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Endemic threshold analysis for the Kermack–McKendrick reinfection model

Hisashi INABA

Abstract. In a seminal series of papers published during the 1930s, Kermack and McKendrick developed an infection–age structured endemic model, which takes into account the demography of the host population, and the waning immunity (variable susceptibility) and reinfection of recovered individuals. The host population is structured by a duration variable for each status, as the susceptibility of the recovered individuals depends on the duration since the last recovery. The idea of reinfection has become increasingly important in understanding emerging and reemerging infectious diseases, since it makes the control of infectious diseases difficult, and waning immunity is widely observed if there is no (natural or artificial) boosting. For the reinfection model, we can introduce the reinfection threshold of R_0 at which a qualitative change in the epidemiological implication occurs for the prevalence and controllability. If any enhancement of epidemiological reproductivity by reinfection exists, we also expect that endemic steady states backwardly bifurcate when the basic reproduction number crosses unity, which implies that attaining a subcritical level of R_0 is not necessarily a complete policy for disease prevention. The main aim of this survey is to demonstrate the possible usefulness of the Kermack–McKendrick reinfection model and its extensions to understand reinfection phenomena in the spread of infectious diseases.

1. Introduction

In a seminal series of papers published during the 1930s, Kermack and McKendrick proposed an infection–age structured *endemic* model that takes into account the demography of the host population, the waning immunity (variable susceptibility) and *reinfection* of recovered individuals ([**14**], [**15**]). Their model has less attention than the well–known outbreak model proposed in 1927 ([**13**]). In their model, the total population is decomposed into three compartments, the never infected (full susceptible), infectious and recovered populations. The host population is structured by a duration variable for each status, while the chronological age is neglected. The susceptibility of recovered individuals depends on the time that has passed since the last recovery, and the model thus has much flexibility to capture many facets of reinfection phenomena.

The concept of reinfection is becoming increasingly important in understanding emerging and reemerging infectious diseases, since it makes the control of infectious diseases difficult, and a waning immunity is widely observed if there is no (natural

or artificial) boosting. In fact, the recovered individuals or vaccinated individuals could be reinfected as time passes owing to the natural decay of host immunity, or a genetic change in the virus. Reinfection often leads to non–clinical infection. It is thus likely that its occurrence is overlooked, and that we will fail in calculating the basic reproduction number and the critical coverage of immunization by neglecting the effect of reinfection.

As was pointed out by Gomes, *et al.* ([**7**]), we can introduce the *reinfection threshold* of R_0 at which a qualitative change in the epidemiological implication occurs for the prevalence and controllability in the reinfection model. Moreover, owing to enhancement of susceptibility or infectivity by reinfection, we expect that there is a backward bifurcation of endemic steady states. In such a case, we have bistable endemic steady states, and attaining a subcritical level of R_0 is not a complete policy for disease prevention.

In this survey, we first reformulate the forgotten Kermack–McKendrick reinfection model as an age–structured population model and examine basic endemic threshold phenomena, where we present a condition that backward bifurcation occurs. We then extend the basic model into a chronological-age dependent model, and calculate basic epidemiological indices. Consideration of the chronological age structure is crucial to real world applications, because prevention policy or vaccination usually targets age classes. Finally, we again extend the basic model to recognize subclinical infection observed in malaria and measles epidemics, and examine conditions under which subcritical endemic steady states exist, because failure to do so is likely to produce incorrect estimates and interpretations of epidemiological indices.

2. Kermack–McKendrick reinfection model

2.1. Basic model: partial differential equations

We first formulate the Kermack–McKendrick reinfection model as an age–structured population model. Let $s(t,\tau)$ be the density of the susceptible population who have never been infected (*virgin* population in the terminology of Kermack and McKendrick) at time t and duration τ (the time elapsed since entry into the s–state) , which can be interpreted as the chronological age when a person enters the s–state at birth. Let $i(t,\tau)$ be the density of the infected and infectious population at time t and infection-age (the time elapsed since infection) τ and let $r(t,\tau)$ be the density of the recovered population at time t and duration τ (the time elapsed since the last recovery). Let m and μ respectively denote the birth (or immigration) rate and the death rate, and $\gamma(\tau)$ denotes the recovery rate at infection–age τ.

We assume that the force of infection applied to the fully susceptible population

(virgin population) is given by

$$\lambda(t) = \int_0^\infty \beta(\sigma) i(t,\sigma) d\sigma, \tag{1}$$

where $\beta(\tau)$ denotes the infectivity for the virgin population at infection–age τ. The force of (re)infection applied to the recovered population at duration τ is assumed to be given by $\theta(\tau)\lambda(t)$, where $\theta(\tau)$ is the relative susceptibility schedule of recovered individuals at time since recovery τ. The relative susceptibility would be inversely correlated with the wanning of immunity.

Assumption 2.1. *It is assumed that $\beta, \gamma, \theta \in L^\infty_+(\mathbb{R}_+)$, and that the state space of the age distribution functions s, i and r is $L^1_+(\mathbb{R}_+)$.*

The Kermack–McKendrick reinfection model is then formulated as

$$\begin{aligned}
&\frac{\partial s(t,\tau)}{\partial t} + \frac{\partial s(t,\tau)}{\partial \tau} = -\mu s(t,\tau) - \lambda(t) s(t,\tau),\\
&\frac{\partial i(t,\tau)}{\partial t} + \frac{\partial i(t,\tau)}{\partial \tau} = -(\mu + \gamma(\tau)) i(t,\tau),\\
&\frac{\partial r(t,\tau)}{\partial t} + \frac{\partial r(t,\tau)}{\partial \tau} = -\mu r(t,\tau) - \theta(\tau)\lambda(t) r(t,\tau),\\
&s(t,0) = m \int_0^\infty (s(t,\tau) + i(t,\tau) + r(t,\tau)) d\tau,\\
&i(t,0) = \lambda(t) \int_0^\infty (s(t,\tau) + \theta(\tau) r(t,\tau))\, d\tau,\\
&r(t,0) = \int_0^\infty \gamma(\tau) i(t,\tau) d\tau,
\end{aligned} \tag{2}$$

with initial data

$$s(0,\tau) = s_0(\tau), \quad i(0,\tau) = i_0(\tau), \quad r(0,\tau) = r_0(\tau). \tag{3}$$

Let $N(t)$ be the total size of the host population given by

$$N(t) := \int_0^\infty (s(t,\tau) + i(t,\tau) + r(t,\tau)) d\tau. \tag{4}$$

It is then easily seen that the total size of the host population is constant if $m = \mu$. In the following we consider the case of a constant total population size, denoted by N, and the boundary condition of $s(t,a)$ is thus replaced by $s(t,0) = \mu N$.

The basic system (2) has a trivial, disease–free (completely susceptible) steady state $(s^*, i^*, r^*) = (\mu N e^{-\mu\tau}, 0, 0)$. The linearized equation for the infected population in the disease-free steady state is then given by

$$\begin{aligned} &\frac{\partial \zeta(t,\tau)}{\partial t} + \frac{\partial \zeta(t,\tau)}{\partial \tau} = -(\mu + \gamma(\tau))\zeta(t,\tau), \\ &\zeta(t,0) = N\int_0^\infty \beta(\tau)\zeta(t,\tau)d\tau, \end{aligned} \tag{5}$$

and it is easily seen that the basic reproduction number for the basic model (2) is given by

$$R_0 = N\int_0^\infty e^{-\mu\tau}\beta(\tau)\Gamma(\tau)d\tau, \tag{6}$$

where $\Gamma(\tau) := \exp(-\int_0^\tau \gamma(x)dx)$ is the survival probability. By the principle of linearized stability, the stability of zero solution of (5) determines the local stability of the disease–free steady state of system (2), and the disease–free steady state is thus locally asymptotically stable if $R_0 < 1$, while it is unstable if $R_0 > 1$. The reader may refer to [**5**], [**6**] and [**11**] for the role of the basic reproduction number in population dynamics.

Model (2) can be rewritten as the Gurtin–MacCamy model for an age–dependent population. Its mathematical well–posedness has been established ([**9**]), and we can use the integrated semigroup formulation to give the solution semiflow ([**17**]). We therefore skip the mathematical well–posedness problem here. However, we remark that an alternative integral equation formulation is possible and informative. This formulation is discussed in the next subsection.

2.2. Integral equation

For simplicity, instead of considering the initial value problem, we assume that the epidemic starts at $t = -\infty$. Integrating the partial differential equations in (2) along the characteristic line, we have a set of equations:

$$\begin{aligned} s(t,\tau) &= \mu N e^{-\mu\tau - \int_0^\tau \lambda(t-\tau+\sigma)d\sigma}, \\ i(t,\tau) &= b_1(t-\tau)e^{-\mu\tau}\Gamma(\tau), \\ r(t,\tau) &= b_2(t-\tau)e^{-\mu\tau - \int_0^\tau \lambda(t-\tau+\sigma)\theta(\sigma)d\sigma}, \end{aligned} \tag{7}$$

where $b_1(t) := i(t,0)$ and $b_2(t) := r(t,0)$. Inserting equations (7) into the boundary conditions of (2), we obtain a set of integral equations:

$$\begin{aligned} b_1(t) &= \lambda(t)\left[\int_0^\infty \mu N e^{-\mu\tau - \int_0^\tau \lambda(t-\tau+\sigma)d\sigma}d\tau \right. \\ &\qquad \left. + \int_0^\infty \theta(\tau)b_2(t-\tau)e^{-\mu\tau - \int_0^\tau \lambda(t-\tau+\sigma)\theta(\sigma)d\sigma}d\tau\right], \\ b_2(t) &= \int_0^\infty b_1(t-\tau)e^{-\mu\tau}\gamma(\tau)\Gamma(\tau)d\tau, \end{aligned} \tag{8}$$

where

$$\lambda(t) = \int_0^\infty e^{-\mu\tau}\beta(\tau)\Gamma(\tau)b_1(t-\tau)d\tau. \tag{9}$$

Inserting the expression for b_2 into the equation for b_1 in (8) and changing the order of integrals, we obtain

$$b_1(t) = \lambda(t)\int_0^\infty S(t,\tau)d\tau, \tag{10}$$

$$\begin{aligned} S(t,\tau) :&= s(t,\tau) + \theta(\tau)r(t,\tau) \\ &= \mu N e^{-\mu\tau - \int_0^\tau \lambda(t-\tau+\sigma)d\sigma} \\ &\quad + b_1(t-\tau)e^{-\mu\tau}\int_0^\tau \theta(\sigma)e^{-\int_0^\sigma \theta(\zeta)\lambda(t-\sigma+\zeta)d\zeta}\gamma(\tau-\sigma)\Gamma(\tau-\sigma)d\sigma, \end{aligned} \tag{11}$$

where $\int_0^\infty S(t,\tau)d\tau$ is the *effective size of susceptibles*. The expression (10) implies a simple fact that the new incidence at time t is given by the force of infection times the size of effective susceptibles ([**2**]).

From (10) and (11), we obtain a linear renewal equation for b_1 if we see the force of infection λ as a given function, and thus, by solving the linear renewal equation formally, we have an expression of b_1 with unknown λ. Inserting this solution into (9), we arrive at a nonlinear "scalar" renewal equation for λ. Alternatively, eliminating λ from (9), (10) and (11), we again obtain a nonlinear scalar integral equation for b_1. We can then establish the well-posedness of the Kermack–McKendrick reinfection model (2) based on the well-known method of the nonlinear integral equation.

If $\theta \equiv 0$, (2) becomes the suceptible–infected–recovered (SIR) model with permanent immunity, and it has a unique endemic steady state if and only if $R_0 > 1$ and it is globally stable ([**17**]). If $\theta \equiv 1$, the recovered population can be identified with the virgin population, and (2) is thus reduced to the infection–age dependent SIS epidemic model, and it is formulated by a nonlinear renewal equation, its endemic steady state is unique but can lose stability and Hopf bifurcations can occur when $R_0 > 1$ ([**3**], [**4**], [**18**]). Under the assumption that θ is monotone increasing and less than unity, it is concluded that if $R_0 > 1$, there exists a unique endemic steady state that is locally asymptotically stable as long as $|R_0 - 1|$ is small enough ([**9**]).

If $\sup\theta > 1$, we conjecture that the subcritical condition $R_0 < 1$ does not necessarily guarantee the eradication of diseases. In fact, from (10), we formally

define a time-dependent (period) reproduction number as

$$\mathcal{R}(t) := \tilde{S}(t) \int_0^\infty \beta(\tau)\Gamma(\tau)e^{-\mu\tau}d\tau, \tag{12}$$

where $\tilde{S}(t) := \int_0^\infty S(t,\tau)d\tau$ is the effective size of susceptibility. Since $\tilde{S}(t)$ can be larger than the total population size N, $\mathcal{R}(t)$ can be larger than R_0, and $R_0 < 1$ would thus not be a sufficient condition for eradication of the disease.

Let $\alpha := \max\{1, \sup_{\tau\geq 0}\theta(\tau)\}$. Then $\tilde{S} \leq \alpha N$ and it follows from (10) that

$$b_1(t) \leq \alpha N \int_0^\infty \beta(\tau)\Gamma(\tau)e^{-\mu\tau}b_1(t-\tau)d\tau. \tag{13}$$

Using the comparison argument, we know that $\lim_{t\to\infty} b_1(t) = 0$ if $\alpha R_0 < 1$. We then have a simple criterion for the global stability of the disease–free steady state.

PROPOSITION 2.2. *If $R_0 < 1/\alpha$, the disease–free steady state of* (2) *is globally asymptotically stable.*

Here we remark that another extreme scenario of recovering susceptibility is that recovered individuals are completely immune, and at recovery–age τ they return to the susceptible class again with the force of reversion $\delta(\tau)$. In this case, instead of (2), we obtain

$$\begin{aligned}
&\frac{\partial r(t,\tau)}{\partial t} + \frac{\partial r(t,\tau)}{\partial \tau} = -\mu r(t,\tau) - \delta(\tau)r(t,\tau),\\
&s(t,0) = \mu N + \int_0^\infty \delta(\tau)r(t,\tau)d\tau,\\
&i(t,0) = \lambda(t)\int_0^\infty s(t,\tau)d\tau,\\
&r(t,0) = \int_0^\infty \gamma(\tau)i(t,\tau)d\tau,
\end{aligned} \tag{14}$$

where we omit the McKendrick equations for s and i, because they are the same as the equations in (2). In other words, we obtain a SIRS model with infection–age, which was studied by Nakata, *et al.* ([**19**]) in the case that δ is constant. In this model, the density of the susceptible population is given by

$$s(t,\tau) := e^{-\mu\tau - \int_0^\tau \lambda(t-\tau+\sigma)d\sigma}\left[\mu N + \int_0^\infty b_1(t-\tau)e^{-\mu\tau}P(\tau)d\tau\right], \tag{15}$$

where

$$P(\tau) = \int_0^\tau \delta(x)e^{-\int_0^x \delta(z)dz}\gamma(\tau-x)\Gamma(\tau-x)dx, \tag{16}$$

denotes the probability density that an infected individual returns to the susceptible status at time τ that has elapsed since the last infection.

2.3. Bifurcation of endemic steady states

We now check the bifurcation of endemic steady states. Let $s^*(\tau)$, $i^*(\tau)$ and $r^*(\tau)$ be the steady state solution. It then holds that

$$\begin{aligned} s^*(\tau) &= \mu N e^{-(\mu+\lambda^*)\tau}, \\ i^*(\tau) &= i^*(0)e^{-\mu\tau}\Gamma(\tau), \\ r^*(\tau) &= r^*(0)e^{-\mu\tau-\lambda^*\int_0^\tau \theta(\sigma)d\sigma}, \end{aligned} \tag{17}$$

where

$$\begin{aligned} i^*(0) &= \lambda^* \int_0^\infty (s^*(\tau) + \theta(\tau)r^*(\tau))d\tau, \\ r^*(0) &= \int_0^\infty \gamma(\tau)i^*(\tau)d\tau. \end{aligned} \tag{18}$$

and λ^* is the force of infection in the steady state given by

$$\lambda^* = \int_0^\infty \beta(\tau)i^*(\tau)d\tau = b^*\langle\beta,\Gamma\rangle. \tag{19}$$

In expression (19), $b^* := i^*(0)$ is the density of the newly infecteds in the steady state and we have used the notation as

$$\langle\beta,\Gamma\rangle := \int_0^\infty \beta(\tau)\Gamma(\tau)e^{-\mu\tau}d\tau. \tag{20}$$

Inserting (19) into the first equation of (18), we obtain

$$b^* = b^*\langle\beta,\Gamma\rangle \int_0^\infty (\mu N e^{-(\mu+\lambda^*)\tau} + r^*(0)\theta(\tau)e^{-\mu\tau-\lambda^*\int_0^\tau \theta(\sigma)d\sigma})d\tau, \tag{21}$$

which shows a renewal relation in a steady state with the force of infection λ^*. Since $\langle\beta,\Gamma\rangle = R_0/N$ and $r^*(0) = b^*\langle\gamma,\Gamma\rangle$, we arrive at an equation for unknown λ^*:

$$\begin{aligned} R(\lambda^*) :&= \frac{\mu R_0}{\mu+\lambda^*} + \langle\gamma,\Gamma\rangle\lambda^* \int_0^\infty \theta(\tau)e^{-\mu\tau-\lambda^*\int_0^\tau \theta(\sigma)d\sigma}d\tau \\ &= \frac{\mu R_0}{\mu+\lambda^*} + \langle\gamma,\Gamma\rangle\left(1 - \int_0^\infty \mu e^{-\mu\tau-\lambda^*\int_0^\tau \theta(\sigma)d\sigma}d\tau\right) = 1, \end{aligned} \tag{22}$$

where we used the notation as

$$\langle\gamma,\Gamma\rangle := \int_0^\infty \gamma(\tau)\Gamma(\tau)e^{-\mu\tau}d\tau. \tag{23}$$

Equation (22) implies that the effective reproduction number, given by $R(\lambda^*)$, must be unity in a steady state.

It follows from (22) that there exists at least one endemic steady state if $R_0 > 1$, because $R(0) = R_0 > 1$ and $\lim_{\lambda\to\infty} R(\lambda) = \langle\gamma, \Gamma\rangle < 1$. Given that $R(\lambda^*)$ is not monotone decreasing, there is a possibility that multiple endemic steady states exist.

PROPOSITION 2.3. *If the inequality*

$$\langle\gamma, \Gamma\rangle\theta^* > 1, \tag{24}$$

holds, where

$$\theta^* := \int_0^\infty \theta(\tau)\mu e^{-\mu\tau} d\tau, \tag{25}$$

then endemic steady states backwardly bifurcate from the disease–free steady state when R_0 crosses unity, i.e., multiple endemic steady states exist if $R_0 < 1$ and $|R_0 - 1|$ is small enough.

PROOF. Define a function $f(\lambda, R_0) := R(\lambda) - 1$, where R_0 is seen as a bifurcation parameter and $f(0, 1) = 0$. Observe that

$$\left.\frac{\partial f}{\partial\lambda}\right|_{(\lambda,R_0)=(0,1)} = \frac{1}{\mu}(\theta^*\langle\gamma, \Gamma\rangle - 1), \quad \left.\frac{\partial f}{\partial R_0}\right|_{(\lambda,R_0)=(0,1)} = 1.$$

Therefore if condition (24) holds, then $f = 0$ is solved as $\lambda = \lambda(R_0)$ with $\lambda(1) = 0$ at the neighborhood of $(\lambda, R_0) = (0, 1)$. Since $d\lambda(1)/dR_0 < 0$, we have $\lambda(R_0) > 0$ for $R_0 \in (1 - \eta, 1)$ for sufficiently small $\eta > 0$. For each $R_0 \in (1 - \eta, 1)$, we have $f(0, R_0) < 1$, $f(\lambda(R_0), R_0) = 0$ and $\lim_{\lambda\to\infty} f(\lambda, R_0) = \langle\gamma, \Gamma\rangle - 1 < 0$, and there are then at least two endemic steady states. □

Condition (24) was first given in [**21**] by using the ordinary differential equation version of (2). It is easily seen that condition (24) does not hold if there is no enhancement of susceptibility, i,e., if $\theta(\tau) \le 1$ for all $\tau \ge 0$.

3. Vaccination model and reinfection threshold

3.1. Reinfection threshold

We now introduce a mass vaccination (host immunization) in the basic model (2). In fact, it is intuitively clear that reinfection phenomena would make disease control more difficult and complex, and we thus need an index to capture the difficulty. An important effect of vaccination policy is the reduction of the effective size of the susceptible population. In the reinfection model, there is a possibility

that a disease can invade a fully vaccinated population, and we are naturally led to the idea of the *reinfection threshold*.

Suppose that newborns or immigrants in the virgin population are mass vaccinated with coverage $\epsilon \in [0,1]$ and, for simplicity, the immunological status of newly vaccinated individuals is identical to that of the newly recovered individuals. This assumption will be relaxed in section 5. The boundary condition in the basic system (2) is then replaced:

$$\begin{aligned} s(t,0) &= (1-\epsilon)\mu N, \\ i(t,0) &= \lambda(t)\int_0^\infty (s(t,\tau)+\theta(\tau)r(t,\tau))\,d\tau, \\ r(t,0) &= \epsilon\mu N + \int_0^\infty \gamma(\tau)i(t,\tau)d\tau. \end{aligned} \tag{26}$$

The disease-free steady state is then given by

$$(s^*, i^*, r^*) = ((1-\epsilon)\mu N e^{-\mu\tau}, 0, \epsilon\mu N e^{-\mu\tau}),$$

and the linearized renewal equation in the initial invasion phase is thus given by

$$\xi(t) = ((1-\epsilon)N + \epsilon N\theta^*)\int_0^\infty e^{-\mu\tau}\beta(\tau)\Gamma(\tau)\xi(t-\tau)d\tau, \tag{27}$$

where $\xi(t) := \zeta(t,0)$ denotes a small perturbation in the infected population density.

Therefore, the effective reproduction number, denoted by $\mathcal{R}(\epsilon)$, in the partially immunized disease–free steady state is given by

$$\mathcal{R}(\epsilon) = (1-\epsilon)R_0 + \epsilon R_1 = (1-\epsilon(1-\theta^*))R_0, \tag{28}$$

where $R_1 := \theta^* R_0$. Then if $\mathcal{R}(\epsilon) < 1$, the disease–free steady state is locally asymptotically stable, while it is unstable if $\mathcal{R}(\epsilon) > 1$. However, it is unclear whether the disease–free steady state becomes globally asymptotically stable when $\mathcal{R}(\epsilon) < 1$.

Here we note that R_1 is the effective reproduction number for the fully vaccinated system. In fact, if $\epsilon = 1$, the virgin population is eradicated, and we obtain the limiting recovered–infected–recovered system as

$$\begin{aligned}
&\frac{\partial i(t,\tau)}{\partial t}+\frac{\partial i(t,\tau)}{\partial \tau}=-(\mu+\gamma(\tau))i(t,\tau),\\
&\frac{\partial r(t,\tau)}{\partial t}+\frac{\partial r(t,\tau)}{\partial \tau}=-\mu r(t,\tau)-\theta(\tau)\lambda(t)r(t,\tau),\\
&i(t,0)=\lambda(t)\int_0^\infty \theta(\tau)r(t,\tau)d\tau,\\
&r(t,0)=\mu N+\int_0^\infty \gamma(\tau)i(t,\tau)d\tau.
\end{aligned} \tag{29}$$

This new system (29) can be seen as a duration–dependent SIS model with vaccination if we view the recovered class as a new susceptible class. Then (29) has a disease–free steady state $(i^*, r^*) = (0, \mu N e^{-\mu\tau})$, and the linearized system in the disease free steady state is given as

$$\begin{aligned}
&\frac{\partial \zeta(t,\tau)}{\partial t}+\frac{\partial \zeta(t,\tau)}{\partial \tau}=-(\mu+\gamma(\tau))\zeta(t,\tau),\\
&\zeta(t,0)=\theta^* N\int_0^\infty \beta(\tau)\zeta(t,\tau)d\tau.
\end{aligned} \tag{30}$$

Therefore the effective reproduction number for the limiting system (29) is given by $R_1 = \theta^* R_0$.

Suppose that $R_0 > 1$. From (28), the critical coverage of immunization ϵ^* such that $\mathcal{R}(\epsilon^*) = 1$ is given by

$$\epsilon^* = \left(1-\frac{1}{R_0}\right)\frac{1}{1-\theta^*}, \tag{31}$$

but it is meaningful only when $\theta^* < 1$. The disease is uncontrollable by the vaccination if $\theta^* \geq 1$. Moreover, if $R_1 = \theta^* R_0 > 1$, we have $\mathcal{R}(\epsilon) > 1$ for all $\epsilon \in [0, 1]$, and the disease is thus again uncontrollable by the vaccination, because the fully vaccinated population can be invaded by the disease.

Let $\sigma := R_1/R_0$, i.e., σ is the ratio of the effective reproduction number of the fully vaccinated system to the basic reproduction number. Given that the qualitative change in the epidemiological implication occurs for the prevalence and controllability at $R_0 = 1/\sigma$, Gomes *et al.* ([**7**], [**8**]) referred to $1/\sigma$ as the *reinfection threshold* of R_0. As seen above, the reinfection threshold of R_0 corresponds to the fact that $\sigma R_0 = R_1 = 1$, i.e., $R_0 = 1/\sigma$ does not imply a bifurcation point of the basic system (2), but the threshold condition $R_1 = 1$ of the fully vaccinated system (29). In the above setting, we have $\sigma = \theta^*$, but its value depends on the basic model assumptions.

3.2. Bifurcation of endemic steady states

Let (s^*, i^*, r^*) be the steady state of the basic system (2) with the boundary condition (26). We then have

$$\begin{aligned} s^*(\tau) &= (1-\epsilon)\mu N e^{-\mu\tau-\lambda^*\tau}, \\ i^*(\tau) &= i^*(0)e^{-\mu\tau}\Gamma(\tau), \\ r^*(\tau) &= r^*(0)e^{-\mu\tau-\lambda^*\int_0^\tau \theta(x)dx}, \end{aligned} \tag{32}$$

where

$$\lambda^* = i^*(0)\langle\beta, \Gamma\rangle,$$

$$\begin{aligned} i^*(0) &= \lambda^* \int_0^\infty (s^*(\tau) + \theta(\tau)r^*(\tau))d\tau, \\ r^*(0) &= \epsilon\mu N + i^*(0)\langle\gamma, \Gamma\rangle. \end{aligned} \tag{33}$$

From the above equations, we can calculate $i^*(0)$ as

$$\begin{aligned} i^*(0) &= \lambda^* \int_0^\infty (s^*(\tau) + \theta(\tau)r^*(\tau))d\tau \\ &= \lambda^* \frac{(1-\epsilon)\mu N}{\mu+\lambda^*} + \lambda^* r^*(0) \int_0^\infty \theta(\tau)e^{-\mu\tau-\lambda^*\int_0^\tau \theta(x)dx}d\tau \\ &= \lambda^* \frac{(1-\epsilon)\mu N}{\mu+\lambda^*} + \lambda^* \left(\epsilon\mu N + i^*(0)\langle\gamma, \Gamma\rangle\right) \int_0^\infty \theta(\tau)e^{-\mu\tau-\lambda^*\int_0^\tau \theta(x)dx}d\tau. \end{aligned} \tag{34}$$

We then have the expression:

$$i^*(0) = \frac{\lambda^* \frac{(1-\epsilon)\mu N}{\mu+\lambda^*} + \epsilon\mu N\lambda^* \int_0^\infty \theta(\tau)e^{-\mu\tau-\lambda^*\int_0^\tau \theta(x)dx}d\tau}{1 - \lambda^*\langle\gamma, \Gamma\rangle \int_0^\infty \theta(\tau)e^{-\mu\tau-\lambda^*\int_0^\tau \theta(x)dx}d\tau}. \tag{35}$$

From (35) and the relation

$$\lambda^* = \frac{R_0}{N} i^*(0),$$

we know that a positive root $\lambda^* > 0$ must satisfy the equation:

$$1 = R_0 \frac{v(\lambda^*)}{u(\lambda^*)}, \tag{36}$$

where

$$
\begin{aligned}
v(\lambda) &:= \frac{(1-\epsilon)\mu}{\mu+\lambda} + \epsilon\mu \int_0^\infty \theta(\tau) e^{-\mu\tau - \lambda\int_0^\tau \theta(x)dx} d\tau, \\
u(\lambda) &:= 1 - \langle \gamma, \Gamma \rangle \phi(\lambda).
\end{aligned} \tag{37}
$$

Here we have used the notation (23) and

$$
\phi(\lambda) := \lambda \int_0^\infty \theta(\tau) e^{-\mu\tau - \lambda\int_0^\tau \theta(x)dx} d\tau. \tag{38}
$$

Observe that

$$
\lambda \int_0^\infty \theta(\tau) e^{-\mu\tau - \lambda\int_0^\tau \theta(x)dx} d\tau = 1 - \int_0^\infty \mu e^{-\mu\tau - \lambda\int_0^\tau \theta(x)dx} d\tau. \tag{39}
$$

ϕ is then an increasing function, and $u(\lambda)$ is thus a decreasing function. We can now conclude the following.

PROPOSITION 3.1. *If $\mathcal{R}(\epsilon) > 1$, there exists at least one endemic steady state. Suppose that the condition*

$$
\theta^* \langle \gamma, \Gamma \rangle > \frac{1 - \epsilon(1-\theta^{**})}{1 - \epsilon(1-\theta^*)}, \tag{40}
$$

holds, where

$$
\theta^{**} := \mu^2 \int_0^\infty e^{-\mu\tau} \theta(\tau) \int_0^\tau \theta(x) dx d\tau. \tag{41}
$$

Endemic steady states then backwardly bifurcate from the disease–free steady state when $\mathcal{R}(\epsilon)$ crosses unity, i.e., multiple endemic steady states exist if $\mathcal{R}(\epsilon) < 1$ and $|\mathcal{R}(\epsilon) - 1|$ is small enough.

PROOF. Relation (36) implies that the effective reproduction number in the endemic steady state with the force of infection λ^* is given by

$$
R(\lambda^*) = R_0 \frac{v(\lambda^*)}{u(\lambda^*)} = \frac{\mathcal{R}(\epsilon)}{v(0)} \frac{v(\lambda^*)}{u(\lambda^*)}.
$$

Then $R(0) = \mathcal{R}(\epsilon)$ and $R(\infty) = 0$, and thus $R(\lambda^*) = 1$ has at least one positive root if $\mathcal{R}(\epsilon) > 1$, which implies that there exists one endemic steady state. If $R(0) = \mathcal{R}(\epsilon) = R_0 v(0) = 1$ and condition (40) holds, $R'(0) = v(0)^{-1} v'(0) - u'(0) > 0$. $R(\lambda^*) = 1$ then has at least one positive root. Moreover, it has at least two positive roots if $R(0) = \mathcal{R}(\epsilon) < 1$ and $|\mathcal{R}(\epsilon) - 1|$ is small enough. To see this precisely, let

us again define a function $f(\lambda, R_0) := R(\lambda) - 1$. Then $f(0, v(0)^{-1}) = 0$ and

$$\left.\frac{\partial f}{\partial R_0}\right|_{(\lambda,R_0)=(0,v(0)^{-1})} = 1, \quad \left.\frac{\partial f}{\partial \lambda}\right|_{(\lambda,R_0)=(0,v(0)^{-1})} = v(0)^{-1}v'(0) - u'(0),$$

where

$$v'(0) = -\frac{1}{\mu}(1 - \epsilon(1 - \theta^{**})), \quad u'(0) = -\frac{1}{\mu}\langle\gamma, \Gamma\rangle\theta^*.$$

If condition (40) holds, $f = 0$ is solved as $\lambda = \lambda(R_0)$ satisfying $\lambda(v(0)^{-1}) = 0$ and $d\lambda(v(0)^{-1})/dR_0 < 0$ in the neighborhood of $(\lambda, R_0) = (0, v(0)^{-1})$. If $R_0 v(0) < 1$ and $|R_0 v(0) - 1|$ is small enough, for each R_0, there exist multiple positive roots such that $f(\lambda, R_0) = 0$, because $f(0, R_0) < 1$, $f(\lambda(R_0), R_0) = 0$ and $f(\infty, R_0) = -1 < 0$. □

Proposition 3.1 tells us that the subcritical condition $\mathcal{R}(\epsilon) < 1$ is not sufficient to eradicate the disease if condition (40) holds. Note that if $\epsilon = 1$ in (40), we know that a backward bifurcation occurs even in the recovered–infected–recovered model if $(\theta^*)^2 > \theta^{**}$, though this condition does not hold when θ is constant.

4. Chronological-age dependent reinfection model

4.1. Basic model

We now extend the Kermack–McKendrick reinfection model to take into account the chronological age structure of host individuals. Let $S(t, a)$ be the density of the susceptible population who have never been infected at time t and chronological age a. Let $i(t, \tau, a)$ be the density of the infected and infectious population at time t, age a and infection–age τ and let $r(t, \tau, a)$ be the density of the recovered population at time t, age a and recovery–age τ (the time elapsed since the last recovery). Let $m(a)$ and $\mu(a)$ denote the age-dependent fertility rate and the force of mortality at age a and $\gamma(\tau)$ denotes the recovery rate at infection–age τ.

We assume that the force of infection applied to the full susceptible population (virgin population) is given by

$$\lambda(t) = \frac{1}{N(t)} \int_0^\infty \int_0^a \beta(\tau) i(t, \tau, a) d\tau da, \tag{42}$$

where $N(t)$ is the total host population size. The Kermack–McKendrick reinfection model can then be extended to a demographic age–structured model:

$$
\begin{aligned}
&\frac{\partial S(t,a)}{\partial t}+\frac{\partial S(t,a)}{\partial a}=-\mu(a)S(t,a)-\lambda(t)S(t,a),\\
&\frac{\partial i(t,\tau,a)}{\partial t}+\frac{\partial i(t,\tau,a)}{\partial \tau}+\frac{\partial i(t,\tau,a)}{\partial a}=-(\mu(a)+\gamma(\tau))i(t,\tau,a),\\
&\frac{\partial r(t,\tau,a)}{\partial t}+\frac{\partial r(t,\tau,a)}{\partial \tau}+\frac{\partial r(t,\tau,a)}{\partial a}=-\mu(a)r(t,\tau,a)-\theta(\tau)\lambda(t)r(t,\tau,a),\\
&S(t,0)=\int_0^\infty m(a)(S(t,a)+I(t,a)+R(t,a))da,\\
&i(t,0,a)=\lambda(t)\left[S(t,a)+\int_0^a \theta(\tau)r(t,\tau,a)d\tau\right],\\
&r(t,0,a)=\int_0^a \gamma(\tau)i(t,\tau,a)d\tau,
\end{aligned}
\tag{43}
$$

with initial data

$$
S(0,a)=S_0(a),\quad i(0,\tau,a)=i_0(\tau,a),\quad r(0,\tau,a)=r_0(\tau,a).
$$

Here I and R respectively denote the age-density functions of infected and recovered populations aggregated with respect to the duration variable:

$$
I(t,a):=\int_0^a i(t,\tau,a)d\tau,\quad R(t,a):=\int_0^a r(t,\tau,a)d\tau.
$$

Let $P(t,a)$ be the age–density function of the host population given by $P(t,a):=S(t,a)+I(t,a)+R(t,a)$, so $N(t)=\int_0^\infty P(t,a)da$. In the following we assume that the host population is in a demographic steady state, and thus the condition

$$
\int_0^\infty m(a)\ell(a)da=1, \tag{44}
$$

holds, where

$$
\ell(a)=\exp\left(-\int_0^a \mu(\sigma)d\sigma\right),
$$

is the demographic survival probability. We thus assume that there exists a constant $B>0$ such that $P(t,a)=P^*(a)=B\ell(a)$ for all $t\geq 0$ and $N(t)=N^*=\int_0^\infty P^*(a)da=Be_0$, where $e_0=\int_0^\infty \ell(a)da$ is the average life expectancy of host individuals.

Although we do not discuss the well–posedness of problem (43), it is again remarked that the basic system (43) can be reduced to a system of integral equations.

In fact, partial differential equations in (43) are integrated along characteristic lines:

$$\begin{aligned} S(t,a) &= B_1(t-a)\ell(a)e^{-\int_0^a \lambda(t-a+\sigma)d\sigma}, \\ i(t,\tau,a) &= B_2(t-\tau,a-\tau)\frac{\ell(a)}{\ell(a-\tau)}\Gamma(\tau), \\ r(t,\tau,a) &= B_3(t-\tau,a-\tau)\frac{\ell(a)}{\ell(a-\tau)}e^{-\int_0^\tau \lambda(t-\tau+\sigma)\theta(\sigma)d\sigma}, \end{aligned} \tag{45}$$

where $B_1(t) := S(t,0)$, $B_2(t,a) := i(t,0,a)$ and $B_3(t,a) := r(t,0,a)$. Note that the force of infection and the density of newly recovered population are determined by the age–specific incidence rate B_2 as

$$\begin{aligned} \lambda(t) &= \frac{1}{N(t)}\int_0^\infty \int_0^a \beta(\tau)\Gamma(\tau)\frac{\ell(a)}{\ell(a-\tau)}B_2(t-\tau,a-\tau)d\tau da, \\ B_3(t,a) &= \int_0^a \gamma(\tau)\Gamma(\tau)\frac{\ell(a)}{\ell(a-\tau)}B_2(t-\tau,a-\tau)d\tau. \end{aligned} \tag{46}$$

Therefore, using the boundary conditions for $S(t,0)$ and $i(t,0,a)$ in (43), we obtain a nonlinear system of renewal integral equations for B_1 and B_2, for which we can adopt a classical fixed point method to show the existence and uniqueness of a local solution. Meanwhile, for a semigroup approach to (43), the reader may refer to Thieme ([**22**]).

4.2. Invasion problem and R_0

For the basic model (43), there exists a disease–free steady state $(S,i,r) = (P^*,0,0)$, in which the linearized equation for the infected population is given by

$$\begin{aligned} &\frac{\partial\zeta(t,\tau,a)}{\partial t} + \frac{\partial\zeta(t,\tau,a)}{\partial\tau} + \frac{\partial\zeta(t,\tau,a)}{\partial a} = -(\mu(a)+\gamma(\tau))\zeta(t,\tau,a), \\ &\zeta(t,0,a) = w^*(a)\int_0^\infty\int_0^a \beta(\tau)\zeta(t,\tau,a)d\tau da, \end{aligned} \tag{47}$$

where ζ is the density of the infecteds in the disease–free steady state and $w^*(a) = \ell(a)/e_0$ is the age profile of the host population in the demographic steady state.

The above linearized equation of the infective population is formulated as an abstract boundary value problem:

$$\begin{aligned} &\frac{\partial\zeta(t,\tau,\cdot)}{\partial t} + \frac{\partial\zeta(t,\tau,\cdot)}{\partial\tau} = A\zeta(t,\tau,\cdot) - \gamma(\tau)\zeta(t,\tau,\cdot), \\ &\zeta(t,0,a) = w^*(a)\int_0^\infty\int_0^a \beta(\tau)\zeta(t,\tau,a)d\tau da, \end{aligned} \tag{48}$$

where A is a linear operator defined by

$$\begin{aligned}&(A\phi)(a) = -\frac{d\phi(a)}{da} - \mu(a)\phi(a),\\ &\phi \in \mathcal{D}(A) = \{\phi \in L^1(0,\infty) : A\phi \in L^1, \phi(0) = 0\}.\end{aligned} \tag{49}$$

Let $T(t) = e^{tA}$ be the strongly continuous semigroup generated by A. We then have the explicit expression as

$$(T(t)\phi)(a) = \begin{cases} 0, & t-a>0,\\ \frac{\ell(a)}{\ell(a-t)}\phi(a-t), & a-t>0. \end{cases} \tag{50}$$

Integrating (48) along the characteristic line, it follows that

$$\zeta(t,\tau,a) = \begin{cases} \Gamma(\tau)(T(\tau)\zeta(t-\tau,0,\cdot))(a), & t-\tau>0,\\ \frac{\Gamma(\tau)}{\Gamma(\tau-t)}(T(t)\zeta_0(\tau-t,\cdot))(a), & \tau-t>0, \end{cases} \tag{51}$$

where $\zeta_0(\tau,a) = \zeta(0,\tau,a)$ denotes the initial data.

Let $E := L^1((0,\infty)\times(0,\infty))$. We remark that the biologically meaningful state space for the age-duration distributions is the subset of E such that $E_0 := \{\psi \in E : \psi(\tau,a) = 0, \text{a.e. if } \tau > a\}$. It is easily seen from (50) that $i(t,\cdot,\cdot) \in E_0$ if $i_0 \in E_0$.

Inserting expression (50) into definition yields

$$\xi(t,a) = w^*(a)\int_0^\infty d\tau \int_\tau^\infty \beta(\tau)\zeta(t,\tau,a)da, \tag{52}$$

where $\xi(t,a) := \zeta(t,0,a)$. We then arrive at an abstract renewal equation in $L^1(0,\omega)$:

$$\xi(t) = G(t) + \int_0^t \Pi(\tau)\xi(t-\tau)ds, \tag{53}$$

where $\xi(t) = \xi(t,\cdot) \in L^1(0,\infty)$, $\Pi(\tau)$ is a positive linear operator on $L^1(0,\infty)$ defined by

$$(\Pi(\tau)\phi)(a) := w^*(a)\int_\tau^\infty \beta(\tau)\Gamma(\tau)(T(\tau)\phi)(\sigma)d\sigma, \quad \phi \in L^1(0,\infty), \tag{54}$$

and

$$G(t,a) := w^*(a)\int_t^\infty d\tau \int_\tau^\infty \beta(\tau)\frac{\Gamma(\tau)}{\Gamma(\tau-t)}(T(t)\zeta_0(\tau-t,\cdot))(\sigma)d\sigma. \tag{55}$$

Therefore the *next generation operator* K on $L^1(0,\infty)$ ([**11**]) is given as

$$\begin{aligned}(K\phi)(a) &= \int_0^\infty (\Pi(\tau)\phi)(a)d\tau \\ &= w^*(a)\int_0^\infty d\tau \int_\tau^\infty \beta(\tau)\Gamma(\tau)(T(\tau)\phi)(\sigma)d\sigma \\ &= w^*(a)\int_0^\infty d\tau \int_\tau^\infty \beta(\tau)\Gamma(\tau)\frac{\ell(\sigma)}{\ell(\sigma-\tau)}\phi(\sigma-\tau)d\sigma \\ &= w^*(a)\int_0^\infty \Psi(a)\phi(a)da,\end{aligned} \tag{56}$$

where

$$\Psi(a) := \int_0^\infty \beta(\tau)\Gamma(\tau)\frac{\ell(a+\tau)}{\ell(a)}d\tau, \tag{57}$$

is the total infectivity of the infecteds who are infected at age a.

In this case, the next generation operator is a one-dimensional positive map, the spectral radius of which is easily calculated as

$$r(K) = R_0 = \int_0^\infty \Psi(a)w^*(a)da. \tag{58}$$

Although we omit the proof, the following is easily obtained.

PROPOSITION 4.1. *If $R_0 < 1$, the disease–free steady state is locally asymptotically stable, while it is unstable if $R_0 > 1$. If $\theta(\tau) \le 1$ for all $\tau \ge 0$, the disease-free steady state is globally asymptotically stable if $R_0 < 1$.*

4.3. Endemic steady states

Let $S^*(a)$, $i^*(\tau,a)$ and $r^*(\tau,a)$ be the age–duration–density functions of susceptibles, infected and recovered individuals, respectively, in the endemic steady state. It is then straightforward to obtain the expressions:

$$\begin{aligned}S^*(a) &= P^*(a)e^{-\lambda^* a}, \\ i^*(\tau,a) &= \frac{\ell(a)}{\ell(a-\tau)}\Gamma(\tau)i^*(0,a-\tau), \\ r^*(\tau,a) &= \frac{\ell(a)}{\ell(a-\tau)}e^{-\lambda^*\int_0^\tau \theta(\zeta)d\zeta}r^*(0,a-\tau).\end{aligned} \tag{59}$$

Let $b_1^*(a) := i^*(0,a)$ and $b_2^*(a) := r^*(0,a)$. Inserting expression (59) into the boundary conditions, we have

$$
\begin{aligned}
b_1^*(a) &= \lambda^* \left(P^*(a) e^{-\lambda^* a} + \int_0^a \theta(\tau) \frac{\ell(a)}{\ell(a-\tau)} e^{-\lambda^* \int_0^\tau \theta(\zeta) d\zeta} b_2^*(a-\tau) d\tau \right), \\
b_2^*(a) &= \int_0^a \gamma(\tau) \Gamma(\tau) \frac{\ell(a)}{\ell(a-\tau)} b_1^*(a-\tau) d\tau.
\end{aligned} \tag{60}
$$

From (60), the age density b_1^* of the newly infecteds in the endemic steady state is given as a unique solution of the renewal equation:

$$
b_1^*(a) = g(a, \lambda^*) + \int_0^a H(a, \eta, \lambda^*) b_1^*(a-\eta) d\eta, \tag{61}
$$

where

$$
\begin{aligned}
&g(a, \lambda^*) := P^*(a) \lambda^* e^{-\lambda^* a}, \\
&H(a, \eta, \lambda^*) := \int_0^\eta \frac{\ell(a)}{\ell(a-\eta)} \lambda^* \theta(\tau) e^{-\lambda^* \int_0^\tau \theta(\zeta) d\zeta} \gamma(\eta - \tau) \Gamma(\eta - \tau) d\tau.
\end{aligned} \tag{62}
$$

We define the resolvent kernel $\mathcal{R}$ as the solution of the resolvent equation:

$$
\mathcal{R}(a, \eta, \lambda^*) = H(a, \eta, \lambda^*) + \int_0^\eta H(a, x, \lambda^*) \mathcal{R}(a - x, \eta - x, \lambda^*) dx. \tag{63}
$$

The renewal equation (61) is then solved as

$$
b_1^*(a) = g(a, \lambda^*) + \int_0^a \mathcal{R}(a, x, \lambda^*) g(a - x, \lambda^*) dx. \tag{64}
$$

From

$$
\lambda^* = \frac{1}{N^*} \int_0^\infty \Psi(a) b_1^*(a) da = \frac{1}{P^*(a)} (K b_1^*)(a), \tag{65}
$$

we can write (64) as

$$
b_1^* = (\tilde{w} + \mathcal{R} * \tilde{w}) \langle \Psi, b_1^* \rangle = (K_e b_1^*)(a), \tag{66}
$$

where

$$
\tilde{w}(a) := w^*(a) e^{-\lambda^* a}, \quad \langle \Psi, b_1^* \rangle := \int_0^\infty \Psi(a) b_1^*(a) da, \tag{67}
$$

and $*$ denotes the convolution operation defined by $(f * g)(t) := \int_0^t f(\tau) g(t - \tau) d\tau$. The integral operator K_e is the *effective next generation operator* in the endemic steady state ([**10**]) and $\tilde{w} + \mathcal{R} * \tilde{w}$ is its positive eigenvector. Therefore, the spectral

radius of K_e should be unity:

$$\langle \Psi, \tilde{w} + \mathcal{R} * \tilde{w} \rangle = 1. \tag{68}$$

This implies that

$$\begin{aligned} \mathcal{F}(\lambda^*) : = & \int_0^\infty \Psi(a) w^*(a) e^{-\lambda^* a} da \\ & + \int_0^\infty \Psi(a) \int_0^a \mathcal{R}(a, a-x, \lambda^*) w^*(x) e^{-\lambda^* x} dx da = 1. \end{aligned} \tag{69}$$

Conversely, if λ^* is a positive root of (69),

$$b_1^* = \lambda^* N^* (\tilde{w} + \mathcal{R} * \tilde{w}), \tag{70}$$

satisfies (64) and (65). Therefore, to show the existence of endemic steady states, it is sufficient to show that $\mathcal{F}(\lambda^*) = 1$ has a positive root.

PROPOSITION 4.2. *Suppose that* $\underline{\mu} := \inf_{a \geq 0} \mu(a) > 0$. *If* $R_0 > 1$, *there exists at least one endemic steady state.*

PROOF. Given that $\mathcal{F}(0) = R_0$, it is sufficient to show that $\limsup_{\lambda^* \to \infty} \mathcal{F}(\lambda^*) < 1$. In (69), by changing the order of integrals, we have

$$\begin{aligned} & \int_0^\infty \Psi(a) \int_0^a \mathcal{R}(a, a-x, \lambda^*) w^*(x) e^{-\lambda^* x} dx da \\ & \qquad = \int_0^\infty dx \int_0^\infty \mathcal{R}(x+z, z) dz w^*(x) e^{-\lambda^* x}. \end{aligned} \tag{71}$$

From resolvent equation (63), it follows that

$$\mathcal{R}(x+z, z) = H(x+z, z) + \int_0^z H(x+z, \zeta) \mathcal{R}(x+z-\zeta, z-\zeta) d\zeta. \tag{72}$$

Meanwhile, we obtain an estimate as

$$H(a, \eta, \lambda^*) \leq L(\eta) := e^{-\underline{\mu}\eta} (\phi_1 * \phi_2)(\eta), \tag{73}$$

where

$$\phi_1(\tau) := \lambda^* \theta(\tau) e^{-\lambda^* \int_0^\tau \theta(\zeta) d\zeta}, \quad \phi_2(\tau) := \gamma(\tau) \Gamma(\tau). \tag{74}$$

Let $G(z)$ be a solution of a renewal equation:

$$G(z) = L(z) + \int_0^z L(\zeta) G(z-\zeta) d\zeta. \tag{75}$$

It then follows from (72) and (73) that $\mathcal{R}(x+z,z) \le G(z)$, and from (75), it follows that

$$\int_0^\infty G(z)dz \le \frac{u_1u_2}{1-u_1u_2}, \tag{76}$$

where $u_j := \int_0^\infty e^{-\underline{\mu}\tau}\phi_j(\tau)d\tau < 1$. Therefore,

$$\int_0^\infty \mathcal{R}(x+z,z)dz \le \frac{u_1u_2}{1-u_1u_2}, \tag{77}$$

and it follows from (71) that $\lim_{\lambda^*\to\infty}\mathcal{F}(\lambda^*) = 0$. That is, $\mathcal{F}(\lambda^*) = 1$ has at least one positive root if $R_0 > 1$. This completes our proof. □

It is still an open problem to determine under what conditions the chronological–age–dependent reinfection model (43) has a unique endemic steady state, or multiple endemic steady states.

4.4. Prevalence and the total infection rate

Let $p_1(a)$ be the age–specific incidence rate and $p_2(a)$ be the age–specific recovery rate in the endemic steady state. We then have

$$p_1(a) = \frac{b_1^*(a)}{P^*(a)}, \quad p_2(a) = \frac{g(a,\lambda^*)}{P^*(a)}. \tag{78}$$

From (60), we obtain a system of equations:

$$\begin{aligned} p_1(a) &= q(a) + \int_0^a \phi_1(\tau)p_2(a-\tau)d\tau, \\ p_2(a) &= \int_0^a \phi_2(\tau)p_1(a-\tau)d\tau, \end{aligned} \tag{79}$$

where $q(a) := \lambda^* e^{-\lambda^* a}$ and ϕ_j are defined by (74). q then gives the probability density that the first infection occurs, $\phi_1(\tau)$ is the probability density that an infection occurs for recovered individuals at recovery–age τ and $\phi_2(\tau)$ is the probability density that a recovery occurs for the infecteds at infection–age τ. Then p_1 is the solution of the renewal equation

$$p_1(a) = q(a) + \int_0^a (\phi_1 * \phi_2)(\eta)p_1(a-\eta)d\eta. \tag{80}$$

Using the probability p_1, we have $i^*(0,a) = P^*(a)p_1(a)$. The *age–specific prevalence* in the endemic steady state is then calculated as

$$\frac{I^*(a)}{P^*(a)} = \frac{1}{B\ell(a)}\int_0^\infty \frac{\ell(a)}{\ell(a-\tau)}\Gamma(\tau)i^*(0,a-\tau)d\tau = \int_0^a \Gamma(a)p_1(a-\tau)d\tau. \tag{81}$$

The ratio of recovered individuals at age a is given by

$$\frac{R^*(a)}{P^*(a)} = \frac{1}{P^*(a)} \int_0^\infty r^*(\tau, a)d\tau = \int_0^a e^{-\lambda^* \int_0^\tau \theta(\zeta)d\zeta} p_2(a-\tau)d\tau. \tag{82}$$

Let P_1 be the total number of infection (*total infection rate*) and P_2 be the total number of recoveries (*total recovery rate*) in the endemic steady state. Integrating (79), we obtain the relations

$$P_1 = 1 + Q_1 P_2, \quad P_2 = Q_2 P_1, \tag{83}$$

where

$$Q_1 := \int_0^\infty \phi_1(\tau)d\tau = 1 - e^{-\lambda^* \int_0^\infty \theta(x)dx}, \tag{84}$$

is the total probability of (re)infection for recovered individuals and

$$Q_2 := \int_0^\infty \phi_2(\tau)d\tau = 1 - \Gamma(\infty), \tag{85}$$

is the total probability of recovery for the infecteds. Therefore, if $Q_1 Q_2 < 1$, we have

$$P_1 = \frac{1}{1 - Q_1 Q_2}, \quad P_2 = \frac{Q_2}{1 - Q_1 Q_2}. \tag{86}$$

4.5. Vaccination

Let $v(a)$ be the force of vaccination at age a. If we can identify the vaccinated status with the recovered status, the basic model with vaccination is formulated as

$$\begin{aligned}
&\frac{\partial S(t,a)}{\partial t} + \frac{\partial S(t,a)}{\partial a} = -(v(a) + \mu(a) + \lambda(t))S(t,a),\\
&\frac{\partial i(t,\tau,a)}{\partial t} + \frac{\partial i(t,\tau,a)}{\partial \tau} + \frac{\partial i(t,\tau,a)}{\partial a} = -(\mu(a) + \gamma(\tau))i(t,\tau,a),\\
&\frac{\partial r(t,\tau,a)}{\partial t} + \frac{\partial r(t,\tau,a)}{\partial \tau} + \frac{\partial r(t,\tau,a)}{\partial a} = -\mu(a)r(t,\tau,a) - \theta(\tau)\lambda(t)r(t,\tau,a),\\
&S(t,0) = \int_0^\infty m(a)(S(t,a) + I(t,a) + R(t,a))da,\\
&i(t,0,a) = \lambda(t)\left[S(t,a) + \int_0^a \theta(\tau)r(t,\tau,a)d\tau\right],\\
&r(t,0,a) = v(a)S(t,a) + \int_0^a \gamma(\tau)i(t,\tau,a)d\tau.
\end{aligned} \tag{87}$$

There then exists a disease–free (immunized) steady state:

$$S^*(a) = B\ell(a)V(a), \quad r^*(\tau, a) = P^*(a)W(a-\tau), \tag{88}$$

where $V(a) := e^{-\int_0^a v(\zeta)d\zeta}$ is the proportion of susceptible individuals who are not yet vaccinated at age a, and $W(a) := v(a)V(a)$ gives the probability density for vaccination at age a.

The effective susceptible population density is then given by

$$S_e(a) = S^*(a) + \int_0^a \theta(\tau)r^*(\tau, a)d\tau = P^*(a)[V(a) + (\theta * W)(a)], \tag{89}$$

and the effective reproduction number is calculated as

$$\mathcal{R}(v) = \int_0^\infty \Psi(a)\frac{S_e(a)}{N^*}da = \int_0^\infty \Psi(a)w^*(a)[V(a) + (\theta * W)(a)]da. \tag{90}$$

Suppose that susceptible individuals are vaccinated at age a_0 and the coverage proportion is $\epsilon \in [0, 1]$. Let $W(a) = \epsilon\delta(a - a_0)$ and $V(a) = 1 - \epsilon H(a - a_0)$, where δ is the Dirac function and H denotes the Heviside function. We can then calculate the effective reproduction number as

$$\begin{aligned}\mathcal{R}(\epsilon) = &\int_0^{a_0} \Psi(a)w^*(a)da \\ &+ (1-\epsilon)\int_{a_0}^\infty \Psi(a)w^*(a)da + \epsilon\int_{a_0}^\infty \Psi(a)w^*(a)\theta(a-a_0)da,\end{aligned} \tag{91}$$

and the critical coverage of immunization ϵ^* such that $\mathcal{R}(\epsilon^*) = 1$ is calculated as

$$\epsilon^* = \frac{R_0 - 1}{\int_{a_0}^\infty \Psi(a)w^*(a)(1-\theta(a-a_0))da}. \tag{92}$$

In particular, if a_0 goes to zero, (i.e., newborns are vaccinated), we have

$$\mathcal{R}(\epsilon) = (1-\epsilon)R_0 + \epsilon R_1, \tag{93}$$

where

$$R_1 := \int_0^\infty \Psi(a)w^*(a)\theta(a)da, \tag{94}$$

is the effective reproduction number of individuals vaccinated at age zero. The reinfection threshold is then given by $R_0 = 1/\sigma$ with

$$\sigma = \frac{R_1}{R_0} = \frac{\int_0^\infty \Psi(a)w^*(a)\theta(a)da}{\int_0^\infty \Psi(a)w^*(a)da}. \tag{95}$$

Although detailed analysis of the chronological-age dependent reinfection model is an open problem, the incorporation of the individual's epidemiological history with the host demography is crucial to developing epidemic models that are more realistic. In fact, the functions β and θ could be understood as a result of virus (or parasite) dynamics in vivo, i.e., they express the continuous process of the developments of individual infectivity and immunity. It is an interesting challenge to understand the spread of infectious diseases according to within host dynamics.

5. Asymptomatic transmission models

As shown above, it is not easy to realize subcritical endemic steady states without enhancement of susceptibility in the reinfection model. However, we can consider more realistic reinfection mechanisms that allow backward bifurcations. Here we consider two examples, malaria and measles.

5.1. Malaria dynamics

Although reinfected individuals are not distinguished from the infecteds resulting from completely susceptible individuals in the original Kermack–McKendrick model, it will become a natural extension if we assume that epidemiological parameters for the reinfecteds are different from parameters of the infecteds produced from completely susceptible individuals. In fact, Águas, *et al.* ([**1**]) developed an age-structured population model for the dynamics of malaria transmission, and observed that stable endemic steady states coexist with stable disease-free steady states. In their model, the infecteds resulting from completely susceptible individuals are clinical malaria cases, and recovery from clinical cases confers protection against the clinical manifestation of diseases, but not against infection *per se*. A recovered individual can then be reinfected and develops a non–clinical form of malaria, which can be called an *asymptomatic infection*.

According to the above consideration, we can extend the basic model (2) to an asymptomatic transmission model. For simplicity, again we here neglect the chronological age structure. Let $i_1(t,\tau)$ be the density of the infecteds resulting from the infection of completely susceptible individuals, and let β_1 be the transmission coefficient and γ_1 be the recovery rate. Let $i_2(t,\tau)$ be the density of reinfected individuals, β_2 the transmission coefficient of the reinfecteds and γ_2 be the recovered rate of the reinfecteds. We can then rewrite the basic model (2) as

$$\begin{aligned}
&\frac{\partial s(t,\tau)}{\partial t}+\frac{\partial s(t,\tau)}{\partial \tau}=-\mu s(t,\tau)-\lambda(t)s(t,\tau),\\
&\frac{\partial i_1(t,\tau)}{\partial t}+\frac{\partial i_1(t,\tau)}{\partial \tau}=-(\mu+\gamma_1(\tau))i_1(t,\tau),\\
&\frac{\partial i_2(t,\tau)}{\partial t}+\frac{\partial i_2(t,\tau)}{\partial \tau}=-(\mu+\gamma_2(\tau))i_2(t,\tau),\\
&\frac{\partial r(t,\tau)}{\partial t}+\frac{\partial r(t,\tau)}{\partial \tau}=-\mu r(t,\tau)-\theta(\tau)\lambda(t)r(t,\tau),
\end{aligned} \tag{96}$$

where the boundary condition is given as

$$\begin{aligned}
&s(t,0)=\mu N,\\
&i_1(t,0)=\lambda(t)\int_0^\infty s(t,\tau)d\tau,\\
&i_2(t,0)=\lambda(t)\int_0^\infty \theta(\tau)r(t,\tau)d\tau,\\
&r(t,0)=\int_0^\infty \gamma_1(\tau)i_1(t,\tau)d\tau+\int_0^\infty \gamma_2(\tau)i_2(t,\tau)d\tau,\\
&\lambda(t)=\int_0^\infty \beta_1(\tau)i_1(t,\tau)d\tau+\int_0^\infty \beta_2(\tau)i_2(t,\tau)d\tau.
\end{aligned} \tag{97}$$

Again we define

$$\langle\gamma_j,\Gamma_j\rangle:=\int_0^\infty e^{-\mu\tau}\gamma_j(\tau)\Gamma_j(\tau)d\tau,\quad \langle\beta_j,\Gamma_j\rangle:=\int_0^\infty e^{-\mu\tau}\beta_j(\tau)\Gamma_j(\tau)d\tau, \tag{98}$$

for $j=1,2$. It is easily seen that the basic reproduction number R_0 and the effective reproduction number R_1 of the fully vaccinated system are given by

$$R_0=N\langle\beta_1,\Gamma_1\rangle,\quad R_1=\theta^*N\langle\beta_2,\Gamma_2\rangle. \tag{99}$$

Let λ^* be the force of infection at an endemic steady state. We then have

$$i_2^*(0)=\frac{\mu N\lambda^*}{\mu+\lambda^*}\frac{\langle\gamma_1,\Gamma_1\rangle\phi(\lambda^*)}{1-\langle\gamma_2,\Gamma_2\rangle\phi(\lambda^*)}, \tag{100}$$

where ϕ is a function defined by (38).

Since

$$\lambda^*=\langle\beta_1,\Gamma_1\rangle\frac{\mu N\lambda^*}{\mu+\lambda^*}+\langle\beta_2,\Gamma_2\rangle i_2^*(0), \tag{101}$$

we have an equation satisfied by the force of infection:

$$R(\lambda^*) = \frac{\mu}{\mu+\lambda^*}\left\{R_0 + \frac{R_1}{\theta^*}\frac{\langle\gamma_1,\Gamma_1\rangle\phi(\lambda^*)}{1-\langle\gamma_2,\Gamma_2\rangle\phi(\lambda^*)}\right\} = 1, \tag{102}$$

where $R(0) = R_0$. Using the same kind of argument as used in section 3, we have the following.

PROPOSITION 5.1. *Endemic steady states backwardly bifurcate at $R_0 = 1$ if the condition*

$$\theta^*\frac{\langle\beta_2,\Gamma_2\rangle}{\langle\beta_1,\Gamma_1\rangle}\langle\gamma_1,\Gamma_1\rangle > 1, \tag{103}$$

holds.

If the net reproductivity of asymptomatic cases, given by $\langle\beta_2,\Gamma_2\rangle$, is larger than that of clinical cases given by $\langle\beta_1,\Gamma_1\rangle$, it is possible to satisfy the condition (103), even when $\theta^* \le 1$. This situation could occur if the duration of infection of the asymptomatic case is much longer, because it does not necessarily need clinical treatment.

We now calculate the effective reproduction number of the extended model (96). Suppose that ϵ denotes the proportion of immunization in the disease–free steady state. Let $\zeta_j(t) := i_j(t,0)$ be the density of the newly infecteds. We then obtain a system of renewal equations describing the disease invasion in the disease–free steady state:

$$\begin{aligned}\zeta_1(t) &= (1-\epsilon)N(\psi_1 * \zeta_1)(t) + (1-\epsilon)N(\psi_2 * \zeta_2),\\ \zeta_2(t) &= \epsilon N\theta^*(\psi_1 * \zeta_1)(t) + \epsilon N\theta^*(\psi_2 * \zeta_2),\end{aligned} \tag{104}$$

where $\psi_j(\tau) := e^{-\mu\tau}\beta_j(\tau)\Gamma_j(\tau)$.

Therefore the next generation matrix is given by

$$K = \begin{pmatrix}(1-\epsilon)N\langle\beta_1,\Gamma_1\rangle & (1-\epsilon)N\langle\beta_2,\Gamma_2\rangle\\ \epsilon N\theta^*\langle\beta_1,\Gamma_1\rangle & \epsilon N\theta^*\langle\beta_2,\Gamma_2\rangle\end{pmatrix}, \tag{105}$$

and the effective (vaccine) reproduction number is given by its spectral radius:

$$\begin{aligned}\mathcal{R}(\epsilon) &= (1-\epsilon)N\langle\beta_1,\Gamma_1\rangle + \epsilon N\theta^*\langle\beta_2,\Gamma_2\rangle\\ &= (1-\epsilon)R_0 + \epsilon R_1\\ &= (1-(1-\sigma)\epsilon)R_0,\end{aligned} \tag{106}$$

where $\sigma := R_1/R_0$, and $1/\sigma$ is the reinfection threshold of R_0. Therefore, the disease is uncontrollable if $R_0 > 1/\sigma$ and condition (103) is written as $\sigma\langle\gamma_1,\Gamma_1\rangle > 1$. If $\sigma > 1$, which is necessary to satisfy condition (103), we have $\mathcal{R}(\epsilon) > R_0$, and

the vaccination thus increases the reproduction number, although it decreases the reproduction number of clinical cases.

5.2. Measles dynamics in a vaccinated population

Finally, as an application of the reinfection model, let us consider an epidemic model of measles with fluctuation of the immunity level for vaccinees. In this model, we again assume that there are two sorts of infectious states. The host population is divided into five subpopulations: the completely susceptible population (s), the vaccinated population (v), the recovered population with complete immunity (r), the classical infectious population for measles (i_1), and the subclinical infectious population for measles (i_2). Different from the assumption of the Kermack–McKendrick reinfection model, the recovered individuals have complete immunity and no susceptibility, and instead, the vaccinated individuals have partial susceptibility (according to the waning of immunity) depending on the duration since vaccination. By (re)infection, some of the vaccinated individuals develop subclinical infection, and the immunity level of the remaining vaccinated individuals is boosted to the level of newly vaccinated individuals. The vaccinated population is structured by the duration since vaccination. We can then formulate vaccine-induced subclinical infection model for measles as

$$\begin{aligned}
&\frac{\partial s(t,\tau)}{\partial t}+\frac{\partial s(t,\tau)}{\partial \tau}=-\mu s(t,\tau)-\lambda(t)s(t,\tau),\\
&\frac{\partial i_1(t,\tau)}{\partial t}+\frac{\partial i_1(t,\tau)}{\partial \tau}=-(\mu+\gamma_1(\tau))i_1(t,\tau),\\
&\frac{\partial i_2(t,\tau)}{\partial t}+\frac{\partial i_2(t,\tau)}{\partial \tau}=-(\mu+\gamma_2(\tau))i_2(t,\tau),\\
&\frac{\partial r(t,\tau)}{\partial t}+\frac{\partial r(t,\tau)}{\partial \tau}=-\mu r(t,\tau),\\
&\frac{\partial v(t,\tau)}{\partial t}+\frac{\partial v(t,\tau)}{\partial \tau}=-\mu v(t,\tau)-\lambda(t)\theta(\tau)v(t,\tau),
\end{aligned} \tag{107}$$

where the boundary condition is given as

$$\begin{aligned}
&s(t,0)=(1-\epsilon)\mu N,\\
&i_1(t,0)=\lambda(t)\int_0^\infty s(t,\tau)d\tau,\\
&i_2(t,0)=(1-\kappa)\lambda(t)\int_0^\infty \theta(\tau)v(t,\tau)d\tau,\\
&r(t,0)=\epsilon p\mu N+\int_0^\infty \gamma_1(\tau)i_1(t,\tau)d\tau+\int_0^\infty \gamma_2(\tau)i_2(t,\tau)d\tau,\\
&v(t,0)=\epsilon\mu N(1-p)+\kappa\lambda(t)\int_0^\infty \theta(\tau)v(t,\tau)d\tau,
\end{aligned} \tag{108}$$

and the force of infection is given by

$$\lambda(t) = \int_0^\infty \beta_1(\tau) i_1(t,\tau) d\tau + \int_0^\infty \beta_2(\tau) i_2(t,\tau) d\tau. \tag{109}$$

Here ϵ is the vaccination coverage of newborns, p is the probability that vaccinated newborns develop complete immunity and κ is the probability that the immunity level of vaccinated individuals is boosted by infection, therefore, $1-\kappa$ gives the probability that the infection of vaccinated individuals leads to subclinical infection. The boosting effect is expressed by the "reset" of local time to zero. Kishida ([**16**]) investigated a special case of model (107)-(108) that γ_j and β_j are constants, and he found that multiple endemic steady states can exist.

Let λ^* be the force of infection in an endemic steady state. Again it is easy to derive the characteristic relation:

$$\begin{aligned} R(\lambda^*) :=& \frac{\mu N(1-\epsilon)}{\mu+\lambda^*} \langle \beta_1, \Gamma_1 \rangle \\ &+ \frac{(1-\kappa)(1-p)\epsilon\mu N \langle \beta_1, \Gamma_2 \rangle}{1-\kappa\phi(\lambda^*)} \int_0^\infty \theta(\tau) e^{-\mu\tau - \lambda^* \int_0^\tau \theta(\zeta) d\zeta} d\tau = 1. \end{aligned} \tag{110}$$

$R(0)$ then gives the effective reproduction number:

$$R(0) = R_e = (1-\epsilon)R_0 + \epsilon R_1. \tag{111}$$

Here R_0 is the basic reproduction number and R_1 is the effective reproduction number of the fully vaccinated system:

$$R_0 = N\langle \beta_1, \Gamma_1 \rangle, \quad R_1 = (1-\kappa)(1-p)N\theta^* \langle \beta_2, \Gamma_2 \rangle, \tag{112}$$

where θ^* is defined by (25).

If $p = 1$ or $\kappa = 1$, we have $R_1 = 0$ and the critical coverage of immunization is given by $\epsilon^* = 1 - 1/R_0$. Meanwhile, if $R_1 > 0$ and the reinfection threshold $\sigma = R_1/R_0$ is less than unity, the critical coverage of immunization is given by

$$\epsilon^* = \frac{1}{1-\sigma}\left(1 - \frac{1}{R_0}\right) > 1 - \frac{1}{R_0}, \tag{113}$$

which shows that if we take into account subclinical infection, the coverage of immunization to eradicate the disease must be larger than the critical proportion of immunization calculated from the standard SIR model neglecting the subclinical cases.

Given that $\lim_{\lambda\to\infty} R(\lambda) = 0$, there exists at least one endemic steady state if $R_e > 1$, while it is unclear whether endemic steady state uniquely exists or not.

We observe that

$$R'(0) = -\frac{1}{\mu}(1-\epsilon)R_0 + \epsilon D R_1, \tag{114}$$

where parameter D is defined by

$$D = -\frac{1}{\theta^*}\int_0^\infty \mu\theta(\tau)e^{-\mu\tau}\int_0^\tau \theta(\zeta)d\zeta d\tau + \kappa\frac{\theta^*}{\mu}. \tag{115}$$

Given that D is independent from β_j and it could become positive, it is possible that $R'(0) > 0$ under the condition $R(0) = R_e = 1$. In such a case, a backward bifurcation occurs at $R_e = 1$ and there exist subcritical endemic steady states.

PROPOSITION 5.2. *Endemic steady states backwardly bifurcate at $R_e = 1$ if the condition*

$$1 < (1 + \mu D\sigma)\epsilon, \tag{116}$$

holds.

Note that if θ is constant, $D = (\kappa-1)\theta/\mu < 0$, and the bifurcation at $R(0) = 1$ is thus supercritical. An introduction of imperfect vaccination would make it difficult to eradicate measles, although it can reduce the number of clinical cases.

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Hisashi Inaba

Graduate School of Mathematical Sciences, The University of Tokyo
3-8-1 Komaba, Meguro-ku, Tokyo 153-8914, Japan
inaba@ms.u-tokyo.ac.jp