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Spontaneous behavioural changes in response to epidemics

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Abstract

We study how spontaneous reduction in the number of contacts could develop, as a defensive response, during an epidemic and affect the course of infection events. A model is proposed which couples an SIR model with selection of behaviours driven by imitation dynamics. Therefore, infection transmission and population behaviour become dynamical variables that influence each other. In particular, time scales of behavioural changes and epidemic transmission can be different. We provide a full qualitative characterization of the solutions when the dynamics of behavioural changes is either much faster or much slower than that of epidemic transmission. The model accounts for multiple outbreaks occurring within the same epidemic episode. Moreover, the model can explain "asymmetric waves", i.e., infection waves whose rising and decaying phases differ in slope. Finally, we prove that introduction of behavioural dynamics results in the reduction of the final attack rate.

Key words: mathematical modelling, evolutionary game theory, infectious diseases

1 Introduction

The epidemic dynamics depends on the complex interplay between the characteristics of the pathogens' transmissibility and the structure and behaviour of the host population. Spontaneous change of behaviour in response to epidemics (Ferguson, 2007), possibly related to risk perception (Bagnoli et al., 2007; Risau-Gusman and Zanette, 2008; Shaw and Schwartz, 2008), has been recently proposed as a relevant factor in the comprehension of infection dynamics. While the merits and influence of such phenomena are still debated (D'Onofrio et al., 2007; Moneim, 2007), experience from the 1918-19 pandemic indicates that a better understanding of behavioural patterns is crucial to improve model realism and enhance the effectiveness of containment/mitigation policies (Bootsma and Ferguson, 2007).

Human behaviour is driven by evaluation of prospective outcomes deriving from alternative decisions and cost-benefit considerations. Past experience, response to the action of others and changes in exogenous conditions all contribute to the balance, to which game theory provides a rich and natural modelling framework (von Neumann and Morgenstern, 1947; Hofbauer and Sigmund, 1998). It is not surprising, therefore, that looking at behaviours through the lens of game theory has recently attracted the attention of the

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epidemiology community, for example when modelling the evolution over time of voluntary vaccination uptakes (Bauch and Earn, 2004; Bauch, 2005).

In this paper we model a fairly general situation in which a population of individuals is subject to an epidemic outbreak developing according to an SIR model, but in which contact rates depend on the behavioural patterns adopted across the population. More specifically, all susceptible individuals can conform to either one or the other of two different behaviours, b_a and b_n, respectively corresponding to an "altered" and a "normal" behavioural pattern. The first gives the individuals an advantage in terms of reduced risk of infection, yet at some extra cost. For example, avoidance of crowded environments reduces the risk of infection, but also entails disadvantages deriving from greater isolation. Individuals adopting the second (b_n) are exposed to a normal risk of infection, but are spared the extra cost associated with b_a. Individuals may choose to switch between b_a and b_n at any time, depending on cost-benefit assessments based on the perception of risk.

The resulting model consists in the coupling of two dynamical systems, one describing the epidemic transmission and the other describing the behavioural changes. In principle, there is no reason for the two phenomena to evolve at the same speed. It is therefore crucial to study the model allowing for different time–scales, embodied in different time-units.

We give a full description of the model when the dynamics of the behavioural changes are "fast" with respect to the epidemic transmission. In particular, we provide sufficient conditions on the parameters for generating sequences of epidemic waves. Moreover, we show that the model is able to account for "asymmetric waves", i.e., infection waves whose rising and decaying phases

differ in slope. However, similar patterns can also be observed when the time–scales of the two dynamics are comparable. When the dynamic of behavioural changes is "slow", the model basically reduces to a classical SIR.

The model's dynamics gives rise to patterns that are morphologically compatible with multiple outbreaks and the same-wave asymmetric slopes recently reported for the Spanish influenza of the 1918–19 (Chowell et al., 2006b,a; Ferguson et al., 2006; Mills et al., 2004). For these phenomena (trivially incompatible with the classical SIR model) a variety of alternative explanations have in fact been advanced: military demobilization at the end of the First World War (Ferguson et al., 2006), genetic variation of the influenza virus (Castillo-Chavez et al., 1989; Andreasen et al., 1997; Boni et al., 2004), exogenous time changes in transmission rates, such as seasonal forcing (Colizza et al., 2006, 2007). Other explanations have been proposed invoking coinfection scenarios (May and Nowak, 1995; Adler and Losada, 2002; Edwards et al., 2004; Merler et al., 2008)

Finally, and regardless of the relative speeds of dynamics, we show that the fraction of susceptible individuals at the end of the epidemic is always larger than that of a classical SIR model in which all individuals adopt the normal behaviour (b_n) with the same parameters.

2 The Model

Our model consists of the coupling of two mutually influencing phenomena: a) the epidemic transitions; b) the behavioural changes in the population of susceptible individuals.

As for the epidemic transitions, whose time unit is t, our model is based on an $S \rightarrow I \rightarrow R$ scheme [1]. We consider that susceptible individuals may adopt two mutually exclusive behaviours, b_n ("normal") and b_a ("altered"). Specifically, we assume that individuals adopting behaviour b_a are able to reduce the number of contacts in the time unit with respect to individuals adopting behaviour b_n. Thus, two transmission rates are considered for the two groups, accounting for the different contact rates associated with behaviours b_a and b_n. In particular, susceptible individuals adopting behaviour b_n, $S_n(t)$, become infected at a rate $\beta_n I(t)$ (and thus $\dot{S}_n(t) = -\beta_n S_n(t) I(t)$), where $I(t)$ represents the pool of infectious individuals, while susceptible individuals adopting behaviour b_a, $S_a(t)$, become infected at a rate $\beta_a I(t)$ (and thus $\dot{S}_a(t) = -\beta_a S_a(t) I(t)$), with $\beta_a < \beta_n$. Introducing the variables $S(t) = S_a(t) + S_n(t)$ and $x(t) = S_n(t)/(S_n(t) + S_a(t))$, corresponding to the whole susceptible population and to the fraction of susceptibles adopting behaviour b_n respectively, the epidemic model can be written as:

$$\begin{cases} \dfrac{dS}{dt}(t) = -\left[\beta_n S(t)x(t) + \beta_a S(t)(1-x(t))\right] I(t) \\ \dfrac{dI}{dt}(t) = \left[\beta_n S(t)x(t) + \beta_a S(t)(1-x(t))\right] I(t) - \gamma I(t) \\ \dfrac{dR}{dt}(t) = \gamma I(t) \\ \dfrac{dx}{dt}(t) = x(t)(1-x(t))(\beta_a - \beta_n) I(t) \ . \end{cases} \tag{1}$$

Notice that the last equation describes the change of behaviours distribution in susceptible individuals deriving from the different rates of infection, β_n and

[1] Since we model single epidemic outbreaks, the vital dynamics of the population is not taken into account.

β_a.

We now allow susceptible individuals to change their behaviour spontaneously, following cost/benefit considerations. This phenomenon can be cast in the language of evolutionary game theory, in which behaviours correspond to strategies in a suitable game, with certain expected payoffs. Adopting b_a reduces the risk of infection, but it is more costly. On the other hand, individuals adopting b_n are exposed to a higher risk of infection. It is clear that whether it is more convenient to adopt the first behaviour or the second depends on the state of the epidemic.

Of course, the two phenomena may not have the same time scales. In fact, while epidemic transmission can occur only through person-to-person contacts, it is fairly reasonable to consider that individuals can access the information required to decide whether to adopt either b_n or b_a, much more frequently by telephone, email, the Internet and, in general, the media.

Let us therefore introduce τ as the time unit of behavioural changes, and let us assume that $t = \alpha\tau$ with $\alpha > 0$.

Payoffs can now be modelled as it follows. All individuals pay a cost for the risk of infection, which we assume depends linearly on the fraction of infected individuals, $I(\tau)$, and it is higher for b_n than for b_a. Moreover, individuals playing b_a pay an extra, fixed cost k. It may be convenient to think of k as deriving from reducing the contacts with people, and therefore less traveling, working, attending school, visiting friends and relatives, etc.. Yet, it is more general than that, as it can account, in fact, for the cost of any self-imposable prophylactic measure. The payoffs associated with b_n and b_a are:

$$\begin{aligned} p_n(\tau) &= -m_n I(\tau) \\ p_a(\tau) &= -k - m_a I(\tau) \quad , \end{aligned} \tag{2}$$

with $m_n > m_a$. We may think of m_n and m_a as parameters related to the risk of developing symptoms (especially for the lethal infections) induced by the two different behaviours b_n and b_a.

The dynamics of behaviours is modelled as a selection dynamics based on imitation (*Imitation Dynamics* (Hofbauer and Sigmund, 1998; Nowak and Sigmund, 2004)). A fraction of the individuals playing strategy b_n can switch to strategy b_a after having compared the payoffs of the two strategies, at a rate proportional to the difference between payoffs, $\Delta P(\tau) = p_n(\tau) - p_a(\tau)$, with proportionality constant ρ. Conversely for the fraction of the individuals playing b_a.

The last equation of system (1), in the time scale of infection transmission, thus becomes:

$$\begin{aligned} \frac{dx}{dt}(t) &= x(t)(1-x(t))(\beta_a - \beta_n)I(t) + \\ &+ \frac{\rho}{\alpha} x(t)(1-x(t))(k - (m_n - m_a)I(t)) \ . \end{aligned} \tag{3}$$

Notice that the first component of the time derivative of $x(t)$ is negative, meaning that the fraction of susceptible individuals adopting the normal beahviour b_n can only decrease over time as an effect of the *selection of behaviours* induced by the epidemic. On the other hand, whenever b_n is more convenient than b_a ($p_n(t) > p_a(t)$), the fraction in the population of susceptibles playing b_n can grow.

Let us briefly comment on the second component of the time derivative of $x(t)$. In principle, since the number of susceptible individuals decreases over time, one can argue that spontaneous changes of behaviour must depend explicitly on $S(t)$, because of the diminished number of contacts among susceptible individuals. However, here we assume that susceptible individuals take their decision on the basis of the composition of the pool of susceptible individuals that they are able to meet somehow (by looking only at the fractions of susceptible individuals adopting the two behaviours b_n and b_a, without considering the size of the sample).

It is worth noticing that $x = 0$ and $x = 1$ are equilibria for Eq. (3). This in particular implies that there is no way to switch to a different strategy (independently of whether it would be convenient) unless there is a non zero fraction of individuals already playing it. To circumvent this (which one may regard as an undesirable effect of strict imitation), *irrational behaviour* can be introduced which allows for rare (in τ time units) random switches of behaviour independent of encounters and pay-offs. Assuming a constant rate, $\chi > 0$, equal for both behaviours, the resulting equation for x is:

$$\begin{aligned}\frac{dx}{dt}(t) &= x(t)(1-x(t))(\beta_a - \beta_n)I(t) + \\ &+ \frac{\rho}{\alpha}x(t)(1-x(t))(k-(m_n-m_a)I(t)) + \\ &+ \frac{\chi}{\alpha}(1-x(t)) - \frac{\chi}{\alpha}x(t)\ .\end{aligned} \tag{4}$$

Therefore, the complete dynamics of infection (coupling behaviour with epidemic transitions) is given by:

$$\begin{cases} \dfrac{dS}{dt}(t) &= -\left[\beta_n S(t)x(t) + \beta_a S(t)(1-x(t))\right] I(t) \\ \dfrac{dI}{dt}(t) &= \left[\beta_n S(t)x(t) + \beta_a S(t)(1-x(t))\right] I(t) - \gamma I(t) \\ \dfrac{dR}{dt}(t) &= \gamma I(t) \\ \epsilon\dfrac{dx}{dt}(t) &= x(t)(1-x(t))(1-mI(t)) + \mu(1-2x(t)) \ . \end{cases} \tag{5}$$

where $\epsilon = \dfrac{\alpha}{k\rho}$, $m = (m_n - m_a)/k + \epsilon(\beta_n - \beta_a)$ and $\mu = \dfrac{\chi}{k\rho}$. As for the constraints on the models parameters, we have: $0 < \beta_a < \beta_n$, $0 < \gamma < \beta_n$ [2], $\epsilon > 0$, $m > \epsilon(\beta_n - \beta_a)$ and $\mu > 0$. For facilitating the reader's understanding, the definitions of the variables recurring throughout the paper are reported in Tab. 1.

3 Study of Dynamics

System (5) admits a continuum of equilibria, namely $(S^\star, 0, 1-S^\star, x^\star)$ with

$$x^\star = \frac{1 - 2\mu + \sqrt{1+4\mu^2}}{2} \ , \tag{6}$$

and $S^\star \in [0,1]$. We consider in detail only the case of an epidemic of a novel pathogen type, so that we are interested only at the equilibrium with $S^\star = 1$, i.e. $(1,0,0,x^\star)$. This is unstable when $\beta_n x^\star + \beta_a(1-x^\star) > \gamma$. Thus, we assume that the initial values for system (5) are the following: $(S(0), I(0), R(0), x(0)) = (1-I_0, I_0, 0, x^\star)$ with I_0 close to 0. Note that $1/2 < x^\star < 1$ and $x^\star \to 1$ when $\mu \to 0$. Moreover, this equilibrium is stable as long as $R_0 < 1$ where the basic reproductive number of system (5) is:

[2] This constraint is required only to ensure that the epidemic occurs (see Eq. 7).

Table 1

Model variables and parameters

Notation	Description
S	Number of susceptible individuals
I	Number of infectious individuals
R	Number of recovered individuals
x	Fraction of susceptibles individuals adopting the "normal" behaviour
β_n	Transmission rate of individuals adopting the "normal" behaviour
β_a	Transmission rate of individuals adopting the "altered" behaviour
γ	Recovery rate
$1/m$	Threshold value determining the switch between "normal" and "altered" behaviour
μ	Rate of irrational behaviour change
ϵ	Relative speed of SIR dynamics to behavioural response

$$R_0 = \frac{\beta_n x^\star + \beta_a(1 - x^\star)}{\gamma} \ . \tag{7}$$

Let us introduce the basic quantities $R_0^n = \beta_n/\gamma$ and $R_0^a = \beta_a/\gamma$. We can rewrite Eq. (7) as $R_0 = R_0^n x^\star + R_0^a(1 - x^\star)$. The quantities R_0^n and R_0^a are reproduction numbers themselves: R_0^n characterizes the situation where the susceptible pool is fully composed by individuals adopting the normal behaviour b_n, whereas R_0^a characterizes the situation where the susceptible pool is fully composed by individuals spontaneously reducing their contacts (behaviour b_a). Thus, Eq. (7) has a straightforward interpretation: a typical in-

fective individual behaving according to b_n (a case occurring with probability $x^\star$) would cause R_0^n new infections during his/her whole period of infectivity. Similarly for R_0^a in case he/she adopts the altered behaviour b_a (which occurs with probability $1 - x^\star$). Note that $R_0 \simeq R_0^n$ for $x^\star \simeq 1$.

We start by analyzing the dynamics of system (5) in two extreme cases, namely $\epsilon \to 0$ and $\epsilon \to +\infty$, which correspond respectively to the situation when the dynamics of the behavioural changes is "fast" or "slow" with respect to the epidemic transmission.

Let us consider first the case $\epsilon \to +\infty$. In this case, the solutions of system (5) approximate, dividing both sides of the last equation by ϵ and remembering $m = (m_n - m_a)/k + \epsilon(\beta_n - \beta_a)$, those of system (1), which is a classical SIR model with two classes of susceptibility. Since $\dot{x}(t) < 0$, the fraction of individuals adopting the normal behaviour b_n will decrease over time as a consequence of the selection of the behaviour b_a induced by the epidemic, even in the absence of spontaneous behavioural changes.

For the case $\epsilon \to 0$ (which is more interesting from both the mathematical and the biological point of view) we are going to apply the singular perturbation methods (O'Malley Jr., 1991).

The solutions of the singularly perturbed initial value problem (5) is approximated by that of the degenerate system:

$$\begin{cases} \dfrac{dS}{dt}(t) &= -\left[\beta_n S(t)x(t) + \beta_a S(t)(1-x(t))\right] I(t) \\ \dfrac{dI}{dt}(t) &= \left[\beta_n S(t)x(t) + \beta_a S(t)(1-x(t))\right] I(t) - \gamma I(t) \\ \dfrac{dR}{dt}(t) &= \gamma I(t) \\ 0 &= x(t)(1-x(t))(1-mI(t)) + \mu(1-2x(t)) \ , \end{cases} \tag{8}$$

obtained from (5) by formally setting $\epsilon = 0$, provided that in the last of (8) we use an asymptotically stable equilibrium of the boundary–layer system

$$\frac{dx}{ds}(s) = x(s)(1-x(s))(1-mI) + \mu(1-2x(s)) \ , \tag{9}$$

obtained by making the transformation of independent variable $s = t/\epsilon$, and then setting $\epsilon = 0$ (which in particular implies that $S(s)$, $I(s)$ and $R(s)$ are constant) (Tikhonov, 1952; Hoppensteadt, 1966).

Notice that, after having set $\epsilon = 0$, parameter m reduces to $(m_n - m_a)/k$. Consequently, as it may be expected, the effect of the selection of behaviours induced by the epidemic is negligible when the dynamics of behaviours is much faster than that of the infection transmission.

We start by analyzing the solutions of Eq. (9), where the fraction of infected individuals I is assumed to be constant. Eq. (9) admits the following equilibrium:

$$x^\star(I) = \begin{cases} \dfrac{1-2P+\sqrt{1+4P^2}}{2} & \text{if } I < 1/m \iff P > 0 \\ \dfrac{1-2P-\sqrt{1+4P^2}}{2} & \text{if } I > 1/m \iff P < 0 \\ \dfrac{1}{2} & \text{if } I = 1/m \ , \end{cases} \tag{10}$$

where $P = \frac{\mu}{1-mI}$, which is asymptotically stable (comparing Eq. (6), note that $x^\star = x^\star(0)$). In conclusion, the following Proposition holds:

Proposition 3.1 *The boundary–layer system* (9) *admits the asymptotically stable equilibrium* (10)*. Furthermore,* $x^\star(I) \to 1$ *if* $I < 1/m$ *and* $x^\star(I) \to 0$ *if* $I > 1/m$ *when* $\mu \to 0$*.*

Concerning the stability of the equilibrium (10), it is sufficient to observe that the equation of $\dot{x}$ is a parabola (which reduces to a straight line when $I = 1/m$) and that the sign of $\dot{x}$ is positive for $x < x^\star(I)$ and negative for $x > x^\star(I)$.

Substituting (10) in the first two equations in (8), one obtains a two-dimensional system in the variables (S, I) that can be analysed using classical methods. Here we restrict ourselves to consider the biologically relevant and mathematically challenging limiting case for $\mu \to 0$. In the limiting case, the right hand side of (10) becomes discontinuous in I, and standard theory cannot be applied. Instead, we obtain the following.

Proposition 3.2 *Under the assumptions* $R_0^n > 1$ *and* $1/m < I_p$ *where* $I_p = 1 - \frac{1}{R_0^n} + \frac{1}{R_0^n} \log \frac{1}{R_0^n}$*, if* $\epsilon \to 0$ *and* $\mu = o(\epsilon^k)$ *with* $k \geq 1$*, the solutions of system* (5) *are characterized as follows:*

S1 *there exists a finite time* $t_1 > 0$ *such that the solutions of system* (5) *approximate those of a classical SIR model with* $R_0 = R_0^n$ *on the interval*

$(0, t_1)$ *and* $I(t_1) = 1/m$*;*

S2.1 *If* $R_0^a S(t_1) \leq 1$*, there exists a finite time* $t_2' > t_1$ *such that the solution of system* (5) *can be approximated in the time interval* (t_1, t_2')*, where* $t_2' = t_1 + \frac{m}{\gamma}(S(t_1) - \frac{1}{R_0^n})$*, by* $S(t) = S(t_1) - \frac{\gamma}{m}(t - t_1)$ *and* $I(t) \equiv 1/m$*. Afterwards, the solutions of system* (5) *approximate those of a classical SIR model (in its decaying phase) with* $R_0 = R_0^n$ *on the interval* $(t_2', +\infty)$*;*

S2.2 *If* $R_0^a S(t_1) > 1$ *there exists a finite time* $t_2 > t_1$ *such that the solutions of system* (5) *approximate those of a classical SIR model with* $R_0 = R_0^a$ *on the interval* (t_1, t_2) *and* $I(t_2) = 1/m$*;*

S2.2.1 *If* $R_0^n S(t_2) > 1$ *there exists a finite time* $t_3 > t_2$ *such that the solutions of system* (5) *can be approximated in the time interval* (t_2, t_3)*, where* $t_3 = t_2 + \frac{m}{\gamma}(S(t_2) - \frac{1}{R_0^n})$*, by* $S(t) = S(t_2) - \frac{\gamma}{m}(t - t_2)$ *and* $I(t) = 1/m$*. Afterwards, the solutions of system* (5) *approximate those of a classical SIR model (in its decaying phase) with* $R_0 = R_0^n$ *on the interval* $(t_3, +\infty)$*;*

S2.2.2 *If* $R_0^n S(t_2) \leq 1$ *the solutions of system* (5) *approximate those of a classical SIR model (in its decaying phase) with* $R_0 = R_0^n$ *on the interval* $(t_2, +\infty)$*.*

Therefore, under the hypotheses of Prop. 3.2, solutions of system (5) can be classified in the three following types:

C1 Solution *S1* in $[0, t_1)$ and *S2.1* in $[t_1, +\infty)$;

C2 Solution *S1* in $[0, t_1)$, *S2.2* in $[t_1, t_2)$ and *S2.2.1* in $[t_2, +\infty)$;

C3 Solution *S1* in $[0, t_1)$, *S2.2* in $[t_1, t_2)$ and *S2.2.2* in $[t_2, +\infty)$.

The possible behaviours of the solutions of system (5), which depends on the values of R_0^a and R_0^n, are shown in Fig. 1.

Let us briefly comment on the hypotheses of Prop. 3.2. The condition $R_0^n > 1$

is the obvious threshold condition for an epidemic to occur. $I_p = 1 - \frac{1}{R_0^n} + \frac{1}{R_0^n} \log \frac{1}{R_0^n}$ is the fraction of infected individuals at the peak for the classical SIR model with basic reproductive number $R_0 = R_0^n$ (this can be easily established by considering that the fraction of infected individuals at the peak is $\frac{1}{R_0}$ and by employing the SIR invariant $S(t) + I(t) - \frac{1}{R_0} \log S(t) = \text{const}$). Thus the condition $1/m < I_p$ imposes that behaviour b_a starts being convenient at some point before the epidemic reaches its peak. Basically, if the condition is not satisfied, system (5) is of scarce interest since all individuals adopt the normal behaviour b_n during the course of the epidemic; thus, system (5) would be equivalent to a classical SIR model with basic reproductive number $R_0 = R_0^n$. No explicit condition is needed on R_0^a. In particular, R_0^a can be less than 1 (which means that no epidemic will occur if the susceptible pool is fully composed by individuals adopting the altered behaviour b_a). Clearly, in this case the solutions of system (5) can only be of type *C1*.

Full proof of Prop. 3.2 is given in App A. Here we only observe that when $I(t) < 1/m$ we have $x^\star(I) \to 1$ (see Prop. 3.1). Thus, the solutions of the degenerate system (8), obtained by solving the system of differential equations after having substituted $x(t) = 1$, are those of a classical SIR model with basic reproductive number $R_0 = R_0^n$. The same happens when $I(t) > 1/m$, but now $x^\star(I) \to 0$, which results in $R_0 = R_0^a$. Let us now assume that ϵ is close to 0. The time intervals in which $I(t) \approx 1/m$ (for solutions of type *C1* or *C2*) can be interpreted as time intervals in which the fraction of infected individuals $I(t)$ is characterized by a sequence of "micro–waves". In fact, as soon as $I(t) > 1/m$, $x(t)$ gets close to 1, so that the effective reproductive number ($R_0^a S(t_1)$ for solutions of type *C1* and $R_0^a S(t_2)$ for solutions of type *C2*) is not sufficiently large to sustain the epidemic and thus $I(t)$ decreases below $1/m$. However,

as soon as $I(t) < 1/m$, $x(t)$ gets close to 1, so that the effective reproductive number ($R_0^n S(t_1)$ for solutions of type *C1* and $R_0^n S(t_2)$ for solutions of type *C2*) is sufficient to sustain the epidemic and thus $I(t)$ increases over $1/m$. The process is repeated as long as the fraction of susceptible individuals in the population is sufficiently large ($R_0^n S(t) > 1$). In the limit $\epsilon \to 0$, these switches are instantaneous, and the solution $I(t)$ is approximately always equal to $1/m$. Finally, as soon as $R_0^n S(t) \leq 1$, the fraction of infected individuals $I(t)$ will start decreasing to 0 over time. In Prop. 3.3 we give sufficient conditions for solutions of type *C1* or *C2* to occur, which in particular implies the presence of sequences of "micro–waves" for small value of ϵ.

Proposition 3.3 *Under the assumptions* $R_0^n > 1$ *and* $1/m < I_p$, *where* $I_p = 1 - \frac{1}{R_0^n} + \frac{1}{R_0^n} \log \frac{1}{R_0^n}$, *if* $\epsilon \to 0$, $\mu = o(\epsilon^k)$ *with* $k > 1$ *and* R_0^a *satisfies the inequalities* $1 < R_0^a < R_0^n \exp\{-R_0^a(1 - 1/R_0^n)\}$ *then the solution of system* (5) *are of type* C1 *or* C2.

First of all, we comment on the hypotheses of Prop. 3.3. Clearly, if $R_0^a S(t_1) \leq 1$ the solutions of system (5) can only be of type *C1*. Condition $R_0^a S(t_1) > 1$ (which in particular implies $R_0^a > 1$) is thus required for solutions of type *C2* to occur, in particular to have that $I(t)$ is increasing in t_1. Condition $R_0^a < R_0^n \exp\{-R_0^a(1 - 1/R_0^n)\}$ is necessary to have that the fraction of susceptible individuals does not decrease too much in the time interval (t_1, t_2), where system (5) is equivalent to an SIR model with basic reproductive number $R_0 = R_0^a$. In fact, if $S(t)$ decreases so much that $R_0^n S(t_2) < 1$, $I(t)$ will decrease again for $t > t_2$, resulting in a solution of type *C3*. Full proof of Prop. 3.3 is given in App. A.

Prop. 3.3 guarantees that, under certain conditions, one (or more) epidemic

waves will occur after the first when $\epsilon > 0$ is sufficiently small; here, a solution showing two (or more) epidemic waves is one for which $\dot{I}(t) > 0$ in two time intervals separated by one interval in which $\dot{I}(t) < 0$. A concrete example is shown in Fig. 2a. In this case, a sequence of small epidemic waves is observed for $t > t_2$. In fact, as soon as the fraction of infected individuals becomes larger than the threshold $1/m$, the dynamics is the same as that of an SIR model with $R_0 = R_0^a$ for which there are not enough susceptible individuals to sustain the epidemic. Thus, the fraction of infected individuals decreases below the threshold value (see the inset in Fig. 2a). A series of waves therefore follows, as long as $R_0^n S(t) > 1$. Fig. 2b shows that, as stated in Prop. 3.2, $S(t)$ decreases linearly while $I(t)$ undergoes this sequence of waves.

Convergence of the solutions of the singularly perturbed system (5) to those of the degenerate system (Eq. 8, $\epsilon = 0$) for $\epsilon \to 0$ is shown in Fig. 2c-d.

If we consider greater values of the parameter ϵ (about which proposition 3.3 does not say anything), the fraction of infected individuals reaches a higher peak, and thus the fraction of susceptible individuals decreases in the time interval (t_1, t_2) more than that of a SIR model with $R_0 = R_0^a$. However, if R_0^a is not too large, the fraction of susceptible individuals at time $t = t_2$ can be sufficient to generate at least a second epidemic wave (see Fig. 3a), that is now quite relevant in size.

As observed previously, if R_0^a is not sufficiently small (as required by Prop. 3.3) the fraction of susceptible individuals in the time interval (t_1, t_2) may decrease so much that $R_0^n S(t_2) < 1$. In this case, no additional waves will be generated and only a change in the slope during the decaying phase may be observed (see Fig. 3b).

One may ask how large ϵ can be to give rise to a second epidemic wave of the type shown in Fig. 3a. Fig. 4a shows a numerical approximation to the minimum value, ϵ_{min}^{-1}, of $1/\epsilon$ giving rise to sequences of at least two epidemic waves, as a function of the threshold parameter m. In this respect, it should be observed that computing, given m, the value of ϵ at which multiple waves start to occur is essentially equivalent to the problem of locating the zero (if it exists) of a one-variable monotonic function, within a suitable interval. It can be observed that ϵ_{min}^{-1} decreases with m and $\epsilon_{min}^{-1} \searrow 0$ as $m \to +\infty$. Moreover, m has to be larger than the theoretical minimum $m = 1/I_p$ (shown as the dotted vertical line in Fig. 4a) in the assumptions of Prop. 3.2; indeed ϵ_{min}^{-1} goes to ∞ (i.e. ϵ_{max} goes to 0) as $m \to 1/I_p$.

The following Proposition shows that, independently of ϵ, the fraction of susceptible individuals at the end of an epidemic described by an SIR model with $R_0 = R_0^n$ is always smaller than that obtained with model (5):

Proposition 3.4 *$S_\infty > S_\infty^{SIR}$, where S_∞^{SIR} is the fraction of susceptible individuals at the end of an epidemic described by a classical SIR model with transmission rate β_n and S_∞ is the fraction of susceptible individuals at the end of an epidemic described by system* (5).

Finally, for $\epsilon \approx 0$, the dependence of S_∞ from m is clarified by the following:

Proposition 3.5 *Under the assumptions $R_0^n > 1$ and $1/m < I_p$, where $I_p = 1 - \frac{1}{R_0^n} + \frac{1}{R_0^n} \log \frac{1}{R_0^n}$, in the limit $\epsilon \to 0$, $\mu = o(\epsilon^k)$ with $k \geq 1$, if R_0^a satisfies the inequalities $1 < R_0^a < R_0^n \exp\{-R_0^a(1 - 1/R_0^n)\}$ then the fraction of susceptible individuals at the end of the epidemic ($S_\infty(m)$) is an increasing function of m and $S_\infty(m) \to 1/R_0^n$ when $1/m \to 0$.*

Proofs of Prop. 3.4 and 3.5 are in appendix A. In Fig. 4b the values of S_∞ are reported for increasing values of $1/\epsilon$ and for different choices of m. We can see that S_∞ is non monotonic in neither $1/\epsilon$ nor in m. However, when $1/\epsilon$ is sufficiently large, S_∞ increases by decreasing $1/m$ and $S_\infty \to 1/R_0^n$ when $1/m \to 0$. For small values of $1/\epsilon$, S_∞ is equivalent to that obtained by employing a classical SIR model with $R_0 = R_0^n$.

We conclude the analysis of the proposed model by showing that, with suitable choices of parameters, its solutions can exhibit some interesting patterns (unaccessible to any classical SIR model), that are morphologically compatible with the evolution of past pandemics. For example, two epidemic waves can be obtained (see Fig. 5a) in the same epidemic episode. However, more than two epidemic waves can be obtained (as it was in fact observed in the 1918-19 Spanish pandemic). Moreover, the peak daily attack rate of the sequence of waves is not necessarily decreasing over time (see Fig. 5b). Difference in slope in the decaying phase (reminiscent of those observed in the Fall wave of the 1918-19 Spanish pandemic in the UK) can also be captured by our model (see Fig. 5c and (Ciofi degli Atti et al., 2008) for a brief discussion). Finally, very long decaying phases, making the epidemic curve strongly asymmetric, can also be obtained (see Fig. 5d).

4 Discussion

When studying the spread of epidemics, behaviour and contact patterns are typically considered "background" for the infection – i.e., they are not themselves variables of the dynamics. It is interesting, however, to address cases for which the population behaviour cannot be merely considered as an indepen-

dent (though time-varying) parameter, but it is better modelled as a variable whose evolution influences, and is influenced by, the dynamics of the infection.

With the introduction of an explicit model for behavioural changes, infection and behaviour both contribute to define the context for the other. Symmetry between these two key-factors is therefore restored, and no *by-principle* prevalence is given (even formally) to one over the other. Not only the dynamics of infection depends now on both the transmission and behaviour, but also the behaviour dynamics depends on behaviour (and infection as well). This is what makes evolutionary game theory especially suited to the case as compared to classical game theory. In fact, application of the latter would result in (rational) instantaneous best responses to the infection dynamics, regardless of the current distribution of behavioural strategies.

The model we propose is (deliberately) simple, and exhibits a transmission dynamics driven by an $S \to I \to R$ scheme coupled with behavioural (contact) patterns driven by imitation dynamics. Still, we were able to prove that the model accounts for multiple waves occurring within the same outbreak, and is able to explain "asymmetric waves", i.e., infection waves whose rising and decaying phases differ in slope. As an interesting feature, the attack rate for the model is always smaller than that of the equivalent SIR model (obtained by fixing $x(t) = 1$).

It should be observed that the model is based on two implicit, yet crucial assumptions: a) that the benefits of behavioral changes be immediately clear to the individuals; b) that individuals be able to recognize whether their contacts are susceptible, infective or removed (since susceptible individuals can change their behaviour only through encounters with other susceptible individuals).

Consequently, our model applies better to severe epidemics, in which it is more likely that these requirements are actually met.

Coming to discuss possible variants and extensions, a first remark concerns the dynamics of behavioural changes that we have adopted. In particular, the payoffs of the underlying game are modelled as the *perceived* risk of infection. Our choice was for a simple linear dependence from the fraction of currently infected individuals. Of course, a number of different options are available; for example one may tie the perception of risk to the number of new infections, or consider the actual probability of infection in place of perceived risk. Cumulation of risk over time could also be addressed by introducing appropriate memory mechanisms.

Independently of how the risk is specifically reckoned, the access to information pertaining the relative efficacy of behaviours may also be collected across more structured networks (e.g., the media). In this respect, considering different time units adds some flexibility to the model, in that it allows for different speeds in the diffusion of infection and behaviour. For example, tuning of key parameter ϵ may be obtained on the basis of empirical evidence.

At first sight, introduction of irrational behaviour changes may appear unnecessary, and contrasting with the model simplicity we tried to keep throughout. Yet, by avoiding extinction of allowed behaviours, irrational behaviour overtakes an unrealistic (and undesirable) effect of strict imitation: the pool of strategies from which an individual can choose is limited to those effectively represented in the population. By allowing exploration of *all possible* behaviours, irrational behaviours may account for erroneous decisions or idiosyncratic attitudes always present in human societies.

The focus of this work is to investigate the effects that behavioural change as a protective response to the state of infection has on the spread of a (severe) epidemic. That's why the behavioural change modelled here affects only susceptible individuals (infected individuals may of course change behaviour as an effect of their status, regardless of the state of epidemic). As a side remark, notice that quarantine or isolation of infected individuals can already be described by our model since they can be modelled as a reduction of the transmission parameters.

A wider class of models can also be considered. The model of behavioural changes can in fact be extended to infected individuals subdivided in symptomatic and asymptomatic, for example treating the infected asymptomatics as susceptibles for anything concerning the behavioural dynamics. A specific class for latent individuals could also be introduced, thereby delaying the epidemic spread and affecting behavioural changes. In general, considering more than two behavioural classes would provide greater flexibility and realism, while of course opening to technical problems of increased complexity.

The class of models introduced in this paper may contribute to elucidate phenomena for which a behavioural basis is apparent, as in reaction to alerts (Wallinga and Teunis, 2004), or hypothesized, as for superspreading events (Lloyd-Smith et al., 2005). In fact, empirical estimation of epidemic parameters (as, for example, the basic reproduction number) or the comparison between intervention strategies have to be carefully reconsidered whenever an underlying behavioural dynamics is suspected. Finally, a better understanding of the distinction between spontaneous and induced changes of behaviour is key for the implementation of more realistic and effective social distancing measures.

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A Proofs

Proof of Proposition 3.2

S1 Let us consider the set $T_1 = \{0\} \cup \{\bar{t} > 0 \mid I(t) < 1/m \; \forall \, t \in (0, \bar{t})\}$. $T_1 \neq \emptyset$ and let us define $t_1 = \sup T_1$. $I(0) := I_0 < 1/m$ implies that $t_1 > 0$. Let us consider any finite time $\tilde{t} < t_1$. For any $t \in [0, \tilde{t}]$, the boundary–layer system (9) with $I = I(t)$ admits the asymptotically stable equilibrium $x^\star(I)$, as defined in Eq. (10) and $x^\star(I) \to 1$ when $\mu \to 0$ (see Prop. 3.1). In fact, $I(t) < 1/m$ for each $t \in [0, \tilde{t}]$. Therefore, the solution of the degenerate system (8) is equivalent to that of a classical SIR model with $R_0 = R_0^n$ on the whole interval $[0, \tilde{t}]$. By Tikhonov theorem (Tikhonov, 1952), this is also the approximation of the solution of system (5). Condition $1/m < I_p$ guarantees that $t_1 < +\infty$. In fact, on $(0, t_1)$ system (5) is equivalent to an SIR model with $R_0 = R_0^n$; thus, if $t_1 = +\infty$, $I(t) = I_p > 1/m$ for some

finite time $t > 0$, against the hypothesis $t_1 = +\infty$. Moreover, by continuity $I(t_1) = 1/m$.

S2.1 Let us now assume that $R_0^a S(t_1) \leq 1$. As observed in the main text, the only admissible value for the fraction of infected individuals is $I(t) = 1/m + O(\epsilon)$ as long as $R_0^n S(t) > 1$. Formally, if $I(t) > 1/m$ in some interval then $x(t)$ would be very close to 0 in almost the whole interval. Thus, $\dot{I}(t)$ would be negative and $I(t)$ would decrease below $1/m$. On the other hand, if $I(t) < 1/m$ in some interval then $x(t)$ would be very close to 1. Thus, $\dot{I}(t)$ would be positive and $I(t)$ would increase over $1/m$.

This has two relevant consequences, namely

- in the slower time scale system we have $\dfrac{dI}{dt}(t) = 0$;
- in the faster time scale system we have $\dfrac{dx}{ds}(s) = 0$ since $\mu = o(\epsilon^k)$ with $k \geq 1$ (see Eq. 9). In particular, this implies that the under the hypotheses of the Proposition, the dynamics of x is not faster than that of the epidemic transmission when $I(t) \approx 1/m$.

By setting $\dot{I}(t) = 0$ in the degenerate system (8) we obtain

$$x = \frac{\gamma - \beta^a S}{\beta^n S - \beta^a S} \ . \tag{A.1}$$

By substituting this value in the equation for $S(t)$ and by setting $I(t) = 1/m$ we obtain

$$\dot{S}(t) = -\gamma/m \ ,$$

whose explicit solution is $S(t) = S(t_1) - \frac{\gamma}{m}(t - t_1)$ as long as $R_0^n S(t) > 1$, that is for all $t \in (t_1, t_2')$ where $t_2' = t_1 + \frac{m}{\gamma}(S(t_1) - \frac{1}{R_0^n})$. Afterwards, the fraction of infected $I(t)$ decreases below $1/m$ and we can apply the line of reasoning applied in *S1* to show that the solution of system (5) approximates that of

an SIR model in its decaying phase with $R_0 = R_0^n$.

One can iterate the procedure to compute the $O(\epsilon)$ terms. In fact, let us assume that $1 - mI = \epsilon v$. The equation for $x(t)$ allows us to estimate v. Indeed, we can rewrite the equation for $x(t)$ as:

$$\dot{x} = x(1-x)v + \epsilon^{k-1}(1-2x) \ , \tag{A.2}$$

where, if $k > 1$, the second righthand term can be ignored. Both $\dot{x}$ and $x(1-x)$ can be explicitly computed from Eq. (A.1). By substituting the resulting expressions in Eq. (A.2) and considering that $1 - mI = \epsilon v$ we have:

$$1 - mI = \frac{\epsilon\gamma(R_0^n - R_0^a)}{m(SR_0^n - 1)(1 - R_0^a S)} \ .$$

If $k = 1$ we can apply the same line of reasoning, though we do not obtain such a simple expression for $1 - mI$.

Going on, one can obtain $O(\epsilon)$ corrections for $x(t)$ and $S(t)$ in (t_1, t_2), but these are not really needed.

S2.2 If $R_0^a S(t_1) > 1$ we are guaranteed that the epidemic is still in its growing phase. Let us consider the set $T_2 = \{t_2\} \cup \{\bar{t} > t_2 \mid I(t) > 1/m \forall\ t \in (t_2, \bar{t})\}$. We can now apply the same line of reasoning applied in *S1*. The only difference is that $x^\star(I) \to 0$. Note that $t_2 < +\infty$ since $I(t) \to 0$ when $t \to +\infty$ and $I(t_2) = 1/m$.

S2.2.1 Similar to *S2.1*, after having observed that $R_0^a S(t_2) < 1$.

S2.2.2 Trivial. □

Lemma A.1 *If $R_0^n > 1$, the set of the solutions of the inequalities $1 < R_0^a < R_0^n \exp\{-R_0^a(1 - 1/R_0^n)\}$ is non empty.*

For fixed values of $R_0^n > 1$, let us consider the function

$$h_{R_0^n}(x) = x - R_0^n \exp\{-x(1 - 1/R_0^n)\} \ .$$

We have that $h_{R_0^n}(R_0^n) > 0$. We are interested to study the sign of the function $k(R_0^n) := h_{R_0^n}(1) = 1 - R_0^n \exp\{1/R_0^n - 1\}$. We have that $k(1) = 0$, $\lim_{R_0^n \to \infty} k(R_0^n) = -\infty$ and $\dot{k}(R_0^n) < 0$. It follows that $h_{R_0^n}(1) < 0$ for each $R_0^n > 1$. Therefore, it does exist $\bar{R}(R_0^n) \in (1, R_0^n)$ such that $h_{R_0^n}(\bar{R}) = 0$. It follows that choosing R_0^a with $1 < R_0^a < \bar{R}(R_0^n)$, we satisfy the inequality in the thesis. □

Proof of Proposition 3.3

Let us consider the time interval $[0, t_1]$ where t_1 is defined in the proof of Prop. 3.2. Since the system is equivalent to a SIR model with $R_0 = R_0^n$, we can employ the SIR invariant $S(t) + I(t) - \frac{1}{R_0^n} \log S(t) = const$ in $[0, t_1]$ to compute $S_1 := S(t_1)$. Since $S(0) = 1 - I_0$, $I(0) = I_0$ and $I(t_1) = 1/m$ it follows that S_1 is a zero of the function $f(x) = x + \frac{1}{m} - \frac{1}{R_0^n} \log x - 1 + O(I_0)$, where $O(I_0) = \frac{1}{R_0^n} \log(1 - I_0)$ can be ignored. Since $f(1) = 1/m > 0$ and $f(1/R_0^n) = 1/m - I_p < 0$ (by hypothesis) it follows that it does exist S_1 such that $f(S_1) = 0$ and

$$S_1 \in (1/R_0^n, 1) \tag{A.3}$$

with $\lim_{1/m \to 0} S_1 = 1$ and $\lim_{1/m \to I_p} S_1 = 1/R_0^n$. Since $f(x)$ is increasing for $x > 1/R_0^n$ the solution is unique.

Let us now assume that R_0^a satisfies the inequalities $1 < R_0^a < R_0^n \exp\{-R_0^a(1 - 1/R_0^n)\}$ (it is possible thanks to lemma A.1), which in particular implies $R_0^a < R_0^n$. Since $R_0^n > 1/S_1$ (see Eq. (A.3)), we can distinguish two cases:

- *Case 1*: $R_0^a > 1/S_1$,
- *Case 2*: $R_0^a \leq 1/S_1$.

In Case 2, we have a solution of type $C1$, satisfying the thesis.

Hence, we look only at Case 1. We are guaranteed that the epidemic is still in its growing phase. Let us consider the time interval $[t_1, t_2]$ where t_2 is defined in the proof of Prop. 3.2. Again, we can employ the SIR invariant in $[t_1, t_2]$ to compute $S_2 := S(t_2)$. Since $S(t_1)$ and $I(t_1)$ are known and $I(t_2) = 1/m$ it follows that S_2 is a non trivial solution of the equation $g(x) = g(S_1)$ where $g(x) = x + \frac{1}{m} - \frac{1}{R_0^a} \log x$. Function g is convex, has a absolute minimum for $x = 1/R_0^a$, is strictly decreasing for $x < 1/R_0^a$ and it is strictly increasing for $x > 1/R_0^a$, $\lim_{x\to 0} = +\infty$ and $\lim_{x\to+\infty} = +\infty$. Since $S_1 > 1/R_0^a$, a unique $S_2 \in (0, 1/R_0^a)$ exists such that $g(S_2) = g(S_1)$.

We now show that $S_2 > 1/R_0^n$. Since $R_0^a < R_0^n \exp\{-R_0^a(1 - 1/R_0^n)\}$ it follows that:

$$1 - \frac{1}{R_0^a} \log \frac{1}{R_0^a} < \frac{1}{R_0^n} - \frac{1}{R_0^a} \log \frac{1}{R_0^n} \ .$$

Since

$$1 - \frac{1}{R_0^a} \log \frac{1}{R_0^a} > S_1 - \frac{1}{R_0^a} \log S_1$$

we have that $g(S_2) = g(S_1) < g(1/R_0^n)$ and thus $S_2 > 1/R_0^n$ since g is decreasing in $(0, 1/R_0^a)$.

We have thus demonstrated that $R_0^n S_2 > 1$ which implies that we have a solution of type *C2*.

Case 2. Trivially, we have a solution of type *C1*. □

Lemma A.2 *When $S(t) = 1/R_0^n$, the solution of system (5) satisfies $I(t) < 1 - \frac{1}{R_0^n} + \frac{1}{R_0^n}\log\frac{1}{R_0^n}$.*

Equations of system (5) for I and S can be written in the general form

$$\begin{cases} \dot{S} = -\beta(t)SI \\ \dot{I} = -\beta(t)SI - \gamma I \end{cases} \tag{A.4}$$

with $\beta(t) \in [\beta_a, \beta_n]$. It is easy to show that for system (A.4) the function

$$S(t) + I(t) - \frac{1}{R_0^n}\log S(t)$$

is decreasing in t. It follows that

$$1 = S(0) + I(0) - \frac{1}{R_0^n}\log S(0) > S(t) + I(t) - \frac{1}{R_0^n}\log S(t)\ .$$

The thesis follows by substituting $S(t) = 1/R_0^n$. □

Proof of Proposition 3.4

Let us define $(S^{\mathrm{SIR}}(t), I^{\mathrm{SIR}}(t))$ and $(S(t), I(t))$ as the fractions of susceptible and infected individuals for a classical SIR model and for system (5), respectively. In the phase plane (S, I) the solution of a classical SIR model goes through the point $(\frac{1}{R_0^n}, 1 - \frac{1}{R_0^n} + \frac{1}{R_0^n}\log\frac{1}{R_0^n})$, corresponding to the epidemic peak. The solutions of system (5) pass through the point $(1/R_0^n, \tilde{I})$ where $\tilde{I} < 1 - \frac{1}{R_0^n} + \frac{1}{R_0^n}\log\frac{1}{R_0^n}$ thanks to lemma A.2.

Let us assume that $S_\infty < S_\infty^{\mathrm{SIR}}$. It follows that in the phase plane the trajectories of the two models must intersect at a certain point $(S^\star, I^\star)$ with $S^\star < 1/R_0^n$. Moreover, at this point both S and I are decreasing and thus we

can assume that the functions $I^{\mathrm{SIR}}(S^{\mathrm{SIR}})$ and $I(S)$ are well defined, and that

$$\frac{dI^{\mathrm{SIR}}}{dS^{\mathrm{SIR}}}(S^\star) > \frac{dI}{dS}(S^\star) \ .$$

hence,

$$-1 + \frac{\gamma I^\star}{S^\star I^\star \beta_n} > -1 + \frac{\gamma I^\star}{S^\star I^\star (\beta_n x + \beta_a (1-x))}$$

which is absurd since $\beta_a < \beta_n$ and $x \in (0,1)$. □

Proof of Proposition 3.5

Let us consider system (5). We have already seen that a time t_3 exists such that $I(t_3) = 1/m$ and $S(t_3) = 1/R_0^n$ (see Prop. 3.2). Moreover, for $t > t_3$ system (5) can be approximated by an SIR model with $R_0 = R_0^n$. Thus we can employ the SIR invariant in $[t_3, +\infty)$. It follows that $S_\infty(m)$ is solution to the equation:

$$S_\infty(m) - \frac{1}{R_0^n} \log S_\infty(m) = S(t_3) + I(t_3) - \frac{1}{R_0^n} \log S(t_3) = \frac{1}{R_0^n} + \frac{1}{m} - \frac{1}{R_0^n} \log \frac{1}{R_0^n}$$

while S_∞^{SIR} is solution of the equation

$$S_\infty^{\mathrm{SIR}} - \frac{1}{R_0^n} \log S_\infty^{\mathrm{SIR}} = 1 \ .$$

Therefore, we have to compare the solutions of the equations:

$$l(x) = 1 \ , l(x) = b(m)$$

where $l(x) = x - \frac{1}{R_0^n} \log x$ and $b(m) = \frac{1}{R_0^n} + \frac{1}{m} - \frac{1}{R_0^n} \log \frac{1}{R_0^n}$.

Condition $1/m < I_p$ implies that $b(m) < 1$. Function l is convex, has an absolute minimum at $x = 1/R_0^n$ with $l(1/R_0^n) < b(m)$, it is strictly decreasing for $x < 1/R_0^n$ and $\lim_{x \to 0^+} = +\infty$.

Since we are interested in solutions $x < 1/R_0^n$, we have that $b(m) < 1$ implies $S_\infty(m) > S_\infty^{\mathrm{SIR}}$. Moreover, $b(m)$ is an decreasing function of m and thus $S_\infty(m)$ is an increasing function of m on $(\frac{1}{I_p}, +\infty)$.

Finally, $b(m) \searrow l(1/R_0^n)$ when $m \to \infty$; thus $S_\infty(m) \to 1/R_0^n$ when $m \to \infty$.

□

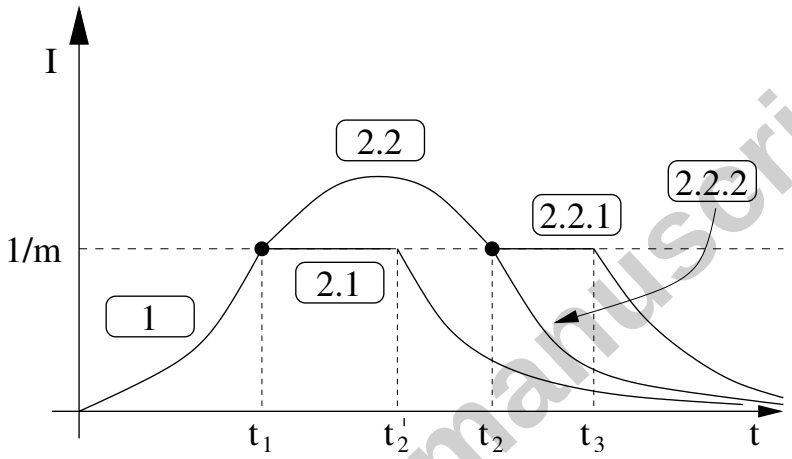

Fig. 1. Possible temporal evolution of the fraction of infected individuals I. Regions above and below $1/m$ correspond to $x^\star(I) \to 0$ and $x^\star(I) \to 1$, respectively. In the two regions the solutions of system (5) approximate those of classical SIR models with basic reproductive numbers $R_0 = R_0^a$ and $R_0 = R_0^n$ respectively.

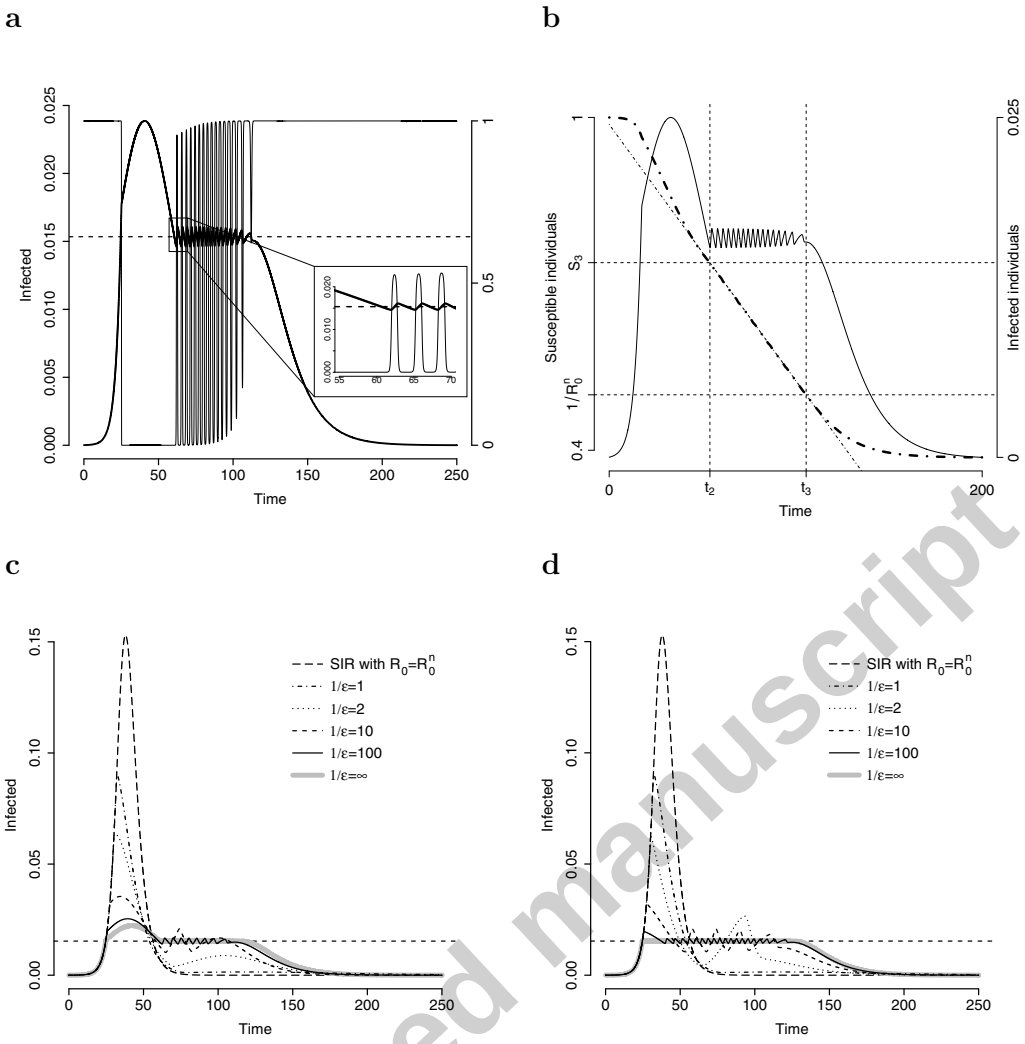

Fig. 2. **a** Fraction of infected individuals (solid bold line, scale on the left) and fraction of individuals playing strategy b_n (solid tiny line, scale on the right) over time for system (5). Parameters employed: $\beta_n = 0.6$, $\gamma = 0.3$, $\beta_a = 0.35$, $\epsilon = 3.33 \cdot 10^{-3}$, $m = 65$, $\mu = 10^{-7}$. The dashed line represents the threshold value $1/m$. **b** Fraction of infected individuals (solid line, scale on the right) and susceptible individuals (bold dot-dashed line, scale on the left) in the same example as in panel **a**. We also plot the straight line $S(t) = S(t_1) - \frac{\gamma}{m}(t - t_1)$ (tiny dot-dashed line, scale on the left)to show the linearity of $S(t)$ in $[t_2, t_3]$ as predicted by Prop. 3.2. **c** Fraction of infected individuals vs. time for different choices of the parameter ϵ (thin black lines) and the piecewise solution of system (5) (heavy gray line) as in Fig. 1; other parameters as in panel **a**. **d** Like panel **c** but with $\beta_a = 0.3$; this implies $R_0^a S(t_1) < 1$ so that the solution is of type C1.

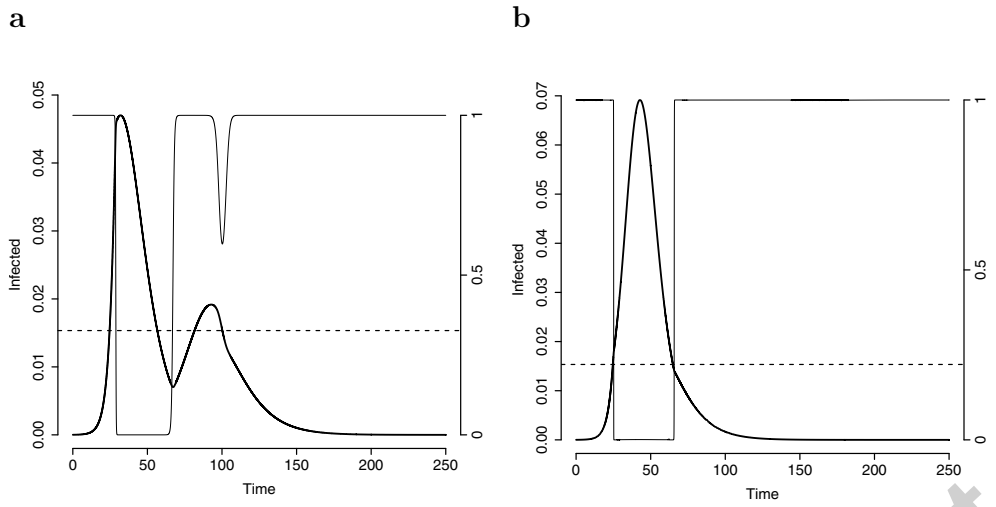

Fig. 3. Other possible behaviour of solutions of system (5). **a** As in Fig. 2a but with $\epsilon = 0.25$. **b** As in Fig. 2a, but with $\beta_a = 0.45$.

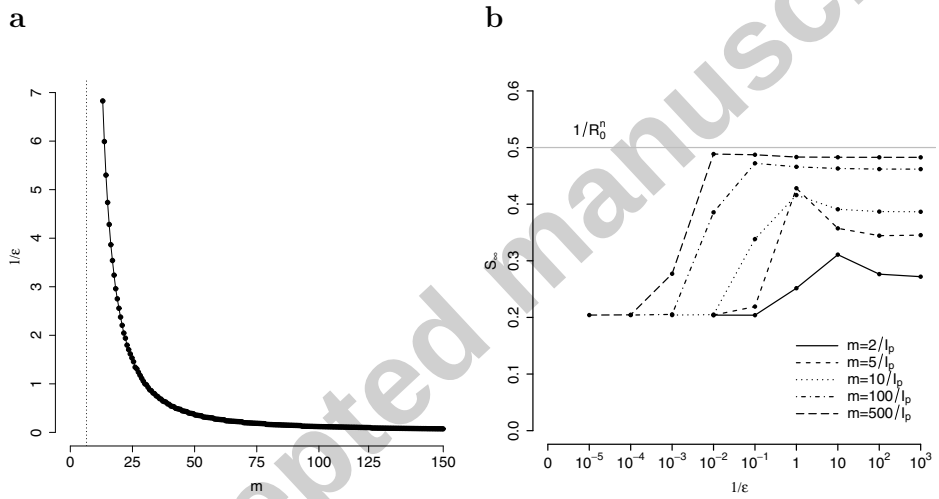

Fig. 4. **a** The minimum values of $1/\epsilon$ giving rise to a sequence of at least two epidemic waves are plotted against m, for system (5). Parameters employed: $\beta_n = 0.6$, $\gamma = 0.3$, $\beta_a = 0.33$, $\mu = 10^{-7}$. The vertical dotted line represents the value of m such that $m = 1/I_p$. Notice that for such choice of parameters, the conditions of Prop. 3.3 are satisfied, which implies that epidemic waves will occur for $\epsilon \to 0$. Notice how multiple waves can occur even for "slow" changes in behaviour (large ϵ values). **b** S_∞ as a function of $1/\epsilon$ for different choices of m for system (5). Parameters employed: $\beta_n = 0.6$, $\gamma = 0.3$, $\beta_a = 0.35$, $\mu = 10^{-7}$.

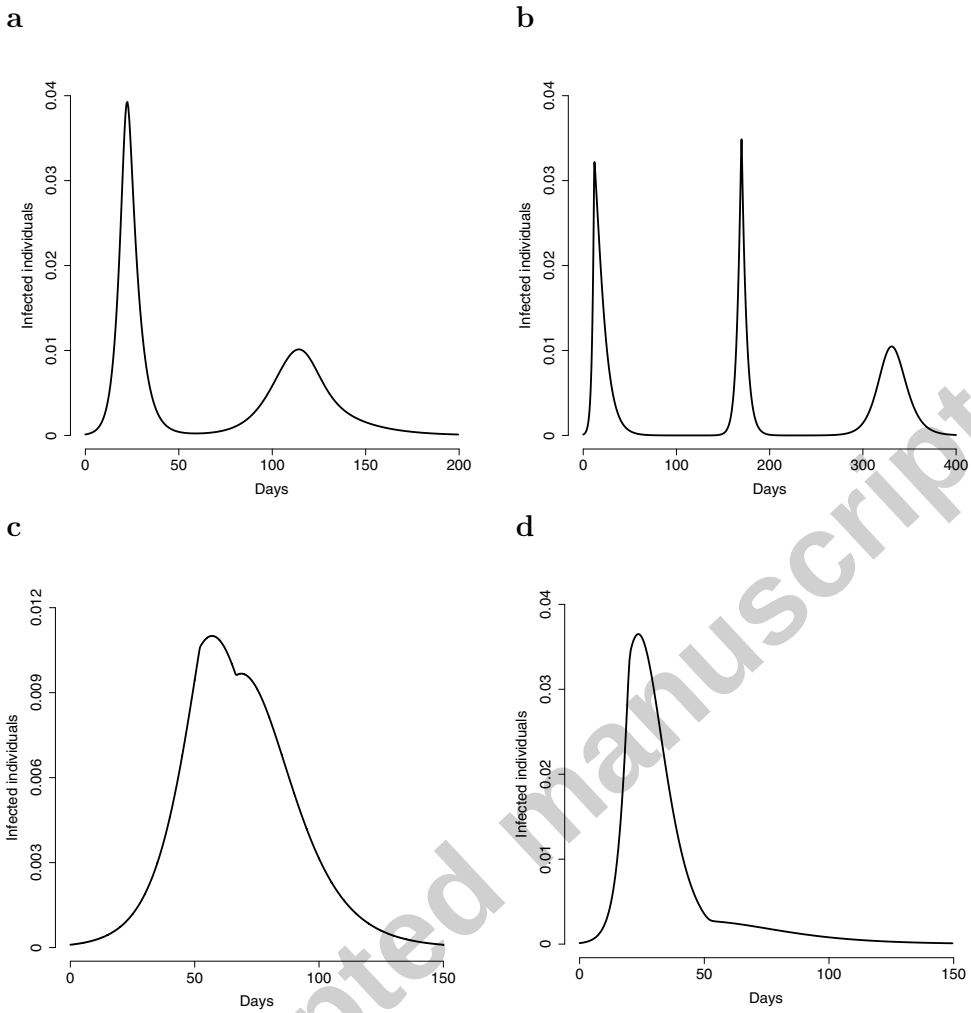

Fig. 5. With suitable choices of parameters, the model (5) can account for interesting epidemic patterns. Here 4 examples are shown to illustrate the potential of the model. **a** Parameters employed: $m = 150$, $\beta_n = 0.8$, $\beta_a = 0.4$, $\gamma = 0.5$, $\mu = 0.01$, $\epsilon = 10$. **b** Parameters employed: $m = 300$, $\beta_n = 1$, $\beta_a = 0.48$, $\gamma = 0.5$, $\mu = 10^{-10}$, $\epsilon = 2$. **c** Parameters employed: $m = 100$, $\beta_n = 0.6$, $\beta_a = 0.54$, $\gamma = 0.5$, $\mu = 10^{-5}$, $\epsilon = 0.01$. **d** Parameters employed: $m = 100$, $\beta_n = 0.8$, $\beta_a = 0.6$, $\gamma = 0.5$, $\mu = 10^{-5}$, $\epsilon = 1$.