions improve other recent results for these previous models [9]. For SIR model (24) with special function $g(I) = wI$, we obtained the global stability conditions of EEP under the technical requirement (**H5**). For SIS model (29), we obtained the global stability of EEP without the technical condition (**H5**). Clearly, the non-monotone function $\beta(M) = \beta_0(1 + pM^2)^{-1}$ and the negative exponential function $\beta(M) = \beta_0 e^{-pM}$ satisfy the stability conditions of Theorem 5.2. The low dimension of the SIS model facilitates global stability studies.

Finally, the results of this work indicate that our method of Lyapunov functions construction and suitable estimates of the derivatives of the Lyapunov functions, can be especially useful for higher-dimension systems with negative feedback.

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