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Renewal equations for delayed population behaviour adaptation coupled with disease transmission dynamics: a mechanism for multiple waves of emerging infections

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Abstract

There are many plausible reasons for recurrent outbreaks of emerging infectious diseases. In this paper, we develop a mathematical model to illustrate how population behavioural adaption and adaptation implementation delay, in response to the perceived infection risk, can lead to recurrent outbreak patterns. We consider the early phase of an infection outbreak when herd immunity is not reached, pathogen mutation is not considered, and seasonality is ruled out as a major contributor. We derive a transmission dynamics model coupled with the renewal equation for the disease transmission effective contacts (contact rate per unit time multiplied by the transmission probability per contact). The model incorporates two critical parameters: the population behavioural adaptation flexibility index and the behavioural change implementation delay. We show that when the behavioural change implementation delay reaches a critical value, the number of infections starts to oscillate in an equilibrium that is determined by the population behavioural adaptation flexibility. We also show that the numbers of infections at the subsequent peaks can exceed that of the first peak. This was an oblique observation globally during the early phase of the COVID-19 pandemic before variants of concern emerged, and it was an observed phenomena with the Omicron variant induced wave in areas where early interventions were successful in preventing the large outbreaks. Our model and anayses can provide partially explanation for these observations.

Keywords: Multiple waves, Behavioural adaptation, Implementation delay

1. Introduction

With a few exceptions, many countries with some degree of success in containing the first wave of the COVID-19 pandemic experienced either a second or further waves with higher amplitudes. This is expected since the population has not reached the herd immunity through either natural infection or mass immunization, while social distancing interventions were escalated to cope with the exponential growth, and then deescalated for partially reopening the economy and social economic activities.

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A fundamental issue that should be addressed is the mechanism behind the seemingly universal resurgence, and how to determine the inter-outbreak interval and peak values of the subsequent waves from some intrinsic characteristics of the community tolerance (flexibility) to fluctuating incidence, and collective response implementation delay, along with the disease basic reproduction number. Classical epidemic models fail to explain the resurgence. While existing studies (see discussions in the final section) and some developed extensions of the classical models incorporating behavioural changes can explain the recurrent patterns as damped oscillations or sustained periodic oscillations, these studies failed to explain why the second wave had a peak value higher than that of the first wave.

Here, we develop a novel approach to formulate the population behavioural changes in terms of a renewal equation for the disease transmission effective contact that is adaptive to the infection risk or disease severity, reported or perceived, with a delay. We introduce a parameter that measures the community flexibility to this (real and/or perceived) infection risk and show that this determines the equilibrium level of the disease transmission when the effective transmission contact reaches the equilibrium determined by the pathogen characteristics (basic reproduction number) and infection removal speed. We then show that the likelihood of recurrent outbreaks and the inter-outbreak interval is determined by the speed that effective transmission contact, determined by the speed at which infection risk is detected (testing and confirmation) and the speed at which non-pharmaceutical interventions can be implemented massively in the population.

2. The framework: community flexibility and renewal equation for effective contacts

We consider a simple SIR model for the outbreak of an infectious disease. We consider the situation where public health interventions, reflected by the behavioural changes of the population in terms of social distancing and personal protection, will be used to prevent a large portion of infections before herd immunity is achieved and variants of concern emerge. Therefore, the susceptible population remains relatively unchanged and the SIR model reduces to

$$I'(t) = \beta I(t) - \gamma I(t),$$

where γ is the remove rate of the infectious individuals (being isolated, or recovered, or dead), and β is the disease transmission effective contact number, i.e.,

$$\begin{aligned}\beta = &\text{ number of contacts per unit time } (c)\\ &\times \text{transmission probability per contact } (p).\end{aligned}$$

We notice that this disease transmission effective contact changes as individuals adjust their behaviors according to the incidence: the higher the incidence, the smaller the effective contact. Behaviorual changes induced by disease severity will also be incorporated into our behavioural change adaptation renewal equation in many different ways. First of all, at the beginning of an outbreak and in the absence of knowledge of disease severity, the number of infections (more precisely, the reported cases) is the only information available to the public to induce behavioural changes. Secondly, the daily cases of infections with disease severity (reflected by the hospital admission, Intensive Care Unit admission, or mortality) are fractions of the daily incidence with delay. We can incorporate this delay in the renewal equation for behavioural adaptation. Namely, the behaviour change adjusts to the incidence with a delay, due to either the delay in case confirmation, or the operational limit for rapid interventions. Therefore, we assume that the effective contact $\beta = cp$ is a decreasing function of the incidence βI with time lag $\tau \geq 0$, i.e.,

$$\beta(t) = f(\beta(t-\tau)I(t-\tau)) \tag{1}$$

with f being a decreasing function of the variable. This gives the renewal equation for the effective contact.

Specific forms of the behavioural adaptation (to incidence) function f are determined from a number of factors including the population tolerance to the incidence and the perceived risk associated with the incidence. The form can be informed from detailed studies of the population behaviours during different phases of the disease outbreaks. Our focus here is to show that insights can be gained by using the most general format in a simple mechanistic model.

In particular, it is natural to assume that $f(0) = \beta_0 > 0$ is the maximum effective contact number per day, $f(x) \geq 0$ for all $x \geq 0$, f is continuously differentiable on $[0, \infty)$, and $f'(x) < 0$ for all $x \geq 0$ so the larger the incidence the smaller the effective contact. With this notation, we note that $R_0 = \beta_0/\gamma$ is the *basic reproduction number*.

We also note that the decreasing property of f implies that for any $x \geq 0$ there exists a unique $b = b(x)$ such that $b = f(bx)$. Clearly, $b(0) = \beta_0$. Substituting $b = b(I)$ to (1), we will be able to find an algebraic equation for the non-trivial equilibrium $b(I^*) = \gamma$, and the corresponding non-trivial equilibrium is $(b(I^*), I^*)$ for the epidemic model coupled with the renewal equation for behavioural changes:

$$\begin{cases} I'(t) = \beta(t)I(t) - \gamma I(t), \\ \beta(t) = f(\beta(t-\tau)I(t-\tau)). \end{cases} \tag{2}$$

It is convenient to introduce the *flexibility index* $k > 0$, called *f-index* hereafter, to measure the degree of the population adaptation to the incidence and write

$$f(x) = F(kx), \qquad x \geq 0.$$

Then the behavioural change function can be parametrized by this f-index $k > 0$. Let $g_k(x)$ be the solution of $b = f(bx) := F(kbx)$. We have

$$b(x) = g_k(x) = g_1(kx), \qquad x \geq 0.$$

Since $b(I^*) = \gamma$ gives the non-trivial equilibrium value of I^*, we can now see how increasing the f-index reduces the equilibrium state the community can tolerate, and what the corresponding equilibrium disease transmission contact is.

In what follows, we will use the prototypical function $F(x) = \frac{\beta_0}{1+x}$, with $\beta_0 = c_0 p_0$ being the maximal disease transmission contacts per day. The corresponding renewal equation for the behavioural change is

$$\beta(t) = \frac{\beta_0}{1 + k\beta(t-\tau)I(t-\tau)}.$$

Remark 1. *We emphasize that we have used the simplest epidemic model to describe the disease transmission, and have implicitly ignored the presence of asymptomatic infections. Furthermore, population behaviours may change in response to incidence. In this case, the delay τ is the sum of the mean duration from exposure until being reported plus the average behavioural change implementation delay. In this case, the f-index is the measure of the community tolerance to infections so that when the incidence reaches $1/k$, the effective contact is half of the effective contact in the absence of infections:*

$$1 + k \times (\textit{daily incidence}) = 2.$$

Similarly, if the population behaviours change in response to daily number of patients in the ICUs, then the delay τ is the sum of the mean duration from exposure to ICU admission, plus the average behavioural change implementation delay. In this case, since only a portion δ of the confirmed cases can end up in the ICUs, the f-index is the measure of the community tolerance to ICU admission so that when the reciprocal of the number of admission reaches $1/k$ the effective contact is half of the effective contact in the absence of infections:

$$1 + k \times \textit{(ICU cases)} = 2.$$

3. Recurrent patterns

Recall that $R_0 = \beta_0/\gamma$ is the basic reproduction number when no behavioural changes have occurred. We will assume $R_0 > 1$ so there will be an outbreak with an introduction of new cases. Clearly, the coupled system has a disease free and effective contact change free equilibrium $(0, \beta_0)$, and it can be shown that this equilibrium is unstable.

To look for the disease-"endemic" equilibrium, we first solve the first equation I for the equilibrium to get $\beta^* = \gamma$ and substitute this to the second renewal equation for β to get

$$\beta^* = \frac{\beta_0}{1 + k\beta^* I^*},$$

from which we get $\gamma(1 + k\gamma I^*) = \beta_0$, so

$$I^* = \frac{R_0 - 1}{k\gamma} > 0. \tag{3}$$

This leads to

Theorem 1. *When $R_0 > 1$, the coupled system has a non-trivial equilibrium $(I^*, \beta^*) = (\frac{R_0-1}{k\gamma}, \gamma)$, where the infection reaches an equilibrium determined by the basic reproduction number and the community flexibility, and the effective contact reaches the equilibrium value γ so the effective reproductive number β^*/γ reaches the threshold of unity.*

It is evident that it is the community flexibility that determines on the equilibrium value of the infection, the larger the k the smaller the value of infected cases at the equilibrium. Obviously, the transmissibility of the disease R_0 also impacts on the equilibrium value.

We now linearize the coupled system at the positive equilibrium (I^*, β^*) and this gives

$$\begin{cases} I'(t) = \beta^* I(t) + I^* \beta(t) - \gamma I(t) = I^* \beta(t), \\ \beta(t) = -\frac{k\beta_0}{(1+k\beta^* I^*)^2}[I^* \beta(t-\tau) + \beta^* I(t-\tau)]. \end{cases}$$

Noting that

$$1 + k\beta^* I^* = \frac{\beta_0}{\beta^*} = \frac{\beta_0}{\gamma} = R_0,$$

we conclude that

$$\frac{k\beta_0 I^*}{(1+k\beta^* I^*)^2} = \frac{k\beta_0}{R_0^2}\frac{R_0-1}{k\gamma} = \frac{R_0-1}{R_0},$$

$$\frac{k\beta_0\beta^*}{(1+k\beta^* I^*)^2} = \frac{k\beta_0\gamma}{R_0^2}.$$

Therefore, the linearized system becomes

$$\begin{cases} I'(t) = \frac{R_0-1}{k\gamma}\beta(t), \\ \beta(t) = -\frac{R_0-1}{R_0}\beta(t-\tau) - \frac{k\beta_0\gamma}{R_0^2} I(t-\tau). \end{cases}$$

To investigate the stability, we consider solution $I(t) = e^{\lambda t} i_0, \beta(t) = e^{\lambda t} b_0$ with non-zero (i_0, b_0). This leads to the algebraic system

$$\begin{cases} \lambda i_0 = \frac{R_0-1}{k\gamma} b_0, \\ b_0 = -\frac{R_0-1}{R_0} e^{-\lambda\tau} b_0 - \frac{k\beta_0\gamma}{R_0^2} e^{-\lambda\tau} i_0. \end{cases}$$

From the second equation of the above algebraic system, we get

$$b_0 = -\frac{\frac{k\beta_0\gamma}{R_0^2} e^{-\lambda\tau}}{1 + \frac{R_0-1}{R_0} e^{-\lambda\tau}} i_0.$$

This implies that $i_0 \neq 0$ and the characteristic equation is

$$\lambda(1 + \frac{R_0-1}{R_0} e^{-\lambda\tau}) = -\frac{R_0-1}{R_0^2}\beta_0 e^{-\lambda\tau}. \tag{4}$$

As the above equation has only one negative real zero when $\tau = 0$, the characteristic equation has zeros with non-negative real parts while increasing τ only when there is a critical value τ when the characteristic equation has a pair of eigenvalues $\pm i\omega$.

We now look for such a critical value. Let $\lambda = i\omega$, then we get

$$(i\omega)(1 + \frac{R_0-1}{R_0} e^{-i\omega\tau}) = -\frac{R_0-1}{R_0^2}\beta_0 e^{-i\omega\tau}.$$

That is

$$(i\omega)(1+\frac{R_0-1}{R_0}(\cos(\omega\tau)-i\sin(\omega\tau)))=-\frac{R_0-1}{R_0^2}\beta_0(\cos(\omega\tau)-i\sin(\omega\tau)).$$

Separating the real and imaginary parts yields

$$\begin{cases} \omega\frac{R_0}{R_0-1}=-\omega\cos(\omega\tau)+\frac{\beta_0}{R_0}\sin(\omega\tau), \\ 0=\omega\sin(\omega\tau)+\frac{\beta_0}{R_0}\cos(\omega\tau). \end{cases}$$

This yields

$$(\omega\frac{R_0}{R_0-1})^2=\omega^2+(\frac{\beta_0}{R_0})^2,$$

and hence the critical value of ω is given by

$$\omega^*=\frac{\beta_0/R_0}{\sqrt{(\frac{R_0}{R_0-1})^2-1}}.$$

In addition, we have from $0=\omega\sin(\omega\tau)+\frac{\beta_0}{R_0}\cos(\omega\tau)$ that

$$\tan(\omega^*\tau)=\frac{-\beta_0/R_0}{\omega^*}=-\sqrt{(\frac{R_0}{R_0-1})^2-1}.$$

Therefore, the minimal critical value for τ is given by

$$\tau^*=\frac{\arctan(-\sqrt{(\frac{R_0}{R_0-1})^2-1})}{\omega^*},$$

with

$$\arctan(-\sqrt{(\frac{R_0}{R_0-1})^2-1})\in(\pi/2,\pi).$$

The transversality condition for Hopf bifurcation of periodic solutions [1, 2] can be easily verified, so we conclude that

Theorem 2. *In the neighbourhood of the nontrivial equilibrium (β^*, I^*), when the delay passes through the critical value τ^*, the coupled system of disease transmission dynamics with the effective contact renewal equation has a Hopf bifurcation of periodic solutions with periods close to $\frac{2\pi}{\omega^*}$.*

We also note that $\frac{2\pi}{\omega^*\tau^*}\to 4$ as $R_0\to 1^+$; and $\frac{2\pi}{\omega^*\tau^*}\to 2$ as $R_0\to\infty$. So we can see the inter-wave distance with large or small basic reproduction number. We avoid the use of normal formal calculation to check the stability of the bifurcated periodic solutions, but rather rely on some numerical simulations to demonstrate the stability and patterns of bifurcated temporal oscillations.

In the simulations summarized in Figure 1, we use $R_0=2.8$, in the estimated range of basic reproduction number for the COVID-19 when it first emerged and then imported to many regions in the world [3, 4, 5]. In Figure 1, we assume the infected individuals are removed form the transmission chain at the speed of $1/14$ (days^{-1}), that is the average time between exposure and recovered/isolation (after test confirmation) is assumed 14 days. The assumption that $k=0.1$ and $\tau=20$ (45, respectively) could be interpreted as follows: with $\delta=0.01$ (1% of the confirmed cases was admitted to the ICUs), the population reduces its effective contact to half of its pre-pandemic level when the incidence reached 1,000 (leading to 10 daily ICU admissions later), and the behavioural changes reacted to the ICU increase with a delay by 20 (45, respectively)-days minus the time from exposure to ICU admission. Importantly, we notice that

1). the values of k and γ determine the equilibrium value to be 252;

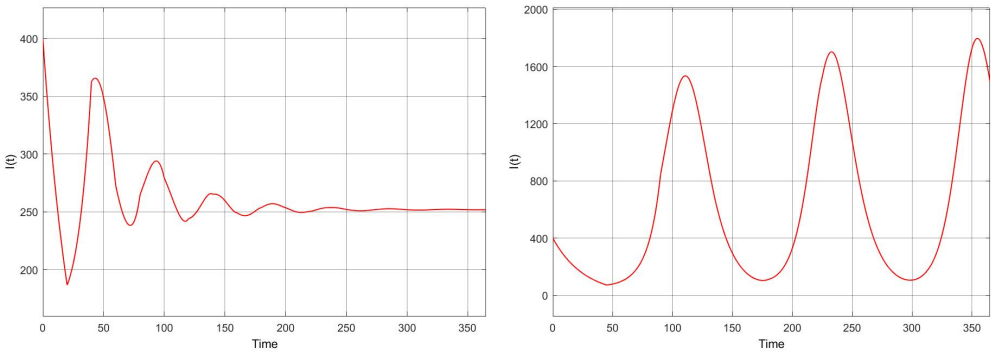

Figure 1. Solutions of model (2) with $k = 0.1$, $\gamma = 1/14$, $\beta_0 = 0.2$. Left panel: $\tau = 20$; Right panel: $\tau = 45$.

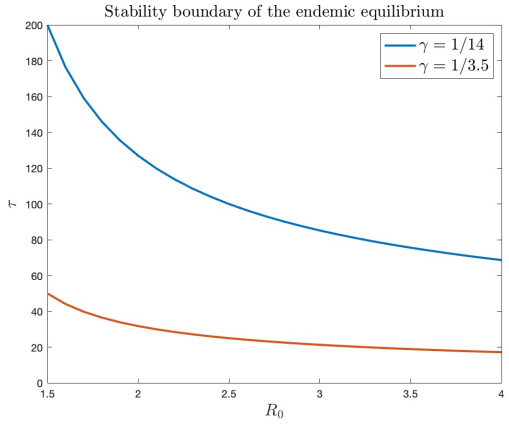

Figure 2. Stability boundaries of the positive equilibrium with $k = 0.1$, $\beta_0 = 0.2$, $\gamma = 1/14$(the blue curve) and $\gamma = 1/3.5$(the red curve).

2). when the delay τ is 20, the transmission dynamics reaches the stable equilibrium with a damped oscillation (left panel);
3). when the delay increases to 45, the transmission reaches a limit cycle after some transition; and importantly
4). the peak values of the subsequent waves increase until the transition to a stable limit cycle.

The stability boundary of the positive equilibrium is given in Figure 2.

In Figure 3, the removal rate is increased to $1/3.92$ (day^{-1}) while the basic reproduction number remains 2.8. With $k = 0.01$ that corresponds to reducing the transmission effective contact to half when the incidence reaches 100, the behavioural change delay with $\tau = 14$ (days) can also lead to a transition of multiple waves with increasing peak values until the system reaches the stable recurrent oscillations with peak-to-peak interval approximating 40 days.

In contrast, we simulated the ”dynamic zero” policy when the population reacts to confirmed cases with much stringent public health measures ($k = 1$) and rapid response (removal of infectious individuals within 4 days), we note from Figure 4 (left panel) that the outbreak had a small first outbreak, followed by damped oscillations in transition to a small equilibrium value (with incidence value approximating 14). Increasing the behavioural delay from 7-days to 11 days may destabilize the equilibrium for a stable limit cycle, but with small amplitudes (right panel).

4. Discussions

During the 2003-04 SARS-1 outbreak, most cities with SARS-importation experienced 2 waves within a single season [6]. This first global public health emergency of the century shows that classical epidemic models do not apply

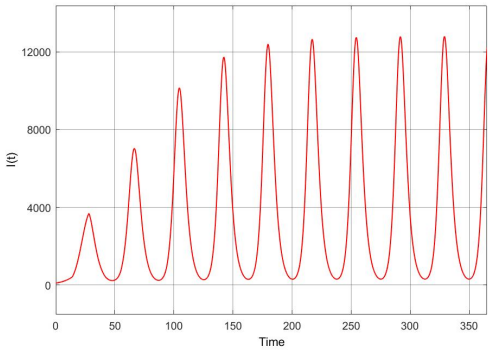

Figure 3. Solution of model (2) with $k = 0.01$, $\gamma = 1/3.92$, $\beta_0 = 0.7143$, $\tau = 14$.

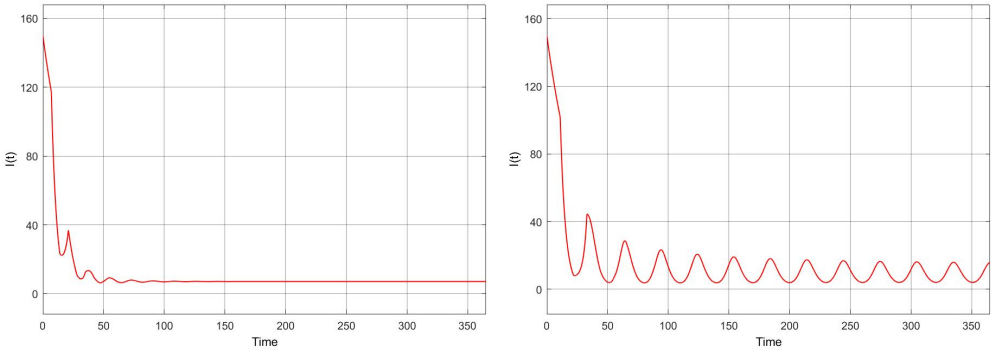

Figure 4. Solutions of model (2) with $k = 1$, $\gamma = 0.2551$, $\beta_0 = 0.7143$. Left panel: $\tau = 7$. Right panel: $\tau = 11$.

to disease outbreaks in the modern era of global connection and massive digital media to inflict behavioural changes. The classical epidemic model predicts a typical outbreak curve with a single wave: the number of infections increasing exponentially until the susceptible population is depleted and then the disease disappears also exponentially. It was suggested that behavioural changes played a role in the occurrence of the ”twin”-peaks. Some mechanistic models were proposed to explain how multiple waves occur when the population behaviours change in response to different perceived infection and disease risks [7].

It was subsequently observed during the 2009-10 H1N1 influenza pandemic that the behavioural changes could be driven by the media: the media, behaviour and disease co-evolves. However, both media coverage and the population behaviour develop rapid fatigue so social distancing measures and population adherence ”switching space” may locate in the region undesirable for avoidance of subsequent, and potentially larger, waves [8]. This lesson was not taken seriously in many countries during the first year of the global COVID-19 pandemic, so the early phase of the global pandemic was characterized by multiple waves [9] even before the emergency of variants of concerns. It has been recognized that behavioural changes must be integrated into epidemic models in order to better predict the epidemic trends and to inform better public health risk communication strategies [10, 11, 12].

Here we propose a coupled system of differential-algebraic system to couple the disease transmission dynamics compartmental model with the behavioural change dynamics in response to infection risk perception. Our model incorporates two key indices to measure the population flexibility (κ) for behavioural changes and resilience, and to measure the rapid response efficiency (τ) of behavioural changes to the infection risk in the population. Using this model, we were able to show that the new equilibrium state (after one outbreak) is determined by the population flexibility for behavioural change, while the rapid response efficiency determines whether a recurrent pattern will emerge, and the peak-to-peak intervals. In contrast with a classical epidemic model with the constant delay, our

model can exhibit escalating multiple waves. The model can be extended in many different ways to include more complicated epidemic models (more disease compartments, and more heterogeneous transmission settings) and more dynamic interactions of information propagation dynamics.

Behaviours may change more abruptly than the linear function $1/(k\beta I)$ due to, for example, lockdowns. We can consider the $f(x) = F(kx)$ with F defined by the sigmoidal function

$$F(x) = \frac{\beta_0(1+e^{-\alpha})}{1+e^{x-\alpha}}.$$

Then $F(0) = \beta_0$ is the maximum effective transmission coefficient per day, and $F(x) \to 0$ as $x \to \infty$. The basic reproduction number is still $R_0 = \beta_0/\gamma$, when no behavioural changes have occurred. To look for the positive equilibrium, we first solve the first equation I for the equilibrium to get $\beta^* = \gamma$ and then substitute this to the second renewal equation for β to get

$$\beta^* = \frac{\beta_0(1+e^{-\alpha})}{1+e^{k\beta^* I^* - \alpha}},$$

from which we get $e^{k\gamma I^* - \alpha} = R_0(1+e^{-\alpha}) - 1$ and hence

$$I^* = \frac{1}{k\gamma}\left[\alpha + \log(R_0(1+e^{-\alpha}) - 1)\right] = \frac{1}{k\gamma}\log K > 0,$$

for

$$K := e^{\alpha}(R_0 - 1) + R_0 = R_0(e^{\alpha}+1) - e^{\alpha} > 1.$$

Therefore, when $R_0 > 1$ the coupled system has a non-trivial equilibrium $(I^*, \beta^*) = (\frac{\log K}{k\gamma}, \gamma)$, where the infection reaches an equilibrium determined by the basic reproduction number and the community flexibility, and the effective contact reaches the equilibrium value γ so the effective reproductive number β^*/γ reaches the threshold of unity. Again, the larger the k the smaller the infection at the equilibrium. To compute the linearization, note that

$$\frac{1}{R_0} = \frac{1+e^{-\alpha}}{1+e^{k\beta^* I^* - \alpha}}$$

and

$$f'(x) = \frac{k\beta_0(1+e^{-\alpha})e^{kx-\alpha}}{(1+e^{kx-\alpha})^2} = \frac{k\beta_0(1+e^{-\alpha})}{1+e^{kx-\alpha}} - \frac{k\beta_0(1+e^{-\alpha})}{(1+e^{kx-\alpha})^2}.$$

Therefore,

$$f'(\beta^* I^*) = -\frac{k\beta_0}{R_0}\left(1 - \frac{1}{R_0(1+e^{-\alpha})}\right) = -\frac{k\beta_0 K}{R_0^2(1+e^{\alpha})},$$
$$f'(\beta^* I^*)\beta^* = -\frac{k\beta_0 K\gamma}{R_0^2(1+e^{\alpha})}, \quad f'(\beta^* I^*)I^* = -\frac{K\log K}{R_0(1+e^{\alpha})}.$$

The linearization at the positive equilibrium (I^*, β^*) becomes

$$\begin{cases} I'(t) = \beta^* I(t) + I^*\beta(t) - \gamma I(t) = I^*\beta(t), \\ \beta(t) = f'(\beta^* I^*)[I^*\beta(t-\tau) + \beta^* I(t-\tau)]. \end{cases}$$

The characteristic equation becomes

$$\lambda\left(R_0(1+e^{\alpha}) + K\log K e^{-\lambda\tau}\right) = -K\log K\gamma e^{-\lambda\tau}.$$

The critical value τ^* when the linearization loses its stability due to the existence of a pair of purely imaginary zeros of the characteristic equation is given by

$$\omega^* = \frac{\gamma K\log K}{\sqrt{R_0^2(1+e^{\alpha})^2 - (K\log K)^2}} = \frac{\gamma}{\sqrt{\left(\frac{R_0(1+e^{\alpha})}{K\log K}\right)^2 - 1}}$$

and

$$\tau^* = \frac{\arctan(-\gamma/\omega^*)}{\omega^*}.$$

The effective contact reduces to half of β_0 when the incidence $x = \beta I$ is given by

$$\frac{1}{2} = \frac{1 + e^{-\alpha}}{1 + e^{kx-\alpha}}.$$

So, when the incidence reaches $k^{-1}\ln(2 + e^{\alpha})$, the effective contact reduces to β_0.

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