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RATIONAL EXEMPTION TO VACCINATION FOR NON-FATAL SIS DISEASES: GLOBALLY STABLE AND OSCILLATORY ENDEMICITY

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Abstract. 'Rational' exemption to vaccination is due to a pseudo-rational comparison between the low risk of infection, and the perceived risk of side effects from the vaccine. Here we consider rational exemption in an SI model with information dependent vaccination where individuals use information on the disease's spread as their information set. Using suitable assumptions, we show the dynamic implications of the interaction between rational exemption, current and delayed information. In particular, if vaccination decisions are based on delayed informations, we illustrate both global attractivity to an endemic state, and the onset, through Hopf bifurcations, of general Yakubovich oscillations. Moreover, in some relevant cases, we plot the Hopf bifurcation curves and we give a behavioural interpretation of their meaning.

1. **Introduction.** Vaccines were able to radically change the interaction between man and diseases, so that they became key factors in the increasing of the standards of living and of health [24]. The major example of this success is the eradication of smallpox [8].

The success of mass vaccination programmes critically depends on the degree of social adherence by individuals to the proposed vaccination. The history of many vaccinations shows that the progress toward increasing degrees of disease control is inter-mixed by episodes of coverage decrease due to the tension between public

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health targets and individual freedom, i.e. between compulsory vaccination and conscientious or philosophical exemption [28].

Moreover, modern societies are increasingly facing the more complex challenge of "rational" exemption, consisting in the family's decision to not vaccinate children as a consequence of a pseudo-rational comparison between the risk of infection and the risk of vaccine-related side effects. Thus, in these societies although a large proportion of the population is immune thanks to sustained vaccination, unfortunately vaccination is more and more on a voluntary basis. As a consequence it has been observed that "even the slightest risk associated with vaccination will outweight the risk from infection" [4]. In other words, we are facing the following paradox: it is the itself vaccination success in controlling diseases which, by making very low the perceived risk of infection, encourages 'rational' exemption behaviours, leading to coverage decline. Rational exemption is myopic since most often the decision to not vaccinate is based only on the current, transient regime of low incidence, forgetting that this low incidence is due to high vaccine coverage. Thus, it does not take into account the future resurgence of infection due to coverage decline. Moreover, it is of interest to note that this kind of exemption is an example of "free riding" [30]. Another paradox is given by the experimental evidences of these behaviours since surveys of vaccine attitudes suggest that these behaviours are particularly relevant among the richer and/or the more educated people [25], [32]. This fact, in our opinion, questions the role of schools and media in giving a general but correct scientific background in medicine to the general public: an educational effort might be very important.

An increasing amount of literature is currently investigating various aspects of these problems [3, 4, 5, 6, 16, 18, 27]. A common result of some of these papers, is that, under a voluntary vaccination policy, rational exemption makes eradication impossible, and may trigger childhood diseases to oscillate.

In [7, 11, 13] the dynamic implications of rational exemption were investigated using a standard SIR model with information-dependent vaccination. The key idea was that vaccination decisions are formed from an "information set" mostly based on the publicly available information on the disease. This led us to model vaccination coverage as a phenomenological function of the current and past state of the disease, analogous however to the game-funded function used in [27], defined as the sum of a steady component plus a variable that is positively correlated with perceived risk of infection.

In this paper we deep our investigation on rational exemption, by examining how it affects the dynamics of vaccine preventable diseases in the relevant case where the disease does not confer a permanent immunity to recovered subjects. In this first work we shall consider, to start, the simple case where the vaccine is able to confer a permament immunity.

As for the 'current' information case we show the conditions to have, under a constant transmission rate, a unique and globally stable endemic state. In addition we give two results on the persistence of the system under the more realistic case of periodic transmission. As for the "delayed" information case, we show the conditions under which vaccination choices based on past information trigger stable oscillations via Hopf bifurcations of the endemic state, as well as conditions to guarantee the global stability of the endemic equilibrium. The theoretical analysis is complemented by some numerical simulations. Some concluding remarks ends the work. An appendix collects all the proofs of our results.

2. **SIS model with information-dependent vaccination.** Here we consider the following family of SIS models for a non-fatal disease in a constant homogeneously mixing population, with state-dependent vaccination coverage by means of a fully effective vaccine providing life-long immunity:

$$S' = \mu\left(1 - p_0 - p_1(M)\right) - \mu S - \beta(t)SI + \gamma I \tag{1}$$

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

where S and I are functions of time respectively denoting the number of susceptible and infective individuals at time t; $\mu > 0$ denotes both the birth and death rate, which are assumed to be identical; $\gamma > 0$ is the rate of recovery from infection; $\beta(t) > 0$ the transmission rate, which is assumed to be constant or bounded and periodically varying with minimal period θ usually equal to one year [1].

Due to the non fatality of the disease and to the identity of the birth and death rate, the population is constant, and as a consequence, the dynamics of fraction of vaccinated subjects is simply given by:

$$V(t) = 1 - S(t) - I(t).$$

The novelty, introduced in [11], stands in the vaccination coverage at birth p which is defined as:

$$p(M) = p_0 + p_1(M) \qquad 0 < p_0 < 1,\ M \in \mathcal{I} \tag{3}$$

where M summarises how information on current and past states of the disease is used by families in deciding on whether to vaccinate or not their children. Thus, M can depend on the current or past state of the disease. Here we assume M to be a continuous function g of current or past values of S and I only, taking all the values of an interval $\mathcal{I} = [M_{\inf}, M^{\sup})$. In the "current" case we assume g increasing in I but not necessarily in S, with $g(S, 0) = 0$ for all S.

As regards current information, two noteworthy examples are $g(S, I) = kI$ meaning that individuals use the available information on the prevalence of the disease, and $g(S, I) = \alpha\beta(t)SI(\alpha > 0)$ where the reported incidence of the disease is taken into account.

An example of delayed information is:

$$g(S, I) = \int_{-\infty}^{t} kI(\tau)\, G(t - \tau)\, d\tau$$

where G is a delaying kernel, representing the weight given to past prevalence.

As regards p_1 we assume that $0 \leq p_1(M) \leq 1 - p_0$ for all $M \in \mathcal{I}$, $p_1(0) = 0$ and p_1 is continuous, differentiable, except at a finite number of points, and increasing.

The previous assumptions state that a fraction p_0 of the population is resilient to information/rumours and vaccinate their children whatever be the state of public information M, while a fraction p_1 vaccinate little in circumstances of low perceived risk, but "run to vaccinate" when the perceived risk from the disease goes up, according to the idea of rational exemption [11]. Thus in real situations we expect that p_1 is S-shaped and saturating to some level $p_1^{\text{sat}} = p_1(M^{\sup})$ less or equal than $1 - p_0$. The case $p_1(M) = p_1(g(I))$ is noteworthy as it can be justified by a game-theoretic approach [27].

Now, note that from the following differential inequality:

$$(S + I)' \leq \mu\left(1 - p_0 - (S + I)\right)$$

it easily follows that our model can be studied in the positively invariant and attractive set:

$$\Gamma^{\text{fra}} = \{(S, I) \mid \ S \geq 0,\ I \geq 0,\ S + I \leq 1 - p_0\}. \tag{4}$$

3. **The case of current information.** In this section we investigate the conditions for disease eradication and endemicity under the hypothesis that M depends on current information: $M = g(S, I)$, and we shall use the following function:

$$f(S, I) = p_0 + p_1\left(g(S, I)\right) \tag{5}$$

3.1. **Disease-free equilibrium and its properties.** Since $p_1\left(g\left(S, 0\right)\right) = 0$, it is easy to verify that the model (1)-(2) has the unique disease-free equilibrium:

$$\text{DFE} = (1 - p_0, 0). \tag{6}$$

Let $\overline{\beta} = \beta$, in case of constant contact rate, and otherwise:

$$\overline{\beta} = \frac{1}{\theta}\int_0^{\theta} \beta\left(u\right) du.$$

The following proposition clarifies that the stability properties of the DFE do not depend on $p_1(M)$:

Proposition 1. *Defining*

$$R_l = \frac{(1 - p_0)\overline{\beta}}{\mu + \gamma} \tag{7}$$

i) if $R_l \leq 1$ then the DFE is GAS in Γ^{fra};

ii) if $R_l > 1$ then the DFE is unstable.

Note that, in case of constant contact rate:

$$R_l = (1 - p_0)R_0,$$

where R_0 is the basic reproduction number of the SIS model in absence of vaccination [1].

3.2. **Endemic equilibrium: Existence, stability and cycles.** In this subsection we assume β constant. The key parameter is obviously R_l. We summarise the results on the existence of the endemic state in the following:

Proposition 2. *If $R_l > 1$ there is a unique endemic equilibrium $EE = (S_e, I_e)$, where $S_e = R_0^{-1}$ and I_e is the unique solution of the equation:*

$$1 - R_0^{-1} - I = f\left(R_0^{-1}, I\right) \tag{8}$$

The next proposition collects our results on the stability of the endemic state:

Proposition 3. *Under the assumptions of Proposition 2:*

i) if

$$-\frac{\partial f}{\partial S}(S_e, I_e) < 1 + \frac{\beta}{\mu} I_e \tag{9}$$

then the unique endemic equilibrium $EE = (S_e, I_e)$ is LAS;

ii) if $\forall (S, I) \in \Gamma^{fra}$

$$-\frac{\partial f}{\partial S}(S, I) < 1 + \frac{\beta}{\mu} I \tag{10}$$

then the EE is also GAS in Γ^{fra};

iii) if

$$-\frac{\partial f}{\partial S}(S_e, I_e) > 1 + \frac{\beta}{\mu} I_e \tag{11}$$

then (1)-(2) has at least one LAS limit cycle in Γ^{fra}.

Condition (9) implies that if $\partial g/\partial S > 0$ at the EE, then the endemic state is always LAS. Conversely if $\partial g/\partial S < 0$ the EE might be destabilised with the onset of oscillations, and this occurs if p_1 is rather steep at EE.

3.3. **Persistence under a periodic transmission rate.** In case of constant contact rate with $R_l > 1$, the disease is strongly persistent [31], i.e. it holds that:

Proposition 4. *If* $R_l > 1$ *then a costant* $c > 0$ *exists (which is called the constant of strong persistence) such that:*

$$\liminf_{t \to +\infty} I(t) > c > 0,$$

and,

$$\liminf_{t \to +\infty} S(t) > c > 0.$$

In fact, using the same arguments from Theorem 4.3 in [17] and Proposition 3.3 in [23], it follows that the necessary and sufficient conditions for strongly persistence is that the DFE is unstable, which is ensured by $R_l > 1$.
In case of periodically varying contact rate, intensive simulations for $R_l > 1$ suggest that this very important property is preserved. From the analytical point of view, the Proposition 5 below provides a constraint on $\beta(t)$ sufficient to have permanence. We prepare our result by the the following lemma showing that $\sigma = S + I$ is strongly persistent:

Lemma 3.1. *If* $\partial g/\partial S \geq 0$*, then there is a constant* $\widehat{\sigma} \in (0, 1 - p_0)$ *such that*

$$\liminf_{t \to +\infty} (S(t) + I(t)) \geq \widehat{\sigma}.$$

The next proposition provides a condition that, although very strong, ensures the strong persistence of the infective fraction

Proposition 5. *If the contact rate is periodic with:*

$$\beta_m = \min \beta(t) > \frac{\mu + \gamma}{\widehat{\sigma}} \tag{12}$$

then the infective fraction is strongly persistent and

$$\liminf_{t \to +\infty} I(t) \geq I_m := \frac{\beta_m \widehat{\sigma} - (\mu + \gamma)}{\beta_M},$$

where $\beta_M = \max \beta(t)$.

4. **Oscillations triggered by delayed information.** For many endemic infections publicly available information is the outcome of long routine procedures (laboratory confirmations, reporting delays to public health authorities, etc), and awareness of these phenomena to the general population takes time. Thus, more realistic formulations should include also past information. We now investigate the case

where the information function M is defined according to the following exponentially distributed-delay model:

$$M(t) = \int_{-\infty}^{t} g(I(\tau)) a e^{-a(t-\tau)} d\tau \tag{13}$$

where g is a generic function of I(in [11] we specifically considered the linear case). It holds:

$$M' = a\left(g(I) - M\right). \tag{14}$$

A common summary measure of the delay distribution is the mean delay $T = a^{-1}$. Embedding (14) in (1)-(2), we obtain:

$$\begin{aligned} S' &= \mu\left(1 - p_0 - p_1(M)\right) - \mu S - \beta(t) S I + \gamma I \\ M' &= a\left(g(I) - M\right) \\ I' &= \left(\beta(t) S - (\mu + \gamma)\right) I \end{aligned} \tag{15}$$

As far as eradication, instability of the DFE and, in case of constant contact rate, persistence and existence of an endemic equilibrium for the disease, it is easy to show that propositions similar to 1, 2 and 4 hold. However, the stability properties of the endemic equilibrium

$$EE = (S_e, M_e, I_e) = (S_e, g(I_e), I_e)$$

are different. In fact, here the delay may trigger the onset of steady general Yakubovich oscillations through a Hopf bifurcation [19] of the endemic state.

We recall here some basic definitions and results on Yakubovich oscillatority [14, 15]. Let us consider a a solution $x\left(t; x_0\right) \in R^n$ of an ODE system

$$x' = f(x), \tag{16}$$

where $f(x)$ is a continuous locally Lipschitz function $f : R^n \to R^n$. Thus, $x\left(t; x_0\right)$ is called a Yakubovich oscillation if it is such that for $i = 1, \ldots, n$

$$\liminf_{t \to +\infty} x_i(t) = \pi_i^-, \limsup_{t \to +\infty} x_i(t) = \pi_i^+$$

where:

$$-\infty < \pi_i^- < \pi_i^+ < +\infty.$$

If, apart the equilibrium points, for almost all x_0 the corresponding solution is a Yakubovich oscillation, then the system is called Yakubovich oscillatory. Moreover, in Corollary 1 of [15] it is shown that if all the equilibrium points of (16) are unstable and the orbits are bounded, then the system is Yakubovich oscillatory.

Thus, having established the above premises, we now may state the following:

Proposition 6. *If $R_l > 1$, then if and only if condition*

$$\mu\beta I_e f'(I_e) > \left(\mu + \beta I_e\right)^2 + 2\sqrt{\mu\beta I_e}\left(\mu + \beta I_e\right) \tag{17}$$

holds, there exist two values a_1 and a_2 with $0 < a_1 < a_2$ such that the endemic state is unstable for $a \in (a_1, a_2)$, whereas it is LAS for $a \notin [a_1, a_2]$. At the points a_1 and a_2 Hopf bifurcations occur. Moreover, in such a case, if $a \in (a_1, a_2)$ then the orbits $x(t) = (S(t), I(t), M(t))$ are oscillatory in the sense of Yakubovich.

Remark 1. Note that the application of the Hopf theorem only gives local informations limited to values of the bifurcation parameter that are sufficiently close to the bifurcation points (here: a_1 and a_2). The application of Yakubovich theory, on the contrary, gives us more global informations, although the nature (periodic, chaotic etc..) of the arising oscillations is not known.

Note the difference with the planar case: although one might use the Yakubovich theory also there, the Poincaré - Bendixson theory allowed us to obtain the far stronger result that it exists at least one LAS limit cycle, i.e. periodic oscillations. Thus, roughly speaking, one might consider the Yakubovich theorem as an extension to $n \geq 3$ (and, for DDE also for infinite dimensional systems) of the Poincaré - Bendixson theorem. However, in that extension the result is more limited since the type of the oscillations is undetermined.

Numerical simulations suggest that our results are global: when the endemic state is LAS it is also GAS, and when it exchanges its stability with a surrounding limit cycle, then the ensuing periodic orbit seems to be GAS.

In case of small and medium delays (i.e. comparable with the epidemic dynamics) it is possible to give an analytical background to the above numerical findings that the LAS of the EE also implies its GAS:

Proposition 7. *In the case $R_l > 1$, the EE is GAS in the set:*

$$\Omega = \left\{(S, I, M) | (S, I) \in \Gamma^{fra}, I > 0, M \leq g(1 - p_0)\right\}$$

provided that

$$\begin{gathered} \mu\Pi < a, \\ \beta(1 - p_0) + \gamma < \beta c + a \left(1 - \max_{I \in [c, 1-p_0]} (g'(I))\right), \end{gathered} \tag{18}$$

where c is the constant of strong persistence and $\Pi = Max_{M \in [c, g(1-p_0)]} p_1'(M)$.

Remark 2. Since c is not simple to establish, one also can use the following slightly stricter GAS criterion

$$\begin{aligned} a &> \mu\Pi, \\ a &> \frac{\beta(1 - p_0) + \gamma}{\left(1 - \max_{I \in [0, 1-p_0]} (g'(I))\right)}, \end{aligned} \tag{19}$$

5. **Illustrations on the delayed model.** The condition (17) suggests that instability of the endemic state requires that the slope of p_1 at equilibrium exceeds a prescribed threshold. This interpretation requires some attention because the r.h.s embeds the equilibrium values of I, which in turn depends on the actual form of p_1 (for a full discussion of the role played by p_1 on stability see [12]). With this caveat the ensuing epidemiological interpretation is appealing: the onset of oscillations require a violent and delayed vaccination response by individuals to epochs of high perceived risk [11].

Since the actual output of the model comes from the interplay of several different factors, in order to better illustrate the working of the delayed model, we make use of simplest assumption on p_1 and on $g(I)$. Namely, in our simulations we assume

that $g(I)$ is linear $g(I) = kI$, and that p_1 is as follows: *i)* linear: $p_1(M) = qM$; *ii)* linear with a threshold:

$$p_1(M) = q\,(M - kI_1)_+\,.$$

Of course, we set also the natural upper bound to $p_1(M)$ so that $f(I) < 1$. Hence, here we consider:

$$p_1(M) = Min\left(q\,(M - kI_1)_+\,, 1 - p_0\right),$$

where $I_1 \geq 0$.

These cases may allow to discuss in depth the structure of the bifurcation curve, providing feeling of what happens under more realistic circumstances. Moreover in those cases the endemic equilibrium may be analytically determined.

Our computations will be based on the following parameter constellation referring to pertussis [2]: $\mu = (1/L)$ days^{-1} where $L = 75 \times 365.25$ days is the life expectancy at birth; $\gamma = (1/\mathcal{D})$days^{-1} where $\mathcal{D} = 24$ days is the average duration of infection. Moreover we take either $R_0 = 10$ or $R_0 = 15$ where $R_0 = \beta/\left(\mu + \gamma\right)$ is the value that the basic reproduction number would take in absence of vaccination (thus for $R_0 = 10$ we have $\beta \approx 0.4169$days^{-1}).

In addition we take $p_0 = 0.75$.

Note that in a standard SIS model without information dependent coverage $R_0 = 10$ implies an endemic susceptible fraction $S_e = 0.1$, and critical coverage $p_c = 0.9$.

5.1. **Linear *f(I)*.** Preliminarly, we note that here both the endemic prevalence:

$$I_e = \frac{1 - \frac{1}{R_0}}{1 + qk} \tag{20}$$

and the characteristic polynomial are function of the product qk, thus it is useful to define the additional parameter $U = qk$. With the above values of parameters we obtain that there may be the onset of oscillations for

$$U > 112.152.$$

In figure 1 it is plotted the Hopf bifurcation curve (and, as a consequence, the instability and the local stability regions) as U versus the $Log_{10}(a)$. In figure 2 we show the behaviour of the ratio $I(t)/I_e$ for parametric values in the instability region.

Finally, concerning the global stability of the endemic equilibrium, the sufficient condition (19) reads here:

$$a > \frac{\beta(1 - p_0)}{1 - k} = \frac{0.1459}{1 - k},$$

and it is illustrated in figure 3.

5.2. **Linear with threshold.** Here we take $f(I) = qk(I - I_1)_+$. We note that I_e may be expressed as function of I_1 and $U = qk$:

$$I_e = \frac{1 - \frac{1}{R_0}}{1 + qk} + \frac{I_1}{1 + qk} = \frac{1 - \frac{1}{R_0}}{1 + U} + \frac{I_1}{1 + U}. \tag{21}$$

In figure 4 we show the Hopf curve in the plane (a, U) for $I_1 = 10^{-4}$ (dotted), $I_1 = 10^{-3}$ (solid) and $I_1 = 10^{-2}$ (dashed). Thus for increasing I_1 the region of instability reduces its size. Respectively, the minimum of the three curves are: 49.36, 56.29 and 149.41.

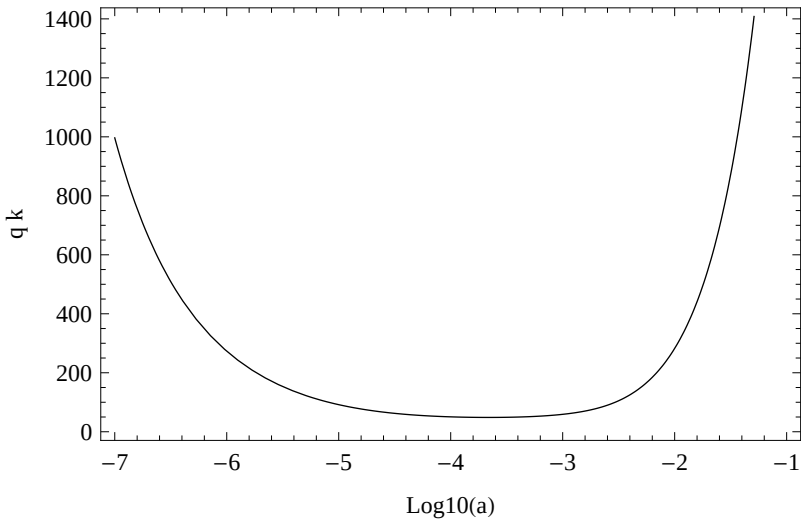

FIGURE 1. Hopf bifurcation curve in the space $(Log_{10}(a), U)$. The region over the curve corresponds to instability via Yakubovich-oscillations, whereas the region under the curve is the local asymptotic stability region. At the curve there is the occurrence of Hopf bifurcations. Other parameters as in the text.

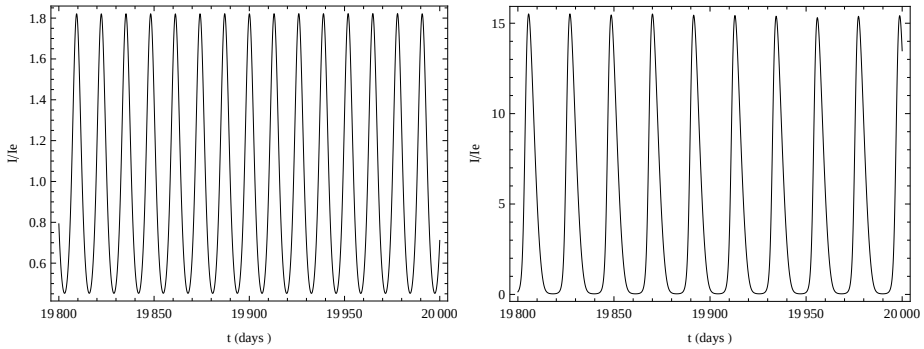

FIGURE 2. Examples of oscillations for parametric values lying in the instability region depticted in fig. 1. The left figure is for $a = 1/365.25$ (i.e. average memory lenght of one year), $q = 200$ and $k = 0.5$. The right figure is for $a = 1/365.25$, $q = 1000$ and $k = 0.5$. Other parameters as in the text.

6. **Concluding remarks.** This work has extended the investigation of the dynamic implications of information - dependent vaccination for SIR vaccine preventable infections started in [7, 11, 13], to the case of non-fatal SIS diseases.

When vaccination decisions are formed on the current information only, our results show the conditions to have a unique and globally stable endemic state in the case of a constant transmission rate, or to have the endemic permanence of the disease if the contact rate is periodic. These results confirm for SIS diseases the finding in [11, 13] that unless the steady component of vaccination is above the

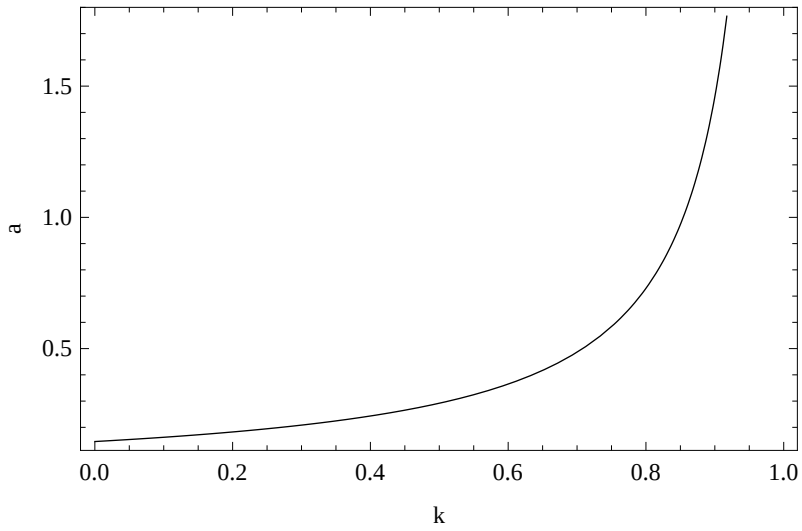

FIGURE 3. Global stability criterion (19) for the endemic equilibrium is fulfilled in points (k, a) in the region over the curve here plotted. Other parameters as in the text.

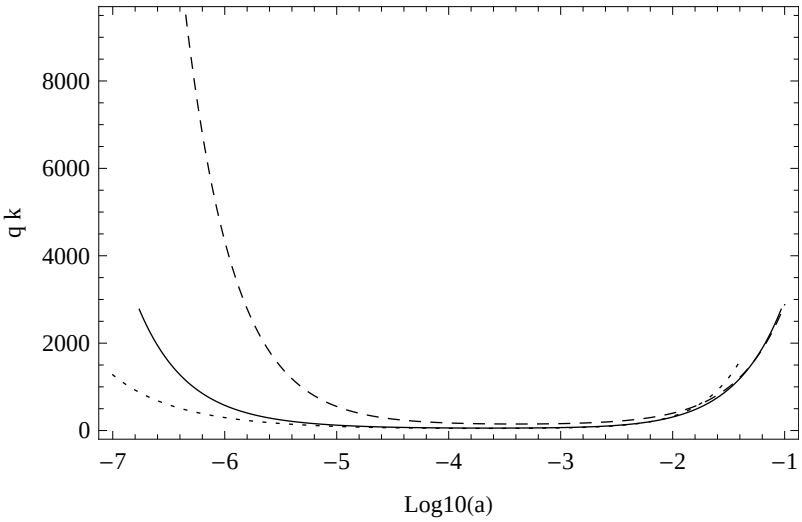

FIGURE 4. Linearly increasing $f(I)$ with threshold: $f(I) = qk(I - I_1)_+$. Hopf bifurcation curves in the space $(Log_{10}(a), U)$ for $I_1 = 10^{-4}$ (dotted), $I_1 = 10^{-3}$ (solid) and $I_1 = 10^{-2}$ (dashed). The region over the curve corresponds to Yakubovich-oscillations, whereas the region under the curve is the local asymptotic stability region. At the curve there is the occurrence of Hopf bifurcations. Other parameters as in the text.

critical elimination threshold, there is no hope to eliminate a disease even if during epochs of high social alarm coverage at birth could temporary achieve levels as high as 100%.

In the case of delayed information we were able to show both the global convergence to an endemic state in case of delays up to few days and, for intermediate delays, the existence of general steady "vaccination-exemption induced" oscillations of Yakubovich type, arising via Hopf bifurcation of the endemic state. Such oscillations are triggered by the prompt but not instantaneous reaction by people with 'memory of the epidemics' to the perceived risk from the disease. In simple words, oscillations appear when parents, in deciding on whether to vaccinate or not their children use the past, and not only the current, information about the disease and moreover tend to react violently to epochs of high perceived risk by promptly and significantly increasing the vaccination coverage.

As we remarked, the Yakubovich theory applied in proposition 6 does not give details on the nature (regular, irregular) of the oscillations. However, we stress here that the exact knowledge of the kind of oscilations in a first step is not essential from the epidemiologic point of view, where the major information is the knowledge that there will be a series of recurrent epidemics.

Then, we show by simulation the features of "vaccination-exemption induced" oscillations, including the regions of stability and instability.

In particular, the bifurcation curves for the case $p_1(M) = M$, $g(I) = kI$ that are plotted in figures 1 and 4 deserve a comment. The parameter k may be seen as a "summary" of two contrasting phenomena: *i)* the unavoidable under-reporting of disease prevalence; *ii)* the level of media and rumours coverage of the disease. Thus, we could decompose k as follows:

$$k = k_{underreporting} \times k_{media},$$

where in all cases $0 < k_{underreporting} \leq 1$, and where generally $k_{media} > 1$. Considering, realistically, constant and large q and, of course, a constant under-reporting parameter $k_{underreporting}$, we have that figures 1 and 4 show that the parameter k_{media} can easily cause the onset of recurrent epidemics, provided that it exceeds a threshold that is dependent on a, as we stressed in [7]. Here we note that the critical value $a = 1/365.25$ corresponding to a realistic average memory time $\theta = 1$ year lies near the minimum of the curves. Moreover, the lower part of that bifurcation curve is remarkably flat in logarithmic scale for a, which, roughly speaking means that the onset of oscillations is quite probable. We further note that for small average delays (large a) and for large average delays (small a) the bifurcation threshold for k_{media} sensibly increases. This might allow interesting considerations on the role of behaviour. However, the rise of the curves for small a appears at huge average memory times, far greater than real human memory. Thus we may say that for large but realistic θ the threshold only slightly increases. More interestingly, the plots show that short-range memory makes the parent less prone to change often their mind, thus they are less sensible to mediatic messages. This is a suggestive result, because it seems to us quite anti-intuitive, and it would deserve some further investigations on the interplay between media exposure of a disease, the perception of it by the general public of vaccinator and non-vaccinator parents and their 'memory of the disease'. Moreover, in case of large a one has also to take into account the small delays in the notification of cases, and an interesting question is: how to split the quite different contribution of memory and of notifications in the information index M?

A possible first rough solution might be the embedding of a constant delay in $g(I)$ in the equation for M. However, this would again be a phenomenological approach.

A challenging effort should be done to provide some mechanistic model of the onset of rational exemption as the resulting of the pseudo-rational choices of individuals with 'memory' that receive informations from media (and rumours).

Summarising the results of this work in comparison with our previous results on SIR model: here we performed a largely different numerical analysis that suggested some new epidemiologic results of some interest; in case of delayed information, we performed a global analysis in the general nonlinear case $g(I)$ (differently from [7] where we studied the special case $g(I) = kI$) and we obtained conditions for the GAS of the endemic equilibrium that are quite different from those of the SIR case [7]; we did not limit ourselves to the classical Hopf bifurcation analysis, but we perfomed a nonlocal study of the arising oscillations, via some recent results on Yakubovich theory. Of course, some results appear similar to those previously obtained by us for the SIR model, as it usually happens in mathematical epidemiology for reasons that are probably linked to biological robustness.

Finally, this is only a first research on rational exemption in SIS models, since here we assumed that the vaccine has no waning, whereas current vaccines for SIS diseases have finite immunization span [2]. However, since the aim of vaccinations is to confer a lifelong protection to the vaccinated subjects, we think that the present study might be of interest.

Appendix.

Proof of Proposition 1. The claim *i)* easily follows from the following differential inequality:

$$I' \leq I\left(\beta\left(t\right)\left(1-p_0-I\right)-\left(\mu+\gamma\right)\right) \tag{22}$$

Concerning claim *ii)*, it follows from the linearized equation at DFE for the infectious:

$$z' = z\left(\beta\left(t\right)\left(1-p_0\right)-\mu-\gamma\right).$$

□

Proof of Proposition 2. Setting $I' = 0$ in (2) and disregarding the solution $I = 0$, we easily obtain:

$$S_e = \frac{\gamma+\mu}{\beta} = R_0^{-1}.$$

Furthermore, setting $S' = 0$ in (1) we easily obtain the equation:

$$\mu\left(1-f\left(R_0^{-1},I\right)-R_0^{-1}\right)-\mu I = 0$$

implying the existence of a unique solution $I_e \in (0,1)$, i.e. the existence of a unique EE. □

Proof of Proposition 3. i) and iii) The characteristic polynomial of the linearized system at EE reads

$$\lambda^2+\left(\mu\frac{\partial f}{\partial S}(S_e,I_e)+\mu+\beta I_E\right)\lambda+\beta I_E\left(\mu\frac{\partial f}{\partial I}(S_e,I_e)+\mu\right)$$

The zero degeee coefficient is always positive, thus the LAS of EE only requires the positivity of the first degree coefficent, giving (9), whereas if this coefficient is negative (i.e. condition (11)) then at least a LAS limit cycle exists.

ii)The GAS of EE follows from Poincaré thrichotomy since the assumptions imply:

$$\operatorname{div}\left(\frac{S'}{I},\frac{I'}{I}\right)<0\ .$$

□

Proof of Lemma 3.1. By $\partial g/\partial S \geq 0$, since we are assuming $\partial g/\partial I > 0$, it holds:

$$f\left(S+I,S+I\right)>f\left(S,I\right).$$

Simple computations on (1)-(2) yield

$$(S+I)' \geq 1-(S+I)-f((S+I),(S+I)).$$

Thus, if $\widehat{\sigma}$ is the unique positive solution of

$$1-\sigma-f(\sigma,\sigma)=0$$

it easily follows the thesis. □

Proof of Proposition 5. From Lemma 3.1,

$$I' \geq I\left(\beta(t)\widehat{\sigma}-(\mu+\gamma)-\beta(t)I\right) \geq I\left(\beta_m\widehat{\sigma}-(\mu+\gamma)-\beta_M I\right)$$

implying that:

$$\liminf_{t\to+\infty} I(t) \geq \frac{\beta_m\widehat{\sigma}-(\mu+\gamma)}{\beta_M}>0.$$

□

Proof of Proposition 6. The Jacobian matrix at equilibrium point is

$$J\left(S_e,M_e,I_e\right)=\begin{pmatrix}-\mu-\beta I_e & -\mu p_1'(M_e) & -\mu\\ 0 & -a & +ag'(I_e)\\ \beta I_e & 0 & 0\end{pmatrix}$$

leading to the characteristic polynomial:

$$\lambda^3+b_2\lambda^2+b_1\lambda+b_0,$$

where

$$\begin{aligned}b_2&=a+\mu+\beta I_e>0\\ b_1&=a\left(\mu+\beta I_e\right)+\mu\beta I_e>0\\ b_0&=a\mu\beta I_e\left(1+p_1'(M_e)g'(I_e)\right)=a\mu\beta I_e\left(1+f'(I_e)\right)>0\end{aligned}$$

We use a as a bifurcation parameter. Note that the coefficients b_i may be written as follows:

$$b_2=a+q_2;\qquad b_1=aq_2+q_1;\qquad b_0=a(r_0+q_1)$$

with $q_1,q_2,r_0>0$. The positivity of b_i by Descartes theorem, rules out the possibility of real positive eigenvalues, so that stability losses can only occur via Hopf bifurcations.

From Routh-Hurwitz theorem, EE will be LAS if and only if $b_2b_1-b_0>0$, equivalently written as:

$$f(a)=q_2a^2+(q_2^2-r_0)a+q_1q_2. \tag{23}$$

Thus if $q_2^2-r_0\geq 0$ then EE is LAS independently of the delay, but if $r_0>q_2^2$ instability is possible. Note that since $f(0)>0$, $f(\infty)>0$ the endemic equilibrium is however always LAS for both small or large values of the delay parameter a, i.e. for large mean delays ($T=1/a\to+\infty$) and for small mean delays ($T=1/a\to 0$).

Thus stability continues to prevail if the discriminant Δ of $f(a)$ is negative or null, whereas if $\Delta > 0$, i.e. if:

$$\Delta = (r_0 - q_2^2 - 2q_2\sqrt{q_1})(r_0 - q_2^2 + 2q_2\sqrt{q_1}) > 0,$$

there are two positive and distinct solutions a_1 and a_2 for the equation $f(a) = 0$, i.e. two meaningful bifurcation values for the delay parameter a. Taking into the account $r_0 > q_2^2$, it yields:

$$r_0 > q_2^2 + 2q_2\sqrt{q_1} = q_2(q_2 + 2\sqrt{q_1}),$$

i.e. the bifurcation condition (17).

As far as the test for nonzero speed both a_1 and a_2 fulfill the test, which, for a third order characteristic polynomial, reads [19]:

$$Sign\left(Real\left(\frac{d\lambda}{da}\mid_{a_i}\right)\right) = Sign\left(\frac{d}{da}(b_1b_2 - b_0)\mid_{a=a_i}\right) = \mp\frac{\Delta}{4} \neq 0.$$

Finally, as far as the Yakubovich oscillatority, since: *i)* the orbits are bounded; *ii)* the endemic equilibrium $E_e(S_e, I_e, g(I_e))$ is unstable; *ii)* the DFE equilibrium is as well unstable and it has as stable manifold the line $(w, 0, 1)$ $w \in [0, 1]$ to which E_e does not belong, thus excluding heteroclinic orbits; *iv)*The orbits are strongly persistent. As a consequence [14, 15] it follows our claim. □

Proof of Proposition 7. We start this proof by shortly summarising the Li - Muldowney geometric theorem for a system of ordinary differential equations. Consider the autonomous dynamical system:

$$\dot{x} = f(x), \tag{24}$$

where $f : D \to \mathbf{R}^n$, $D \subset \mathbf{R}^n$ open set and simply connected and $f \in C^1(D)$. Let x^* be an equilibrium of (24), i.e. $f(x^*) = 0$.

In the paper [22] it has been shown that x^* is globally asymptotically stable in D provided that the following hypothesis are satisfied:
$(H1)$ there exists a compact absorbing set $K \subset D$;
$(H2)$ the equation (24) has a unique equilibrium x^* in D.
$(H3)$ A function P(x) and a Lozinskiĭ measure L exist such that the following inequality:

$$\limsup_{t\to\infty} \sup_{x_0\in\Gamma} \frac{1}{t}\int_0^t L(B(x(s, x_0)))ds < 0, \tag{25}$$

is satisfied.

In (25) B is given by:

$$B = P_f P^{-1} + PJ^{[2]}P^{-1},$$

where $P(x)$ is a $\binom{n}{2} \times \binom{n}{2}$ matrix-valued function that is C^1 on D and where the matrix P_f is

$$(p_{ij}(x))_f = (\partial p_{ij}(x)/\partial x)^T \cdot f(x) = \nabla p_{ij} \cdot f(x),$$

The matrix $J^{[2]}$ is the second additive compound matrix of the Jacobian matrix J, i.e. $J(x) = Df(x)$. L represents the Lozinskiĭ measure of B with respect to a vector norm $|\cdot|$ in $\mathbf{R}^N$, $N = \binom{n}{2}$, i.e.

$$L(B) = \lim_{h\to 0^+} \frac{|I + hB| - 1}{h}.$$

The function P and the vector norm in $\mathbf{R}^N$ must be suitably chosen.

Coming now to the study of the GAS of the endemic equilibrium, we start noticing that from the following differential inequality:

$$(S+I)' \leq \mu(1-p_0-S-I)$$

it follows that $\Gamma^{fra} \times [0,+\infty)$ is positively invariant for (15). Furthermore, from:

$$-aM \leq M' \leq a\,(g(1-p_0)-M)$$

it follows that also Ω is positively invariant.

Now, system (15) under the assumption $R_l > 1$, satisfies conditions (H.1)-(H.2). In fact, as we noticed in section 4, also in the delayed case, similarly to subsection 3.3, the condition $R_l > 1$ ensures not only the instability of DFE but also the strong persistence of the disease, i.e. there exists a constant $c > 0$ such that any solution $(S(t), M(t), I(t))$ with $(S(0), M(0), I(0))$ in the interior of Ω, satisfies:

$$\min\{\liminf_{t\to\infty} S(t), \liminf_{t\to\infty} M(t), \liminf_{t\to\infty} I(t)\} > c.$$

The strong persistence together with boundedness of Ω, is equivalent to the existence of a compact set in the interior of Ω which is absorbing for (15), see [20]. Thus, (H.1) is verified. Moreover, EE is the only equilibrium in the interior of Ω, so that (H.2) is also verified.

It remains to find conditions for which the Bendixson criterion given by (25) is verified.

By introducing the variable $Z = S + I$, model (15) becomes:

$$\begin{array}{l} Z' = \mu\,(1-p_0-p_1(M)) - \mu Z \\ M' = a\,(g(I)-M) \\ I' = \beta I\,(Z-I) - (\mu+\gamma)\,I \end{array} \tag{26}$$

The Jacobian matrix $J(Z, M, I)$ corresponding to (26) is:

$$J = \begin{pmatrix} -\mu & -\mu p_1'(M) & 0 \\ 0 & -a & ag'(I) \\ \beta I & 0 & \beta Z - 2\beta I - (\mu+\gamma) \end{pmatrix};$$

and the second additive compound matrix $J^{[2]}(Z, M, I)$ is:

$$J^{[2]} = \begin{pmatrix} -\mu - a & ag'(I) & 0 \\ 0 & -\mu + \beta Z - 2\beta I - (\mu+\gamma) & -\mu p_1'(M) \\ -\beta I & 0 & -a + \beta Z - 2\beta I - (\mu+\gamma) \end{pmatrix}.$$

Now we take the function,

$$P = P(Z, M, I) = diag\left\{\frac{Z}{I}, \frac{Z}{I}, \frac{Z}{I}\right\}. \tag{27}$$

It follows,

$$P_f P^{-1} = \text{diag}\left\{\frac{Z'}{Z} - \frac{I'}{I}, \frac{Z'}{Z} - \frac{I'}{I}, \frac{Z'}{Z} - \frac{I'}{I}\right\},$$

and, $PJ^{[2]}P^{-1} = J^{[2]}$, so that,

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

where $B_{11} = \frac{Z'}{Z} - \frac{I'}{I} - \mu - a$, $B_{12} = \left[\begin{array}{cc} ag'(I), & 0 \end{array}\right]$, $B_{21} = \left[\begin{array}{cc} 0, & -\beta I \end{array}\right]^T$, and,

$$B_{22} = \left[\begin{array}{cc} -\mu + \Psi & -\mu p_1'(M) \\ 0 & -a + \Psi \end{array}\right],$$

where:

$$\Psi = \frac{Z'}{Z} - \frac{I'}{I} + \beta Z - 2\beta I - (\mu + \gamma).$$

Consider now the norm in $\mathbf{R}^3$ as:

$$|(u, v, w)| = \max\{|u|, |v| + |w|\}, \tag{28}$$

where (u, v, w) denotes the vector in $\mathbf{R}^3$ and denote by L the Lozinskiĭ measure with respect to this norm. It follows, [26]:

$$L(B) \leq \sup\{g_1, g_2\} \equiv \sup\{L_1(B_{11}) + |B_{12}|,\ L_1(B_{22}) + |B_{21}|\}, \tag{29}$$

where $|B_{21}|$, $|B_{12}|$ are matrix norms with respect to the L^1 vector norm and L_1 denotes the Lozinskiĭ measure with respect to the L^1 norm[1].

$$L_1(B_{11}) = \frac{Z'}{Z} - \frac{I'}{I} - \mu - a, \tag{30}$$

By recalling that $g(I)$ is an increasing function,

$$|B_{12}| = ag'(I), \tag{31}$$

$$|B_{21}| = \beta I, \tag{32}$$

$$L_1(B_{22}) = \frac{Z'}{Z} - \frac{I'}{I} + \beta Z - 2\beta I - (\mu + \gamma) + \max\{-\mu; -a + |-\mu p_1'(M)|\}. \tag{33}$$

Taking into account of (29) and (30)-(33), the general expressions of g_1 and g_2 for system(26) are thus:

$$g_1 = \frac{Z'}{Z} - \frac{I'}{I} - \mu - a + ag'(I), \tag{34}$$

and

$$g_2 = \frac{Z'}{Z} - \frac{I'}{I} + \beta Z - \beta I - (\mu + \gamma) + \max\{-\mu; -a + |-\mu p_1'(M)|\}. \tag{35}$$

Observe that system (26) provides the following equality:

$$\frac{\dot{I}}{I} = \beta(Z - I) - (\mu + \gamma), \tag{36}$$

hence, from (34) one gets,

$$g_1 = \frac{Z'}{Z} - \beta Z + \beta I + \gamma - a + ag'(I), \tag{37}$$

and, from (35),

$$g_2 = \frac{Z'}{Z} + \max\{-\mu; \mu p_1'(M) - a\}.$$

Hence, from (29),

$$\begin{aligned} L(B) \quad &\leq \sup\{g_1, g_2\} = \\ &= \frac{Z'}{Z} + \max\{-\beta Z + \beta I + \gamma - a + ag'(I);\ \ -\mu;\ \ \mu p_1'(M) - a\}, \end{aligned}$$

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

i.e.

$$L(B) \leq \frac{Z'}{Z} + \max\left\{-\beta c + \beta(1-p_0) + \gamma - a\left(1 - \max_{I\in[\epsilon,1-p_0]}(g'(I))\right); -\mu; \mu\Pi - a\right\},$$

where c is the constant of strong persistence and $\Pi = Max_{M\in[c,g(1-p_0)]}p_1'(M)$.

Now, impose that:

$$\begin{gathered}\mu\Pi < a,\\ \beta(1-p_0) + \gamma < \beta c + a\left(1 - \max_{I\in[\epsilon,1-p_0]}(g'(I))\right).\end{gathered} \tag{38}$$

This allows to conclude that:

$$L(B) \leq \frac{Z'}{Z} - \omega,$$

where,

$$\omega = \min\{\beta c - \beta(1-p_0) - \gamma + a\left(1 - \max_{I\in[\epsilon,1-p_0]}(g'(I))\right);\ \ \mu;\ \ a - \mu\Pi\}, \text{ and } \omega > 0.$$

Hence

$$\frac{1}{t}\int_0^t L(B)ds \leq \frac{1}{t}\log\frac{S(t)}{S(0)} - \omega,$$

and the Bendixson criterion given by (25) is thus verified. □

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