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Information-related changes in contact patterns may trigger oscillations
in the endemic prevalence of infectious diseases.

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Abstract

It is well known that behavioral changes in contact patterns may significantly affect the spread of an epidemic outbreak. Here we focus on simple endemic models for recurrent epidemics, by modelling the social contact rate as a function of the available information on the present and past disease prevalence. We show that social behaviour change alone may trigger sustained oscillations. This indicates that human behavior might be a critical explaining factor of oscillations in time-series of endemic diseases. Finally, we briefly show how the inclusion of seasonal variations in contacts may imply chaos.

Key words: Behavior, Hopf Bifurcations, force of infection, SIR models.

1 Introduction

The prevailing wisdom suggests that besides major factors, such as the sanitary revolution, or the discovery of vaccines, the role of men in affecting the disease's dynamics has always been a minor one. This is mirrored by the amazing simplicity of the role paid to humans in models of diseases. However, the threat posed by the possibility of a pandemic of avian flu, and the related need to develop predictive and control tools, has clearly indicated that while we dispose of richer and richer models, we are still poor in the understanding of human behavior (Ferguson 2007). Though serious progresses have been recently made, for instance for the first time we dispose of a body of standardised international data on social contact patterns (Mossong et al. 2008), yet these data give a static picture of the contacts an individual has in one average day. This fact - human behavior as static - is postulated in most models of both endemic and epidemic diseases, but is clearly a coarse abstraction. Individuals are neither static nor passive: they elaborate the available information and can change their social behavior to respond to changes in their perceived risk. Symptoms of behavioral change were evident during the pandemic of Spanish flu

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(Epstein et al. 2007). A recent, well documented instance of individual's behavior change in response to an epidemic outbreak is given by the dramatic decline in travels and social contacts during the 2003 SARS epidemics in Hong Kong and Singapore (Ferguson 2007). Large behavioral changes were observed in the S. Francisco homosexual community as a response to the spread of HIV (McKusick et al. 1985). Similarly the control of HIV/AIDS in some low resources settings, for instance the Uganda 'success story' (Green et al. 2006), has mainly been achieved thanks to dramatic changes in sexual behaviour, which are now documented in other Sub-Saharan Africa contexts as well (Gregson et al. 2006).

The onset of the bioterrorism scare and of SARS have given great impulse to improve our understanding of the possible effects of the responses by the public to epidemic threats in the globalisation era (Ferguson et al. 2003, Chowell et al. 2004, Del Valle et al. 2005). The increased risk of a H5N1 pandemic flu has definitively put better modelling of human behavior, particularly the individuals' response to an epidemic threat, at the top of the current research agenda (Ferguson 2007). A variety of epidemic models including 'thinking agents' who elaborate the available information, and consequently strategically adapt their social behaviour not only to changing epidemiological conditions but also to changes in other agents' behaviour, have been proposed, e.g. (Bauch et al. 2003, Vardavas et al. 2006, Breban et al. 2007, Epstein et al. 2007). This is a long way from the first epidemiological model dealing with behavioral changes during an epidemic, the SIR epidemic model by Capasso and Serio in seventies (Capasso and Serio 1978, Capasso 2008). **Capasso and Serio allowed the contact rate β , until then taken as constant, to be a decreasing function of the disease prevalence (i.e. the infective fraction in the total population) I . This implies that the Force of Infection (FoI), i.e. the per-capita rate at which susceptible individuals acquire the infection per unit of time, takes the following non-linear form (Capasso 2008):**

$$FoI(I) = \beta(I)I \quad (1)$$

with: $\beta'(I) < 0$. Capasso and Serio pointed out that, differently from standard mass action formulations, this could make the FoI to become a non-monotone function of the prevalence (e.g. if $\beta(I) = \beta_0(1+hI^2)^{-1}$). Capasso and Serio motivated their formulation with behavioral changes: in epochs of high prevalence the perceived risk of infection might become very large yielding dramatic changes in individuals' behavior, therefore also reducing the actual risk of getting the disease. Since that seminal paper, several other works have been devoted to epidemic models with a non-linear FoI (Liu et al. 1986, van den Driessche and Watmough 2000, Ruan and Wang 2003, Alexander and Moghadas 2004, Wang 2006, Zhou et. al. 2007), in order to mirror the existence of some degree of change in contact patterns. **However, in some works (Liu et al. 1986, Alexander and Moghadas 2004, Wang 2006, Zhou et. al. 2007) (and references therein) it is assumed that contact rate is an increasing function of I in all $\leq I \leq 1$ (Liu et al. 1986, Alexander and Moghadas 2004), or that it is increasing in an initial interval $0 \leq I \leq \bar{I} < 1$ and then decreasing (Wang 2006, Zhou et. al. 2007). It is worth of note that in these cases multiple equilibria and oscillations, through Hopf bifurcations of endemic states, may arise. We stress, however, that local and global increases of the contact rate (for their biological roots, see (Liu et al. 1986)) can hardly be related to social phenomena, such as behavioural change, which is the focus of our paper. Indeed it would not be epidemiologically realistic to assume that susceptible subjects do intensify their contacts patterns as the prevalence of the disease increases.**

Most investigations of human behavior in relation to diseases dynamics have however been carried out in relation to STDs, particularly HIV, motivated by the need to understand the possible impact of information campaigns on sexual behavior, and as a consequence on infection trends. In particular, the long time scales of HIV/AIDS have motivated the study of the role of behavioral changes not only in

relation to epidemic control but also under endemic conditions. Most such papers have incorporated behavioral changes extending in various ways the phenomenological approach à la Capasso-Serio (e.g., among the first papers appeared on the subject, (Velasco-Hernandez et Al. 1996)), but there have also been some works including the individual's behavioral choice within HIV diffusion models (Kremer 1996).

On the other hand no investigation has been carried out on the impact of behavioral changes on the dynamics of common endemic close-contacts diseases, such as measles. This is surprising given that, despite the anecdotic importance paid to pandemics (e.g. of plague), the history, and growth, of mankind has largely been regulated by devastating recurrent epidemics of essentially endemic diseases such as smallpox, typhoid fever, or measles. In the absence of any knowledge on the diseases' etiology and of therapies, measures of social distancing and behavioral changes were the only walls against the fatal impact of these diseases. Though most available documentation regards especially social distancing, as for the plague, there is evidence that behavioral changes also played a role, as documented in historical, anthropological and social medicine studies (Fassin 1986, Halverson 2007).

Now actual forms and extent of such behavioral changes, i.e. the relative importance of quarantine vs absenteeism (to assist sick people) vs reduced contact rates vs more hygienic practices - are probably largely dependent on the social, say developed vs developing countries or urban vs rural areas, and historical context considered. Nonetheless we speculate that, differently from big but largely spaced epidemics, where knowledge of the disease impact could only be anecdotic, already in historical times the adopted mechanisms of behavioral changes for endemic periodic diseases could have been based on the available information on the state of the disease, filtered through the knowledge about it, acquired through the social and family history during past epidemics.

Consistently with this idea in this paper we study a new simple model for the dynamics of recurrent endemic diseases with behavioral changes, where the contact rate is a function of some information index M summarising the current and past history of the disease prevalence:

$$F \circ I(M) = \beta(M)I, \quad (2)$$

where $\beta'(M) < 0$, and where M is related to past prevalence through a suitable function $g(\cdot)$ as follows:

$$M(t) = \int_0^{+\infty} g(I(t - \tau))\varrho(\tau)d\tau. \quad (3)$$

where $\varrho(t)$ is a delaying kernel (MacDonald 1989, Farkas 1994) **i.e. a positive function such that:** $\int_0^{+\infty} \varrho(\tau)d\tau = 1$. The chosen approach, based on a phenomenological modelling of behaviour change, is a first step toward more involved modelling of social behaviour change that has the advantage of providing simpler and clearcut mathematical results.

A similar approach has been recently used to model a different behavior-related phenomenon: the social acceptance of vaccination programmes and rational exemption (Reluga et al. 2006, d'Onofrio et al. 2007, d'Onofrio et al. 2008, Buonomo et al. 2008). In this case the vaccination coverage has been represented as an increasing function of M .

The fact to relate current contact patterns to the delayed information on the disease, is realistic also in today's world since for most diseases information is made available after articulated routine procedures (e.g. laboratory confirmations), but it was central in historical times due to the low speed of information spread on the geographic scale.

2 The model

Consistently with our aims, we investigate the implications of our information-dependent FoI on a basic Susceptible-Infectious-Removed (SIR) model with vital dynamics (Capasso and Serio 1978):

$$S' = \mu(1 - S) - \beta(M)IS \quad (4)$$

$$I' = \beta(M)IS - (\mu + \nu)I \quad (5)$$

completed by eq. (3), governing the dynamics of M , and by the balance equation of the removed fraction $R(t)$: $R(t) = 1 - S(t) - I(t)$. The parameters μ and ν denote the general mortality rate and the removal rate from the disease, respectively.

As for $g(I)$, one might take any function such that $g(0) = 0$ and $g'(I) > 0$. The simplest example is $g(I) = kI$ where $k \in (0, 1)$ which could represent for instance the actually reported incidence of serious case of the disease. Another example could be (d'Onofrio et al. 2007, d'Onofrio et al. 2008, Buonomo et al. 2008); $M = wI/(1+qI)$ where M is a non-linear increasing function of the disease prevalence which can be taken as a measure of the perceived, rather than actual, risk of infection (Reluga et al. 2006, d'Onofrio et al. 2007, d'Onofrio et al. 2008, Buonomo et al. 2008).

As for the delay kernel $\varrho(\tau)$, some noteworthy cases are the following (δ is the Dirac's delta function): *i*) the no-delay case: $\varrho(\tau) = \delta(\tau)$; *ii*) the fixed delay: $\varrho(\tau) = \delta(\tau - T)$, with $T > 0$; *iii*) the Erlang distribution of order n :

$$\varrho(\tau; a) = \frac{a^n}{(n-1)!} \tau^{n-1} e^{-a\tau}, \quad (6)$$

where the mean delay is given by $T = n/a$ and the standard deviation is $\sigma = T/\sqrt{n}$. This model has been largely used in literature (MacDonald 1989, Farkas 1994, Anderson and Watson 1980, Grossman et al. 1999, Lloyd 2001, Keeling and Grenfell 2002, d'Onofrio 2004) since it is more realistic than the case of constant delay, and it allows a finite dimensional reduction of the integral equation (3) to the following n ODEs:

$$\begin{aligned} M_1' &= a(g(I) - M_1) \\ M_j' &= a(M_{j-1} - M_j), \quad j = 1, \dots, n-1, \end{aligned} \quad (7)$$

where $M = M_n$. The case $n = 1$ corresponds to the exponentially fading kernel, the case $n = 2$ to the so called strong kernel (MacDonald 1989, Farkas 1994), and the case $n \rightarrow +\infty$ implies $\rho(\tau, n/T) \rightarrow \delta(t - T)$.

2.1 The Basic Reproduction Number

It is possible to define a basic reproduction number (BRN) (Capasso 2008) **for our model**:

$$\mathfrak{R}_0 = \frac{\beta(0)}{\mu + \nu}, \quad (8)$$

which may be interpreted as the average number of secondary cases caused by a single infectious individual entering a fully susceptible population that has no information on the disease.

In this section we shall show how this parameter influences the behavior of the model in case of generic delay kernel. In fact, it is easy to verify that if $\mathfrak{R}_0 \leq 1$ then:

$$\lim_{t \rightarrow +\infty} (S(t), I(t)) = (1, 0), \quad (9)$$

i.e. the disease-free state (DFS) is Globally Asymptotically Stable (GAS). This result immediately follows from the differential inequality:

$$I' \leq I(\beta(0)(1 - I) - (\mu + \nu)).$$

Moreover, since the linearized equation for I is:

$$i' = i(\beta(0) - (\mu + \nu)),$$

it is easy to show that if $\mathfrak{R}_0 > 1$ then the DFS is unstable and it exists a unique endemic state $EE = (S_e, I_e)$ where $S_e = (\mu + \nu)/\beta(g(I_e))$ and $I_e > 0$ is the unique solution of:

$$\mu \left(1 - \frac{\mu + \nu}{\beta(g(I))} \right) - (\mu + \nu)I = 0. \quad (10)$$

Moreover, in case of Erlang kernel, we demonstrate in the appendix that if $\mathfrak{R}_0 > 1$ then the disease is strongly persistent.

Summing up, \mathfrak{R}_0 indicates that the capability of the disease to invade the host population or to go extinct is governed by the baseline behavior in absence of information on the disease spreading, independently on the structure of the memory kernel.

3 Oscillations around the Endemic Equilibrium

As we showed in the previous section, the existence and stability of DFS is independent of the delay properties, as well as the existence and location of the endemic equilibrium. On the contrary, the stability properties of the EE critically depend on $\varrho(\tau)$.

3.1 Analytical results

We notice first that in the no-delay case, **, i.e. when the information index M is related to the current prevalence $M = g(I)$, if $\mathfrak{R}_0 > 1$ then the endemic state is GAS, as it was previously obtained by (Wang 2006), although without modelling the information.** In fact EE is LAS and since:

$$\text{div} \left(\frac{S'}{I}, \frac{I'}{I} \right) = -\frac{\mu}{I} - \beta(g(I)) + g'(I)\beta'(g(I)) < 0$$

the Dulac's criterion (see the appendix of (Capasso 2008)) rules out the possibility of closed orbits. Second, in the case of the exponentially fading kernel, **which leads to the following three-dimensional family of models:**

$$S' = \mu(1 - S) - \beta(M)IS \quad (11)$$

$$I' = \beta(M)IS - (\mu + \nu)I \quad (12)$$

$$M' = a(g(I) - M) \quad (13)$$

for which some lengthish algebra shows (see appendix) that independently of the specific functional forms of g and β the endemic state is (at least) locally asymptotically stable (LAS).

However, we note that the exponentially fading Kernel gives maximum weight to the current, i.e. at the time $\tau = 0$, rather than past, disease history. This might be un-realistic in many cases. Therefore, in the

following we consider the Erlang strong kernel $\varrho(\tau) = a\tau e^{-a\tau}$, so that the integral equation (3) can be replaced by a pair of ODE given by(7) with $n = 2$, **leading to the following family of models:**

$$S' = \mu(1 - S) - \beta(M)IS \quad (14)$$

$$I' = \beta(M)IS - (\mu + \nu)I \quad (15)$$

$$M_1' = a(g(I) - M_1) \quad (16)$$

$$M' = a(M_1 - M). \quad (17)$$

Thus, in principle, it is possible to analytically characterise the local stability of the endemic state for the **above** four-dimensional system. However, also for very simple $g(I)$ and $\beta(M)$, the problem becomes analytically cumbersome. Some partial results on the local stability of the endemic state are thus reported in the appendix **for generic β and g** . Though partial these results indicate some biologically meaningful facts: *i*) for very small delay the endemic state is locally stable. This makes sense since in this case the system approaches the undelayed case (therefore the system is also GAS in this case); *ii*) likewise for large delays the system is LAS; *iii*) the LAS of the endemics state continues to prevail for arbitrary delays if at equilibrium $\beta(g(\cdot))$ is sufficiently large and β' sufficiently small. The latter point means that stability always prevails if contact patterns at equilibrium are not dramatically altered by behaviour change as a response to small changes in the endemic prevalence.

3.2 Numerical results

To get further insight in the system behaviour, in particular as regards the possibility of instability triggered by social behaviour change, we shall investigate numerically the 'benchmark' case $g(I) = kI$ and

$$\beta(M) = \frac{\beta_0}{1 + \alpha \frac{M}{I_*}}, \quad (18)$$

where the parameter I_* is simply a reference value, which we introduce to keep α small. This choice of $\beta(M)$ allows to analytically calculate I_e :

$$I_e(\alpha, k) = I_{SIR} \frac{I_* \mathfrak{R}_0}{\frac{k\alpha\mu}{\mu+\nu} + I_* \mathfrak{R}_0} \quad (19)$$

where $I_{SIR} = \mu(1 - (\mu + \nu)/\beta_0)/(\mu + \nu)$ denotes the equilibrium in the classical mass-action SIR model with contact rate β_0 (Capasso 2008), and $S_e(\alpha, k) = 1 - (1 + \nu/\mu)I_e(\alpha, k)$. Note that the presence of behavioral changes affects the location of the endemic state, compared to the standard SIR model, by lowering the endemic prevalence I_e and increasing the susceptible fraction S_e . As bifurcation parameters we choose α and a that are the new parameters introduced by our model. The former tunes unambiguously the degree of change of contact patterns: given the level of the prevalence I , the greater α is, the smaller the contact rate as a function of I . In particular: α represents the relative rate of decline of the contact rate for an infinitesimal increase of the infective prevalence; a tunes, as already pointed out, the delaying kernel.

In our simulations, we consider the following measles-like parameter constellation: $\mu = (75 \cdot 365)\text{days}^{-1}$, (implying a life expectancy $A = 1/\mu = 75$ years), $\nu = 1/7 \text{ days}^{-1}$, implying an average duration of the infectious period of one week, and $k = 1$. As for the contact rate, we set $\beta_0 = 20(\mu + \nu)$, which implies $\mathfrak{R}_0 = 20$. Finally we set $I_* = 0.9\mu/(\mu + \nu)$ which is the endemic prevalence in the classical SIR model with a BRN equal to 10, implying $I_e = I_*$. Note that at the steady state the value of the contact rate is one half of the value at $M = 0$. This choice of parameters is sufficiently realistic and it allows a

straightforward comparison with the the behavior of the classical SIR model.

Figure 1 reports the the Hopf stability boundary in the (α, a) space and the related stability/instability regions. The figure shows that for low α ($\alpha < \alpha_{min} \approx 0.0386$) the endemic state is LAS independently of the delay, whereas for $\alpha > \alpha_{min}$ the equilibrium is unstable for low or high values of the mean delay, and it is unstable for intermediate delays. For example for $\alpha = 0.5$ instability prevails for an average memory length $T = 2/a$ between 76 days and 3.36 years, whereas for $\alpha = 1$ it prevails for T between 55.4 days and 10.4 years. Finally, at Hopf curve there are supercritical Hopf bifurcations.

In figure 2 we show, for $\alpha = 1$, the off-transient behavior of the ratio $I(t)/I_e$ for an average memory length of two months ($a = 1/30 \text{ days}^{-1}$). As it is easy to see, there are sustained oscillations such that the maximum peak is about 2.4 times the value that would have been reached in case of instantaneous reaction to the disease prevalence. Quite interestingly, the minimum value is roughly equal to one quarter of I_e . This detail is of some interest, since the values of the minima reported in literature for regimes of sustained oscillations are usually very small. In this circumstance low populations stochastic effects should be taken into account. Furthermore, increasing the average delay both reduces the minimum of $I(t)$ and increases the period of the oscillations. For example, for $T = 6$ months and $\alpha = 0.50$ to 1, the oscillation becomes biennial. Thus, seemingly plausible values of the average delay T and of the rate of decline of the contact rate α have the potential to generate the classical biennial inter-epidemic period of measles.

Finally, as in the classical SIR model (London and Yorke 1973, Aron and Schwartz 1984, Earn et al. 2000), nonlinear resonance and chaos may be triggered by seasonal variations of the contact patterns (Grassly and Fraser 2006, Keeling and Grenfell 2002, Bacaer and Abdurahman 2008, Wang and Zhao 2008), modelled by one-year periodically modulating the FoI:

$$FoI(M, t) = \varphi(t)\beta(M)I.$$

For example, by assuming the simple seasonal law:

$$\varphi(t) = 1 + \varphi_1 \cos(\omega t)$$

where $\omega = 2\pi/(1 \text{ year})$), one has that for $T = 6$ months and $\alpha = 1$ the behavior of the system for $\varphi_1 = 0.3$ is chaotic and the Maximum Liapunov Exponent (Wolf et. al. 1985) of the associated Poincare's map is equal to 3.067 (value calculated by using the software NDT by Dr. Joshua Reiss (Reiss 2001)).

4 Concluding Remarks

The results of this paper show facts that, at the best of our knowledge, were never pointed out in the literature on endemic diseases. The main one regards the issue of oscillations, a central one in theoretical epidemiology. As well documented, a variety of diseases, first of all pre-vaccination measles in developed countries, exhibit steady oscillations in large communities, with a period around two years for the measles. Nonetheless, the classical SIR (and SEIR) model has a unique GAS endemic state where oscillations are only a transient, non sustained, phenomenon (Capasso 2008). A huge work has therefore been devoted to the explanation of regular and chaotic oscillations in time-series of infectious diseases (Keeling and Grenfell 2002, London and Yorke 1973, Aron and Schwartz 1984, Earn et al. 2000, Grassly and Fraser 2006) as the 'output' of external periodic forcing due to periodic changes in contacts rates, plus further concurring factors such as time heterogeneity in susceptible recruitment. The present paper indicates that oscillations of endemic disease could have been triggered by a further, largely neglected, factor, i.e. changing human social behavior as a response to the recurrent disease threat. More precisely it shows that

the existence of some degree of behavioral changes in response to the disease threat, coupled with some information delay as a memory of the past disease history may be a source of oscillations of common SIR diseases. Comparing this result to the oscillations triggered by changing vaccination behavior (Reluga et al. 2006, d’Onofrio et al. 2007, d’Onofrio et al. 2008, Buonomo et al. 2008), where an exponentially fading memory is sufficient to destabilize the endemic state and trigger sustained oscillations, here it happens that memories more focused in the disease past history, as exemplified by the Erlangian strong kernel, are needed. Moreover, here sustained oscillations occur even under moderate values of the parameter α tuning the individuals’ reaction to changing prevalence of the disease. In particular, in the oscillatory regime, seemingly plausible values of the average delay T and of the rate of decline of the contact rate α have the potential to generate a large range of values of the inter-epidemic period of the sustained oscillation, including annual and biennial oscillations, as commonly observed for pre-vaccination measles. In the end, although empirical work is needed to better support our results, all this suggests the interesting possibility that behavioral changes could have been important factors in shaping endemic profiles of diseases in history, as recently suggested by (Ferguson 2007).

5 Acknowledgements

We thank very much two anonymous referees and an anonymous associated editor whose suggestions were very helpful.

6 Appendix

If the referees should think it appropriate, this appendix might be put online.

6.1 Persistence of the disease

As we mentioned in the main text, it holds that:

Lemma 1 *If $\mathfrak{R}_0 > 1$ then the disease is strongly persistent, i.e. it exists a $\epsilon_0 > 0$ such that if $I(0) > 0$, $S(0) > 0$ and $M(0) > 0$ then:*

$$\text{MinLim}_{t \rightarrow +\infty} I(t) \geq \epsilon_0 > 0, \text{MinLim}_{t \rightarrow +\infty} S(t) \geq \epsilon_0 > 0, \text{MinLim}_{t \rightarrow +\infty} M(t) \geq \epsilon_0 > 0.$$

For the sake of the notation simplicity, we proof this lemma in the case of weak Erlang kernel, but the proof substantially holds for all Erlang delay kernels.

We start noticing that the set:

$$\Omega = \{(S, I, M) | S \geq 0, I \geq 0, S + I \leq 1, 0 \leq M \leq g(1)\} \quad (20)$$

is positively invariant for our model. Moreover, the disease free state $DFS = (1, 0, 0)$ is on $\partial\Omega$, as well as its stable manifold which is the set $\{(S, I, M) \in \Omega | I = 0\}$. Thus, in virtue of the Freedman-Ruan-Tang theorem (Freedman et al. 1994), if DFS is unstable (i.e. if $\mathfrak{R}_0 > 1$) and, of course, the initial point is in the interior of Ω , then the state variables are strongly persistent.

6.2 LAS of the EE under the exponentially fading memory

In case of exponentially fading kernel, linearizing the system at the endemic equilibrium $(S_e, I_e, g(I_e))$, the characteristic polynomial is:

$$\lambda^3 + (a + \mu + I_e \beta(g(I_e)))\lambda^2 +$$

$$+ \lambda \left(I_E(\mu + \nu)\beta(g(I_E)) + a \left(\mu + I_E\beta(g(I_E)) - \frac{aI_E(\mu + \nu)g'(I_E)\beta'(g(I_E))}{\beta(g(I_E))} \right) \right) +$$

$$a \left(I_E(\mu + \nu)\beta(g(I_E)) - \frac{I_E\mu(\mu + \nu)g'(I_E)\beta'(g(I_E))}{\beta(g(I_E))} \right),$$

whose coefficients are all positive. Thus the Routh-Hurwitz condition for the local stability of the endemic state is:

$$\left(\mu + I_E\beta(g(I_E)) - \frac{I_E(\mu + \nu)g'(I_E)\beta'(g(I_E))}{\beta(g(I_E))} \right) a^2 + ((\mu + I_E\beta(g(I_E)))^2 - I_E^2(\mu + \nu)g'(I_E)\beta'(g(I_E))) a +$$

$$+ I_E^2(\mu + \nu)\beta(g(I_E))^2 + I_E\mu(\mu + \nu)\beta(g(I_E)) > 0,$$

which is fulfilled in all cases since the coefficients of the above second order polynomial in a are positive for all positive increasing $g(I)$ and positive decreasing $\beta(M)$. As a consequence, EE is locally asymptotically stable.

6.3 LAS of the EE under the strong Erlangian kernel

In this case one may show that the Routh-Hurwitz condition takes the form:

$$RH = \sum_{h=1}^5 q_h a^h > 0$$

where the rhs is a 5-th order polynomial in a with null zero degree coefficient and:

$$q_1 = 2I_E^2(\mu + \nu)^2\beta(g(I_E))^2(\mu + I_E\beta(g(I_E))) > 0$$

$$q_5 = 2\mu + 2I_E\beta(g(I_E)) - \frac{2I_E(\mu + \nu)g'(I_E)\beta'(g(I_E))}{\beta(g(I_E))} > 0$$

since $\beta'(M) < 0$ and $g'(I) > 0$. The positivity of q_1 implies that for sufficiently small a (i.e. for large average delays) the EE is LAS. The positivity of q_5 implies, on the contrary, that for sufficiently large a (i.e. for very small average delays) the EE is LAS. Moreover, for large a it also holds that:

$$a^{-1}M'_1 \approx 0 \Rightarrow M_1 = g(I)$$

$$a^{-1}M' \approx 0 \Rightarrow M = M_1 \Rightarrow M = g(I),$$

Therefore by singular perturbation approximation the system reduces to the undelayed one, which is not only LAS but also GAS. As far as the other coefficients are concerned, they have variable sign:

$$q_2 = 4I_E(\mu + \nu)\beta(g(I_E))(\mu + I_E\beta(g(I_E)))^2 + \frac{I_E(\mu + \nu)(\mu^2 - I_E\nu\beta(g(I_E)))g'(I_E)\beta'(g(I_E))(\mu + I_E\beta(g(I_E)))}{\beta(g(I_E))}$$

$$q_3 = 2(\mu + I_E\beta(g(I_E)))(\mu^2 + I_E\beta(g(I_E))(4\mu + 2\nu + I_E\beta(g(I_E)))) +$$

$$+ \frac{2I_E(\mu + \nu)(\mu^2 + I_E\beta(g(I_E))(\mu + \nu - I_E\beta(g(I_E))))g'(I_E)\beta'(g(I_E))}{\beta(g(I_E))}$$

$$q_4 = 4(\mu + I_E\beta(g(I_E)))^2 - \frac{I_E^2(\mu + \nu)^2g'(I_E)^2\beta'(g(I_E))^2}{\beta(g(I_E))^2} + \frac{I_E(\mu + \nu)(\mu - 3I_E\beta(g(I_E)))g'(I_E)\beta'(g(I_E))}{\beta(g(I_E))}$$

However, a sufficient condition to have $q_2 > 0$ is:

$$\mu^2 - I_E \nu \beta(g(I_E)) < 0 \quad (21)$$

Similarly we have the following a sufficient condition guaranteeing that $q_3 > 0$:

$$\mu^2 + I_E \beta(g(I_E))(\mu + \nu - I_E \beta(g(I_E))) < 0 \quad (22)$$

and $q_4 > 0$:

$$\frac{\beta(g(I_E)) \left(\mu - 3I_E \beta(g(I_E)) - \sqrt{17\mu^2 + I_E \beta(g(I_E))(26\mu + 25I_E \beta(g(I_E)))} \right)}{2I_E(\mu + \nu)g'(I_E)} < \beta'(g(I_E)) < 0. \quad (23)$$

As a consequence, if at the EE all the conditions (21), (22) and (23) are met then the EE is LAS. These conditions are quite general, since they are not linked a priori to particular forms of the contact rate or of $g(I)$. Although, they are complex and of difficult reading, a simple biological interpretation is given in the text.

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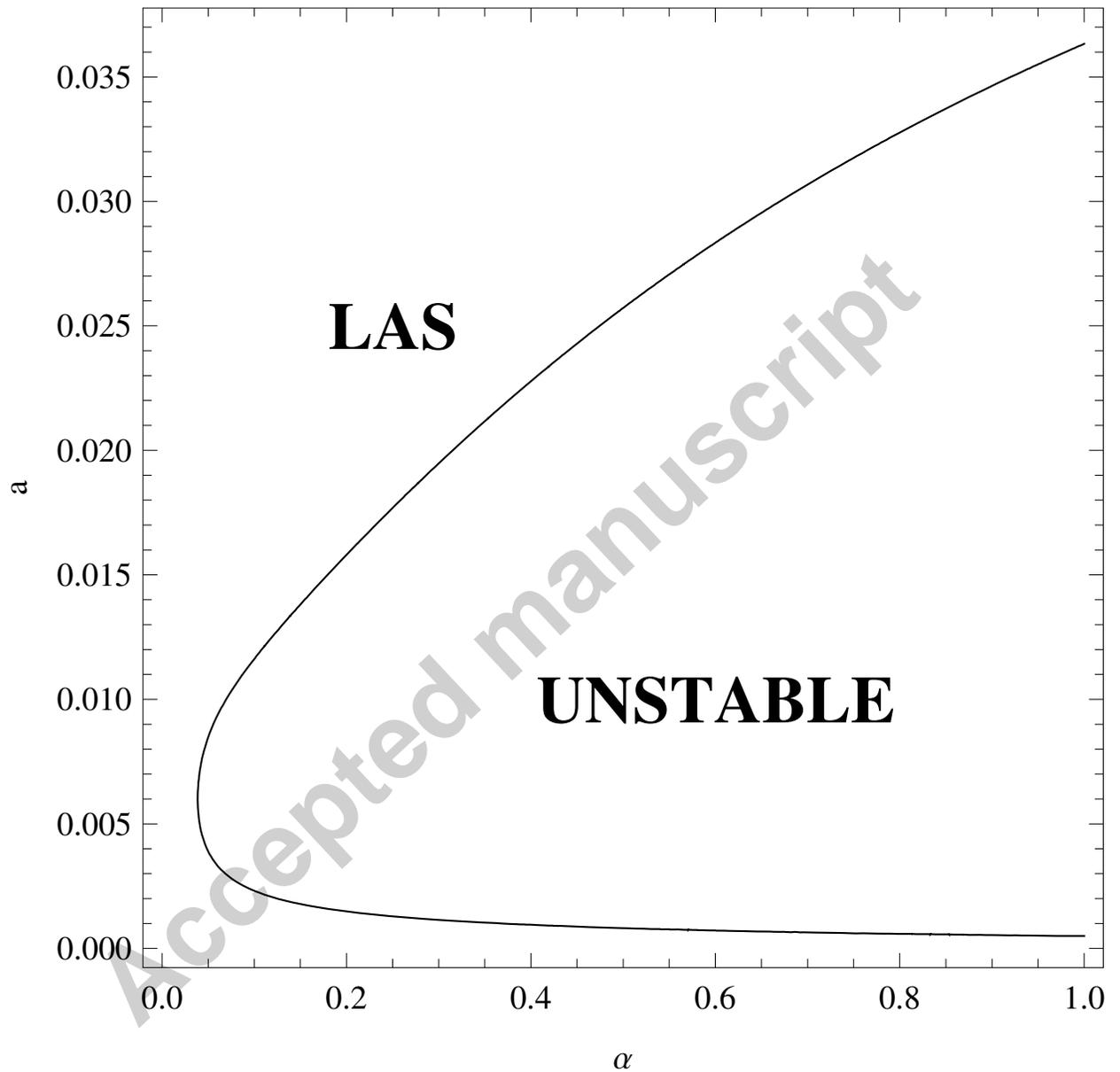


Figure 1: Strong Erlangian kernel. Local stability boundary, and corresponding regions of Local Asymptotic stability and instability in the parameter space (α, a) , where α is the relative rate of decline of the contact rate for an infinitesimal increase of the infective prevalence, and a is inversely related to the average information delay T ($a = 2/T$).

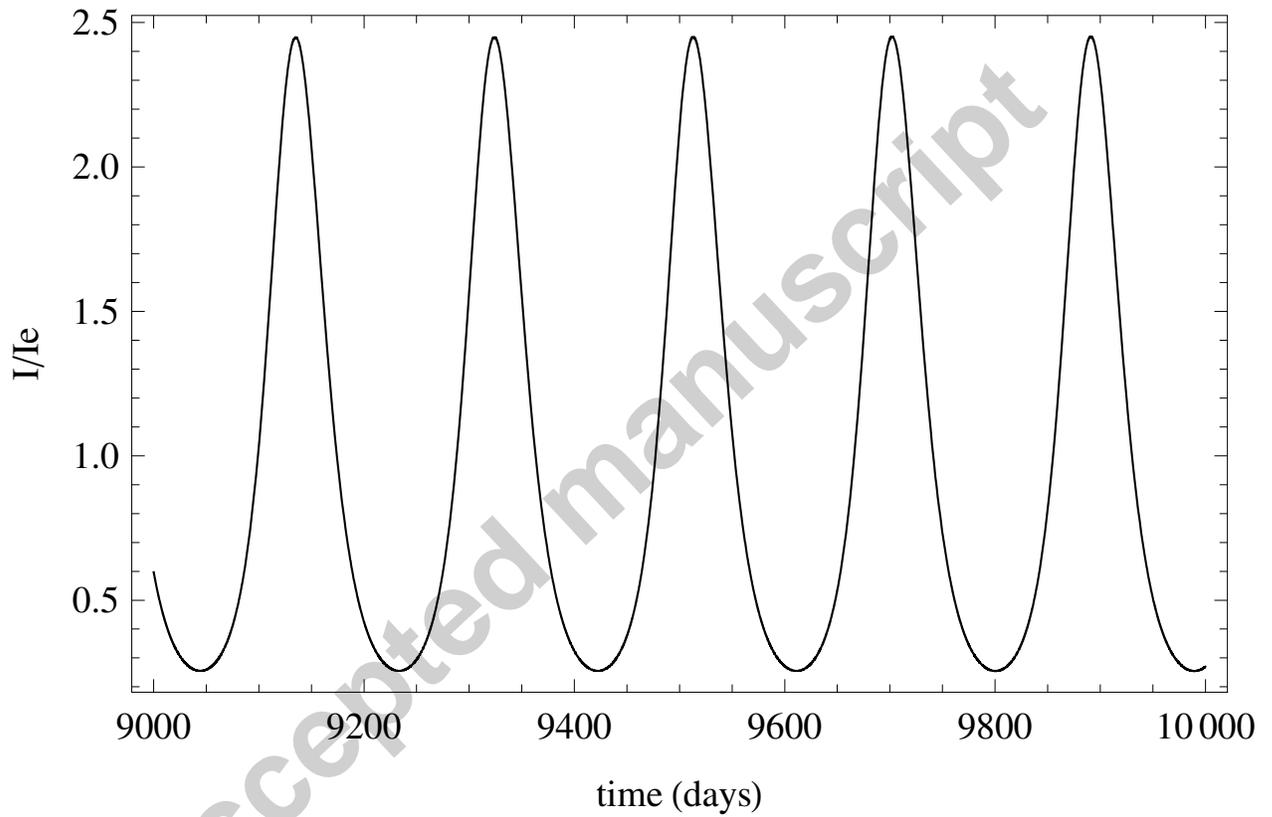


Figure 2: After-transient sustained oscillations of infectious fraction (scaled to the equilibrium value $I_e(\alpha)$) that are induced by information-dependent FoI. Here $\alpha = 1$ and $a = 30^{-1} \text{ days}^{-1}$ corresponding to an average memory of two months: $T = 60$ days