Behavioral SIR models with incidence-based social-distancing

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A B S T R A C T

Most available behavioral epidemiology models have linked the behavioral responses of individuals to infection prevalence. However, this is a crude approximation of reality because prevalence is typically an unobserved quantity. This work considers a general endemic SIR epidemiological model where behavioral responses are incidence-based i.e., the agents perceptions of risks are based on available information on infection incidence. The differences of this modeling approach with respect to the standard 'prevalence-based' formulations are discussed and its dynamical implications are investigated. Both current and delayed behavioral responses are considered. We show that depending on the form of the 'memory' (i.e., in mathematical language, of the information delaying kernel), the endemic equilibrium can either be globally stable or destabilized via Hopf bifurcations yielding to stable recurrent oscillations. These oscillations can have a very long inter-epidemic periods and a very wide amplitude. Finally, a numerical investigation of the interplay between these behavior-related oscillations and seasonality of the contact rate reveals a strong synergic effect yielding to a dramatic amplification of oscillations.

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Time-delays
Oscillations

1. Introduction

The birth of mathematical epidemiology dates back to a century ago when a few pioneers established the key ideas of the discipline [1,2]. Their ground-breaking idea lied in the description of the key process namely, infection transmission from an infected to a susceptible individual, by the law of mass action of Kinetic Theory [3,4]. Accordingly, they represented social contacts between individuals as the random encounters of the particles of a perfect gas colliding in a box [5] and modeled transmission as a chemical reaction occurring with a given probability upon the occurrence of a random encounter:

$$Susceptible + Infectious \rightarrow Infectious + Infectious$$

In particular, the two key parameters, namely the per-capita *contact* rate per unit time, and the *infection transmission probability* per contact, were taken as natural constants of human behavior, possibly mirroring the social characteristics of a given community or setting, at a certain time moment.

Building on extensions of this simple idea, more recent pioneering contributions aiming to better integrate models with data, allowed mathematical models of infectious diseases to become central supporting tools for public health decisions about e.g., the fraction of new-born children to be immunized for a vaccine-preventable infection or the social-distancing measures needed to mitigate a deadly epidemic, or the proportions to be screened to prevent a serious sexually transmitted infection [6,7].

Some of contemporary models are highly sophisticated in both their mathematical/computational structure as well as in their data requirements, as apparent also from some of the main models adopted during the COVID-19 pandemic crisis, of which we just quote a few here [8–10]. In all these highly sophisticated models, the key ingredients are represented by the determinants of infection transmission, namely the patterns of social contacts with which individuals contact each other, which can be classified according to a range of characteristics (e.g. age, level of social/sexual activity, the contacts' site etc.). However, even in these highly sophisticated models a layer is still under-developed, namely that of humans' behavior. Indeed, until very recently the individuals' social behavior has been taken as unaffected by the state of the infection and its serious consequences i.e., even during severe epidemic outbreaks individuals are assumed to continue to contact each other at the same rate regardless of how low or high is the perceived risk of acquiring the infection.

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This static human behavior is an unrealistic abstraction because, by their very nature, humans are neither static nor passive. This is even more true in contemporary scenarios in view of the dramatic amount and speed of information circulation. The current COVID–19 pandemics, with its pervasive impact on society, has shown an endless list of instances of spontaneous behavior changes by individuals in response to the pandemic threat and its mitigation measures (see e.g., the review in [11] and references therein).

The need to seriously account for human behavior has led in the last 15 years (i.e., long before the COVID–19 pandemic) to the birth of a new branch of mathematical epidemiology, which we termed the *behavioral epidemiology* (BE) of infectious diseases [12,13]. Summaries of the many different facets of BE can be found in a number of reviews on the subject [12–14]. It is worth noting that BE models have been, of course, proposed to model various aspects of the current pandemic (see e.g., [15, 16] and references therein).

A key ingredient of BE modeling is represented by the so–called *information index* [12,17]. This is any summary measure of observable epidemiological dimensions (e.g., the incidence of cases of serious infection, or of hospitalization and deaths due to invasive disease following infection) that agents acquire from the communication systems and use to formulate their risk perception and the ensuing behavioral response. By far, the commonest type of information index adopted in a first generation of BE models has been the prevalence of infection, yielding to a wide number of *prevalence-based models* [12–14], sometimes expanded to account for a number imperfections in the availability of information (e.g., the presence of time-delays), or for the presence of heterogeneities (e.g., distribution of perceived risks across space), or of uncertainty, have pervaded the development of BE. Nonetheless, infection prevalence can hardly represent the type of information used by agents to elaborate their perceptions of risks, simply because prevalence is most often an unobserved epidemiological quantity. For example, in the case of childhood vaccine preventable infections (but also for COVID–19), appropriate knowledge of prevalence requires costly serological surveys [18]. These issues were noted in early discussions of the topic, where indeed both prevalence-dependent and incidence-dependent models for vaccinating behavior were proposed [17,19–21]. Consistently, in this work we extend the class of simple BE models by proposing a general - *behavior–implicit* - susceptible-infective-removed (SIR) framework for an endemic infectious disease as e.g., measles, where the agents' behavioral response to the risks perceived from infection is modeled by an incidence-based transmission rate. This will lead to a specific new class of behavior-epidemiology models. The framework adopted here has been termed as 'behavior-implicit' because it considers a phenomenological formulation for the behavioral response, but can be readily extended to a number of *behavior–explicit* formulations [12–14].

In particular, we consider both the scenario of a *current* behavioral response - whereby individuals respond to changes in current incidence only - as well as delayed responses, whereby individuals also respond to changes in past incidence, resulting in delay- differential models with distributed delay. For the latter case we could show delayed behavioral responses can trigger of a quite general nature. Finally, we study the interplay between these behavior-induced oscillations with those induced by the seasonality in the contact rate. The article is organized as follows. In Section 2 we introduce the main ideas of behavior–implicit social distancing. In Section 3 we formulate our general model with a (behavior–implicit) incidence-based transmission rate, whose qualitative properties are analytically studied in Section 4. Section 5 investigates the effects of incidence-based temporal delays, whose examples and simulations are reported in Section 6. The interplay between the agents' memory and seasonality is numerically investigated in Section 7. Concluding remarks follow.

2. Behavior–dependent contact rates and information indexes

Let us consider a standard SIR transmission model for an endemic infectious diseases in the absence of vaccination:

$$S' = \mu - \mu S - \beta(t)SI \tag{1}$$

$$I' = I(\beta(t)S - (\mu + \nu)) \tag{2}$$

$$R = 1 - S - I \tag{3}$$

where S,*I*,*R* denote the fractions of individuals who are, respectively, susceptible to infection, infective, i.e. able to retransmit infection to others, and *removed* because of e.g., permanent immunity acquired after recovery from infection. The infective fraction *I* is also termed the *infection prevalence*.

The critical parameter is represented by function $\beta(t)$, termed the *transmission rate*, which incorporates two distinct sub-processes namely, the social mixing process and the contagion process. The transmission rate is typically time-dependent, think e.g. to the case of childhood vaccine preventable infections, where $\beta(t)$ is forced periodically by the alternance between school and holiday terms, and possibly also by weather influence. Grown from some pioneering work ([22–24] and references therein), there is nowadays a well-established literature investigating the effects of periodically and stochastically forced transmission in basic epidemiological models.

Functions

$$J = \beta IS$$

and

$$FoI = \beta I$$

are called, respectively, the *infection incidence*, representing the per-capita flow of new infections per unit time, and the *force of infection*, representing the per-capita rate at which susceptible individuals acquire infection. Infected individuals are assumed to recover at a constant *recovery rate* $\nu > 0$.

Finally, $\mu > 0$ denotes both the birth and death rates, which are assumed identical to ensure the stationarity of the population over time, which is a common assumption in Mathematical Epidemiology [7]. For $\mu = 0$, the model collapses into the classical epidemic SIR model.

In the case of a constant transmission rate $\beta(t) = \beta$, the model behavior is summarized by the *basic reproduction number*

$$R_0 = \beta/(\nu + \mu)$$

representing the number of secondary infections generated by a single infective individuals during her infective period (of duration $D = 1/(\nu + \mu)$ in a wholly susceptible population. For $\mathscr{R}_0 \leq 1$ there is a unique steady state, the so-called disease free equilibrium: $DFE = (1,0,0)$, which is globally asymptotically stable (GAS). For $\mathscr{R}_0 > 1$ a unique endemic equilibrium $EE = (S^e, I^e, R^e)$ appears which can be proven to be GAS.

In the mathematical epidemiology literature, Capasso and Serio [25] were first in modeling the influence of human behavior in simple SIR epidemiological models. In their work, which focused on the epidemic case ($\mu = 0$), the transmission rate β was taken as a decreasing function of infection prevalence: $\beta = \beta(I(t))$, with: $(d/dI)\beta(I) < 0$. This was done to account for 'psychological effects....because in the presence of a very large number of infectives the population may tend to reduce the number of contacts per unit time' [25]. The authors pointed out that, unlike standard mass action approaches, this formulation could cause the force of infection to become a non-monotone function of infection prevalence (e.g., if $\beta(I) = \beta_0(1 + hI^2)^{-1}$).

In contemporary behavioral epidemiology language, the model by Capasso and Serio represented the first *behavior implicit* epidemic

model with *prevalence-dependent social distancing*. Since [25], several other works have investigated epidemic models with a non-linear force of infection ([12,13] and references therein).

In previous work on behavior-implicit SIR models with vaccination, we represented the individual's propensity to be vaccinated as a generic function of the available information on the present and past states of the infection [17], or of the information on vaccine adverse events [26]. Formally, we did this by introducing suitable variables $M(t)$ that we termed *information indexes*. In other works [27], we extended these ideas to social distancing, by allowing the contact rate β to be a generic function of an appropriate information index M, such that:

$$FoI(M) = \beta(M)I \tag{4}$$

where $(d/dM)\beta(M) < 0$. In this case, the information index M represents any summary of the available information on the risk of infection and its sequelae, which is used by agents to modulate their behavior at risk.

Once plugged into the above SIR model, assumption (4) yields the following family of SIR models with behavior-dependent contact rate: [27]:

$$S' = \mu(1 - S) - \beta(M)IS \tag{5}$$

$$I' = \beta(M)IS - (\mu + \nu)I \tag{6}$$

completed by the balance equation of the removed fraction $R(t)$: $R(t) = 1 - S(t) - I(t)$ and by the specific model of the index M. In [27] we showed that - regardless of the specific hypotheses made on M - models (5)–(6) always admits the disease free equilibrium

$$DFE = (1, 0)$$

which is GAS when $\beta(0) \le (\mu + \nu)$, whereas it is unstable for $\beta(0) > (\mu + \nu)$. Equivalently, the DFE is unstable when $\mathscr{R}_0(0) = \beta(0)/(\mu + \nu) > 1$ i.e., when the basic reproduction number $\mathscr{R}_0(0)$ in conditions of 'negligible' information - i.e., when no behavioral response is triggered - exceeds one. In the following, we will assume that the DFE is unstable i.e., the disease is always endemic in the absence of behavioral responses.

In a first approximation, one could assume that M is a continuous function ω of current values of S,I variables i.e., $M(t) = \omega(S,I)$ with $\partial_I\omega(S,I) > 0$. More realistically M will also depend on past values of state variables [12,17], yielding the equation

$$M(t) = \int_{-\infty}^{t} \omega(S(\tau), I(\tau))K(t - \tau)d\tau \tag{7}$$

where function K is a delaying, or memory, kernel [28] .

In the special case $K(t) = \delta(t)$, where δ is the Dirac function, the unlagged case $M(t) = \omega(S(t),I(t))$ is recovered.

In particular in [27] we only focused on the *prevalence-dependent* case i.e., $M = f(I)$. This allowed us to show that the system has a unique endemic equilibrium, which is globally stable in the memoryless case [27] as well as in the case of exponentially fading memories [29], but can be destabilized by appropriate nontrivial memory kernels [27].

3. SIR models with incidence-based behavioral responses: the memoryless case

The novelty of this work is that the individuals' social distancing response is assumed to depend on the information on the infection incidence. We will first introduce incidence-based information indexes, and use them to formulate general SIR endemic models with incidence-based social distancing.

In particular, we depart from the memoryless case, where only information on current incidence is used by agents to formulate their risk perceptions. In this case, the information index M is defined (implicitly) as an increasing function of current incidence:

$$M = A(\beta(M)SI) \tag{8}$$

where $(d/dM)\beta(M) < 0$ and $(d/du)A(u) > 0$.

It follows that:

$$J = \beta(M)SI = A^{-1}(M(S,I)) \tag{9}$$

yielding the following differential-algebraic system

$$S' = \mu(1 - S) - A^{-1}(M) \tag{10}$$

$$I' = A^{-1}(M) - (\mu + \nu)I \tag{11}$$

$$M = A(\beta(M)SI) \tag{12}$$

to be complemented by equation $R = 1 - S - I$.

Systems (10)-(11)-(12) defines a general family of behavior-implicit SIR models with *incidence-based social distancing*.

In relation to Systems (10)-(11)-(12), note that

$$\partial_I M = A'(\beta(M)SI)\frac{\beta(M)S}{1 - \beta'(M)A'(\beta(M)SI)SI} > 0$$

(where, for the sake of the notation simplicity, $f'(z) = (d/dz)f(z)$) and, similarly, $\partial_S M > 0$.

As a consequence, a function $\phi(X)$ exists with $\phi'(X) > 0$, allowing to define the information index as

$$M = \phi(SI) \tag{13}$$

It follows that the force of infection reads

$$FoI = \frac{A^{-1}(M(S,I))}{S}$$

implying

$$\partial_I FoI > 0 \tag{14}$$

This leads to the following important epidemiological consequence:

Corollary If social distancing is uniquely determined by incidence then the force of infection cannot be unimodal. That is, unlike the prevalence-dependent case, the force of infection cannot decrease for large I.

In what follows, for sake of generality we will assume $A(x) = kx$ where $k \in (0,1)$ is the under–reporting rate of actual incidence.

4. The memoryless case: equilibria and global stability analysis

Systems (10)-(11)-(12) always has a disease free equilibrium:

$$DFE = (1, 0)$$

which is GAS if $\beta(0) \le (\mu + \nu)$ and unstable if $\beta(0) > \mu + \nu$. Of course, in the unstable case the stable manifold of the DFE is given by the axis $I = 0$ (we omit the trivial proof).

To investigate the existence of endemic states we shall focus on the case $\beta(0) > \mu + \nu$ with initial conditions in the set

$$\Omega = \{(S,I) | S \ge 0, I > 0, S + I \le 1\}$$

If the model admits endemic states

$$EE = (S_e, I_e)$$

they must be such that

$$S_e = 1 - \alpha I_e \tag{15}$$

$$M_e = A((\mu+\nu)I_e) \tag{16}$$

$$\beta(M_e)S_e = \mu+\nu \tag{17}$$

where $\beta_e := \beta(M_e)$. This implies

$$S_e = 1 - \alpha I_e \tag{18}$$

$$M_e = A((\mu+\nu)I_e) \tag{19}$$

$$r(I_e) = \frac{1}{1-\alpha I_e} \tag{20}$$

where

$$r(I_e) = \frac{\beta(A((\mu+\nu)I_e))}{\mu+\nu}$$

which is a decreasing function of I_e, such that $r(0) = \beta(0)/(\mu+\nu) > 1$. Therefore, Eq. (20) implies that Eq. (20) has a unique solution $I_e \in (0,\mu/(\mu+\nu))$ i.e., the system has a unique endemic equilibrium point.

As for the local stability of the endemic state, for the sake of simplicity we will consider here only the baseline case $A(x) = kx$ [17], but the present discussion easily extends to the case where $A(x)$ is a generic increasing function. In this case, our system reads

$$S' = \mu(1-S) - \frac{M}{k} \tag{21}$$

$$I' = \frac{M}{k} - (\mu+\nu)I \tag{22}$$

$$M = k\beta(M)SI \tag{23}$$

A linearization of system (21)-(22)-(21) at the endemic state *EE* by setting $(S,I,M) = (S_e,I_e,M_e) + (s,i,m)$ yields the equations

$$s' = -\mu s - \frac{m}{k} \tag{24}$$

$$i' = \frac{m}{k} - ri \tag{25}$$

$$m = k\frac{hs+ri}{1+k\sigma} \tag{26}$$

where $h = \beta(M_e)I_e$, $r = \beta(M_e)S_e = \mu+\nu$ and $\sigma = -\beta'(M_e)S_eI_e > 0$.

The corresponding characteristic polynomial reads

$$P(\lambda) = \left(\lambda+\mu+\frac{h}{1+k\sigma}\right)\left(\lambda+\frac{rk\sigma}{1+k\sigma}\right)+\frac{hr}{1+k\sigma}$$

showing that both eigenvalues have negative real part, implying that the endemic state is locally asymptotically stable (LAS).

To exclude the existence of closed orbits in Ω, an application of the Dulac-Bendixon theorem to Systems (21)–(22) using various standard integrating factor (such as $1/I$), failed. Therefore, we applied the following continuous transformation of variables:

$$(X,I) = (SI,I)$$

leading to the system

$$X' = \mu(I-X) - I\frac{\phi(X)}{k} \tag{27}$$

$$I' = \frac{X}{I}\frac{\phi(X)}{k} - (\mu+\nu)I \tag{28}$$

Systems (27)–(28) does not admit closed orbits since its divergence is always negative:

$$div(X',I') = -\mu - I\frac{\phi'(X)}{k} - \frac{X}{I^2}\frac{\phi(X)}{k} - (\mu+\nu) < 0$$

It follows that, for the original system, the endemic state *EE* is always GAS in Ω. In words, no incidence-based social distancing response is able to destabilize the endemic state of the basic endemic SIR model (similarly to what occurs for the corresponding prevalence-based system analysed in [27]).

5. SIR models with incidence-based behavioral responses: the role of memory

In this section we analyse the effects of *'delayed'* incidence-based social distancing, by which individuals determine their behavioral responses accounting not only for current incidence but also for past incidence trends.

In this case, the following general SIR model arises:

$$S' = \mu(1-S) - \beta(M)IS \tag{29}$$

$$I' = \beta(M)IS - (\mu+\nu)I \tag{30}$$

$$M(t) = \int_{-\infty}^{t} k\beta(M(\tau))S(\tau)I(\tau)K(t-\tau)d\tau \tag{31}$$

To understand the dynamical implications of delayed incidence-based social-distancing, we now turn to the analysis of the local stability of the endemic state *EE* under two main subcases of kernel $K(.)$, namely, (i) the classical case of an *exponentially fading memory*, and (ii) the more realistic case of an *acquisition-fading memory* [30], where an exponentially declining phase is preceded by an initial phase of information acquisition.

5.1. The effects of an exponentially fading memory

Exponentially fading memory kernels are represented by $K(t) = a\exp(-at)$, with expectation $<t>$ given by the fading time scale $T = 1/a$ [28]. By applying the linear chain trick [28] to Eqs. (29)–(30)–(31), we get the following three-dimensional system:

$$S' = \mu(1-S) - \beta(M)IS \tag{32}$$

$$I' = \beta(M)IS - (\mu+\nu)I \tag{33}$$

$$M' = a(k\beta(M)SI - M) \tag{34}$$

The corresponding Jacobian matrix J at the endemic state reads as follows:

$$J = \begin{pmatrix} -(\mu+h) & -r & \sigma \\ h & 0 & -\sigma \\ akh & akr & -a(1+k\sigma) \end{pmatrix}$$

whose characteristic polynomial is given by

$$P(\lambda) = \lambda^3 + c_2\lambda^2 + c_1\lambda + c_0$$

where:

$$\begin{aligned} c_2 &= \mu + h + a(1+k\sigma) > 0 \\ c_1 &= hr + a(hr + k\sigma(\mu+r)) > 0 \\ c_0 &= ar(h + \mu k\sigma) > 0 \end{aligned}$$

The Routh-Hurwitz condition for local stability reduces to the condition $c_1c_2 > c_0$ which reads:

$$h_2 a^2 + h_1 a + h_0 > 0$$

which is true for all $a > 0$ since it is straightforward to verify that

$$h_2 = (1 + k\sigma)(hr + k\sigma(\mu + r)) > 0$$
$$h_0 = hr(\mu + h) >$$

and

$$h_1 = -hr + hr(1 + k\sigma) - \mu rk\sigma + (\mu + h)(hr + k\sigma(r + \mu)) =$$
$$h^2 + hk\mu\sigma + 2hkr\sigma + 2h\mu + k\mu^2\sigma + \mu^2 > 0$$

As a consequence, under an exponentially fading memory the EE is LAS for all $a > 0$.

In plain words, no 'exponentially delayed' incidence-based social distancing response is able to destabilize the endemic state, regardless of how far into the past goes the information used by agents to elaborate their behavioral response.

5.2. The effects of an acquisition-fading kernel

The *acquisition-fading* kernel, introduced in [30], has the following form:

$$K(t) = \frac{1}{T_b - T_a}\left(e^{-t/T_b} - e^{-t/T_a}\right) \tag{35}$$

This kernel models two sub-processes occurring independently and at different time-scales: *i)* the first one is represented by formation and acquisition of information, with time-scale T_a, as mirrored by the fact that $K(0) = 0$ i.e., one has no instantaneous knowledge of infection spread; *ii)* the second one is a process of fading of the acquired information, with time-scale T_b. Often the first process is much faster than the second i.e., $T_b > T_a$. The acquisition-fading kernel has expectation $< t > = T_a + T_b$ and $Var(t) = T_a^2 + T_b^2$. Note that the second order Erlang kernel $K(t) = a^2 t \exp(-at)$, most often used in the literature on time-lags [28] corresponds to Eq. (35) for $b \to a$.

Again, by an application of the linear chain trick [28], we get the 4-dimensional system:

$$S' = \mu(1 - S) - \beta(M_2)IS \tag{36}$$

$$I' = \beta(M_2)IS - (\mu + \nu)I \tag{37}$$

$$M_1' = a(g(S, I) - M_1) \tag{38}$$

$$M_2' = b(M_1 - M_2) \tag{39}$$

where $a = 1/T_a, b = 1/T_b, M(t) = M_2(t)$. Note that the equations for M_1 and M_2 mimick the fact that the type of time lag described by the acquisition–fading distribution is a sequence of two independent exponentially fading memories.

The Jacobian matrix at the endemic equilibrium reads:

$$J = \begin{pmatrix} -(\mu + h) & -r & 0 & \sigma \\ h & 0 & 0 & -\sigma \\ akh & akr & -a & -ak\sigma \\ 0 & 0 & b & b \end{pmatrix}$$

The resulting characteristic polynomial is

$$P(\lambda) = \lambda^4 + \sum_{i=0}^{3} q_i \lambda^i$$

where

$$q_3(a, b) = a + b + h + \mu > 0,$$
$$q_2(a, b) = ab(1 + k\sigma) + (h + \mu)(a + b) + hr > 0,$$
$$q_1(a, b) = ab(\mu + h + (\mu + r)k\sigma) + hr(a + b),$$
$$q_0(a, b) = abr(h + \mu k\sigma) > 0$$

The Routh-Hurwitz condition for the local stability of the endemic state *EE* can be written as follows:

$$RH(a, b) = \left(\frac{q_1}{q_3}\right)^2 - \left(\frac{q_1}{q_3}\right)q_2 + q_0 \; < \; 0$$

Therefore, at the points (a,b) of the locus $RH(a,b) = 0$, the system undergoes a Hopf bifurcation, with pulsation

$$\omega_H^2 = \frac{q_1}{q_3}$$

As it is easy to verify, if the pair (a,b) is such that both $0 < a << 1$ and $0 < b << 1$ then $R(a,b) < 0$, i.e. EE is LAS. Similarly, if both a and b are very large: $a,b >> 1$, then the EE is LAS. Thus the endemic equilibrium could only destabilized for intermediate values of a and b. Since $R(a,b)$ is a fourth–order polynomial in the variables $a > 0$ and $b > 0$, the exact shape of the instability region can be analytically determined by Cardano's formula for the solution of fourth-order algebraic equation. However, since this formula is cumbersome, we omit it. In the practice one has to resort to numerical computations, even in the case $a = b$.

To sum up, unlike the exponentially fading kernel, the acquisition-fading memory (and, probably, other unimodal kernels) can destabilize the endemic state of an SIR model with incidence-based social distancing. This in particular holds true for the special case of the second order Erlangian kernel, as is easy to check.

6. Incidence-based social distancing: an illustration based on the acquisition-fading memory

In this section we report a few examples of the working of the more interesting model presented here, namely the delayed model with acquisition-fading memory. The purpose of this section is primarily that of illustrating the potential richness of the model.

In these illustrations, we will resort to the following simple functional specification of $\beta(M)$

$$\beta(M) = \beta_0(1 - qM)_+$$

where $(x)_+$ equal to x if $x \geq 0$, and equal to 0 if $x < 0$.

This type of piecewise linear behavioral responses (used in [12,17]) postulates the annihilation of the risk of infection beyond some threshold incidence. The parameter q tunes the strength of the behavioral response. Moreover, we assume $\beta(0) > \mu + \nu$ to ensure the existence of an endemic state.

Finally, as information index we choose the reported incidence:

$$M = k\beta(M)SI$$

These hypotheses imply:

$$M(S, I) = \frac{kSI}{1 + qkSI}$$

and

$$\beta(M)SI = \frac{SI}{1 + qkSI}$$

In particular, the equation determining the endemic prevalence I_e is as follows

$$(1 - krI)(1 - \alpha I) = \frac{1}{\mathbb{R}}$$

i.e.

$$kr\alpha I^2 - (\alpha + kr)I + p_{cr} = 0$$

so that

$$I_e = \frac{(\alpha + kr) - \sqrt{\alpha^2 + k^2r^2 + 2\alpha kr(1 - 2p_{cr}))}}{2}$$

In order to assign the behavioral parameter q, we use the following argument. Consider a standard SIR model with standard incidence and constant contact rate equal to $\beta_0>0$. The corresponding endemic equilibrium (S_{sir},I_{sir}) is as follows

$$S_{sir} = \frac{1}{\mathbb{R}_0}$$

$$I_{sir} = (1 - S_{sir})\frac{\mu}{\mu + \nu}$$

Therefore, the endemic incidence reads as

$$J_{sir} = \beta_0 S_{sir} I_{sir}$$

Then, as regards q, since for $M=1/q$ it is $\beta(M) = 0.5\beta_0$, and since, in turn, β is assumed to be proportional to the incidence with proportionality factor k, we assumed:

$$q = \frac{1}{kfJ_{sir}} 1 - S_{sir}$$

with $f \in (0,1)$. Note that $q^{-1} = kfJ_{sir}$. In other words: the baseline transmission rate is halved when M is equal to a fraction f of the behavior–independent steady state incidence.

Last, we use the following values for the other parameters (time unit: days^{-1}): $mu = 1/(50 * 365.25)$, corresponding to a life expectancy of 50 years (reasonable for low-income settings), $\nu = 1/7$ corresponding to a duration of the infective phase of seven days, $\mathbb{R}_0$=15 (adimensional), a typical value of the basic reproduction number of measles, and consequently: $\beta_0 = \mathbb{R}_0(\mu + \nu)$. We obtain

$$I_e \approx 3.33 \times 10^{-5}$$

$$S_e \approx 0.913478$$

$$J_e = 4.73 \times 10^{-6}$$

The resulting stability boundary is plotted in the left panel of Fig. 1. The region where local stability prevails (i.e., the points (a,b) where $RH(a,b)<0$) is the clear one, while the instability region is the one in dark color. The stability region can be roughly approximated by the condition

$$a + b > w$$

where $w \approx 2 \times 0.0662$. However, in the previous section we noted that for small values of (a,b) the endemic equilibrium is LAS. And indeed, our numerical computations confirm that for tiny - possibly unrealistic - values of a and/or b the *EE* is again LAS, as shown (for the sake of the curiosity) in the right panel of Fig. 1. For example, for $b = 0.1a$, we obtained that the endemic state *EE* was LAs for $0 < a < 0.000015615$ (corresponding to acquisition delays of at least 175.33 years, a totally unrealistic duration) and, more interestingly, $a > 0.12043$. This threshold corresponds to approximately 8.3 days and a fading time of about 83, so that the minimal average delay capable to destabilize the system is in the range of ≈91 days. Fig. 2 reports the output of a simulation of the system over a period of 40 years, starting from the endemic equilibrium of the baseline SIR model in the absence of a behavioral response, i.e., from the initial condition:

$$(S(0), I(0), M_1(0), M_2(0)) = , (S_{sir}, I_{sir}, J_{sir}, J_{sir})$$

and setting $a = 0.05$/day (corresponding to an average delay of acquisition of information of $T_a = 20$ days) and $b = 0.005$, corresponding to a

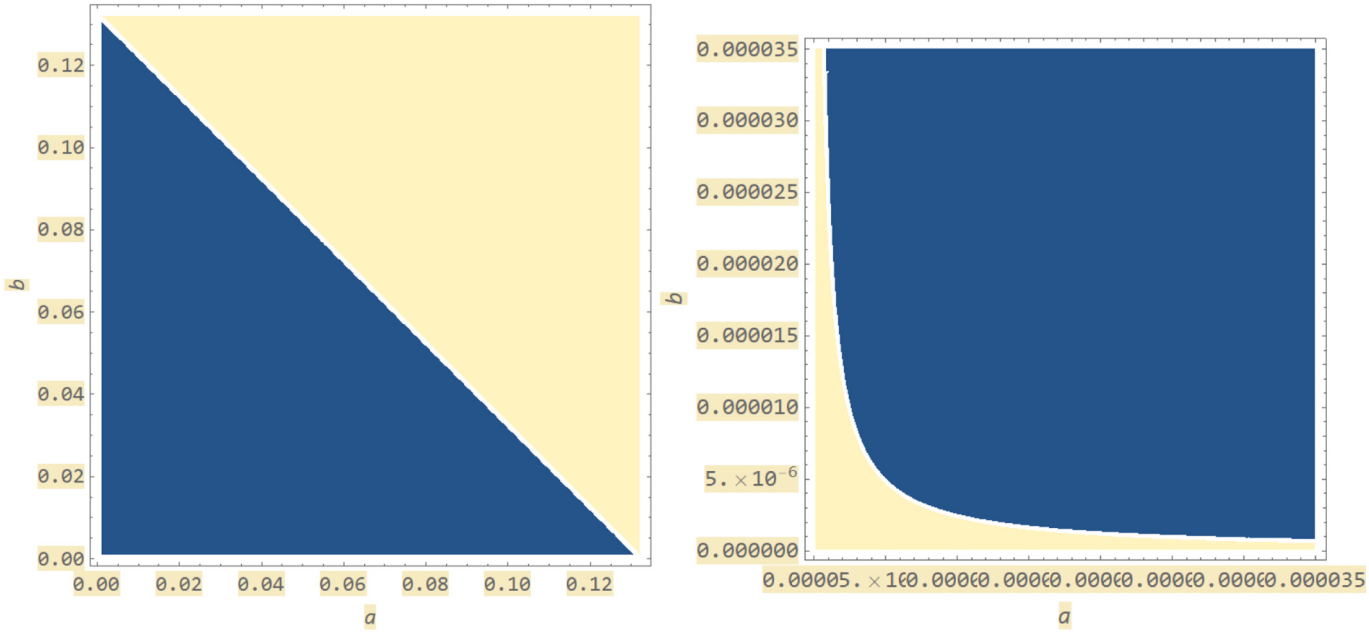

Fig. 1. The SIR model with incidence-based transmission response and acquisition-fading memory kernel: regions of local stability vs instability of the endemic state. Left Panel: the practical stability region (white) vs the instability region (dark blue), which can be approximated by the relationship $a + b \geq \omega$. Right panel: appearance of an additional (tiny) LAS region for tiny values of a and/or b, corresponding to unrealistically large T_a and/or T_b. Parameter values: as specified in the main text.

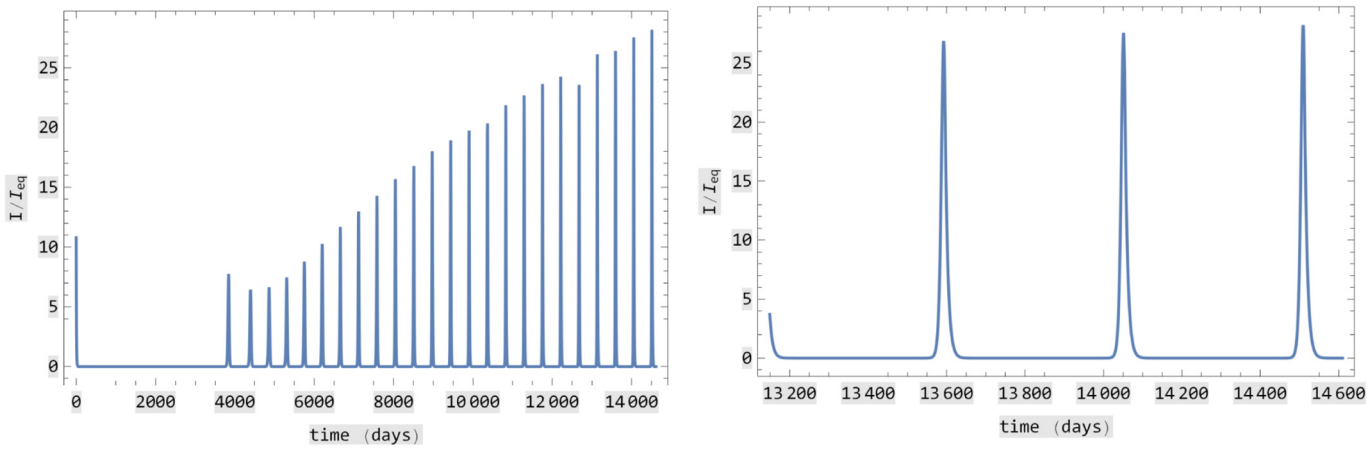

Fig. 2. The SIR model with incidence-based transmission response and acquisition-fading memory kernel temporal trend of the model for $a = 0.05$ and $b = 0.1a$ showing the onset of large oscillations with a period of about 450 days. Left Panel: trend of infective prevalence (normalized by the equilibrium prevalence I_e) from $t = 0$ to a max time of 40 years. Right panel: 'zoomed' trend of infective prevalence in the time interval from 36 to 40 years. Parametric values: as in specified in the text.

fading time of information of 200 days. This yielded the onset of large oscillations (left panel of Fig. 2) with a resulting long-term period of about 450 days (see zoom at the right panel of Fig. 2). The corresponding long-term amplitude was about 28 times larger than the value of the steady state prevalence I_e of the baseline SIR model.

Alternative choices of the delay parameters T_a, T_b are capable to bring oscillations with any desired inter-epidemic period and very large amplitude, in the spirit of [17].

7. The interplay between behavioral responses and seasonal forcing of the contact rate

In this section we briefly study the interplay between delayed behavioral responses and the presence of seasonality in the transmission rate. Seasonality in contact rate is one of the most important and studied topics in in the mathematical modeling of infectious diseases [31].

Assuming, for the sake of the simplicity, that the transmission rate is time periodic by means of a multiplicative periodic function $\psi(t)$ having period of one year and an average value equal to one

$$M = A(\psi(t)\beta(M)SI) \tag{40}$$

where we recall that $A'(u) > 0$. From Eq. (40) it follows that

$$M = \phi(\psi(t)SI)$$

yielding the following DAE system:

$$S' = \mu(1 - S) - A^{-1}(M) \tag{41}$$

$$I' = A^{-1}(M) - (\mu + \nu)I \tag{42}$$

$$M = A(\psi(t)\beta(M)SI) \tag{43}$$

For example, in the case, considered in the previous section, where $\beta(M) = \beta_0(1 - qM)$ one has that:

$$M(S, I, t) = \frac{k\psi(t)SI}{1 + qk\psi(t)SI}$$

In our simulations we set

$$\psi(t) = 1 + Q \sin\left(\frac{2\pi}{T}t\right)$$

where $0 < Q < 1$.

The effect of the seasonal oscillations can be very strong also in the short term of 40 years. Indeed, see Fig. 3, in 40 years the oscillations reach a peak amplitude of about $110I_e$ and the epidemic peaks are shifted of 800 days about. Extending the simulation up to 400 years the oscillations reach a peak amplitude of $\approx 200I_e$.

8. Concluding remarks

In this work we expanded the class of behavioral epidemiology models by considering endemic SIR models with incidence-based social-distancing. The idea is that individuals at risk of infection modify their social behavior in response to the available information on some appropriate function of the (current or past) incidence of the infection. In many cases, this is more realistic than the typical prevalence-based models routinely used in the behavioral epidemiology literature [12–14]. This leads, at variance with many epidemic models, to a differential–algebraic system.

We considered both current and delayed behavioral responses. As for the latter we considered two forms of the delaying kernel, namely the classical exponentially fading memory and an acquisition-fading memory that includes the 'classical' humped Erlang kernels as special cases.

Our results showed that the system has a unique endemic equilibrium. This endemic state was shown to be globally attractive when the behavioral response is based only on information on current incidence.

In presence of delayed behavioral responses, we showed that the endemic state (i) remains locally stable independently of the delay under exponentially fading memories, (ii) can be destabilized, by way of a Hopf bifurcation, in the presence of the more realistic acquisition-fading memory. The latter mechanism can induce recurrent behavior-induced epidemics without the need for seasonal forcing.

Finally, the inclusion of seasonality of the baseline contact rate did not induce chaos. However, it had a strong effect on the above–mentioned recurrent epidemics: the period of the solutions was remarkable increased, and the epidemic peaks were strongly amplified.

Although providing useful new pieces of information, our work has some limitations. First, it would be interesting to extend our behavior implicit formulation to a fully behavior explicit setting [12, 14]. Second, we proposed a deterministic model, which is only an adequate approximation in the case of a large population. Moreover we did not considered the possible presence of extrinsic stochastic perturbations, which can be affect both small and large populations. Further, the proposed model assumes that the population is homogeneously mixing. A spatial approach, especially based on a networked meta–population approach

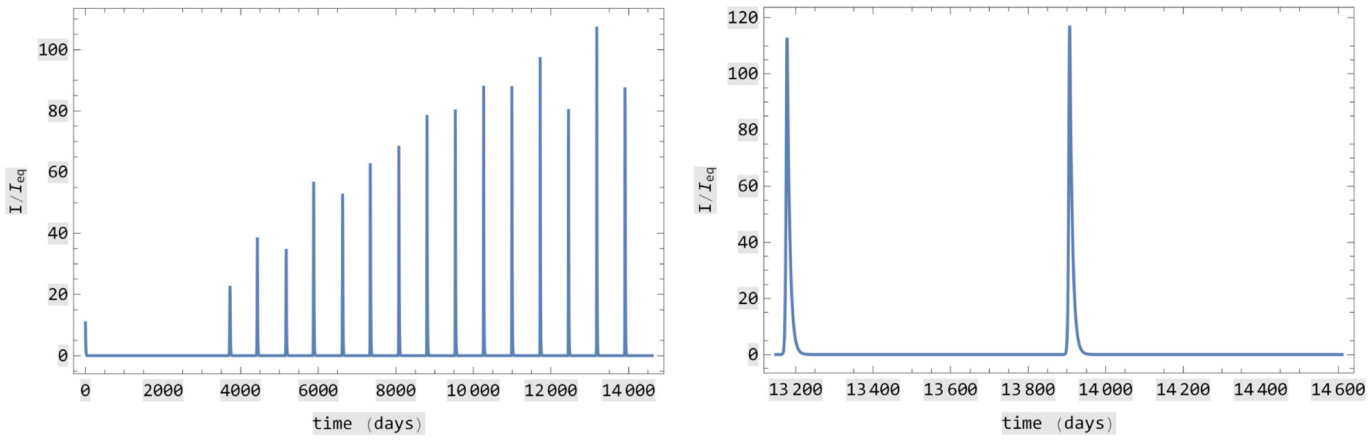

Fig. 3. The SIR model with incidence-based transmission response and acquisition-fading memory kernel: the interplay with seasonality in transmission. Here $\psi(t) = 1 + Q \sin(2\pi t/T)$ with $Q = 0.9$, T is equal to one year, $a = 0.05$ and $b = 0.1a$. Onset of very large oscillations of a period of about 800 days and of a peak amplitude $\approx 110 I_e$ (which running for a longer maximal time the simulation reaches a value $\approx 200 I_e$).Other parameters are specified in the text.

as in [13,32] would improve the applicability of the proposed model to real scenarios. Moreover, the networked approach could reveal emergent and topology–dependent features such as phase transitions, which are not possible in non–spatial models. An interesting example is, for example, the phase transitions observed in the vaccination–related behavioral epidemic model in [32]. Finally, a parallel modeling of the formation and spread of the information (see for example for other scenarios: [13,33]) would be of interest.

CRediT authorship contribution statement

Alberto d'Onofrio: Conceptualization, Methodology, Software, Formal analysis, Writing – original draft, Writing – review & editing. **Piero Manfredi:** Conceptualization, Methodology, Formal analysis, Writing – original draft, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper. We received no funds to conduct this research.

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