Global stability properties of a class of renewal epidemic models

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Abstract
We investigate the global dynamics of a general Kermack–McKendrick-type epidemic model formulated in terms of a system of renewal equations. Specifically, we consider a renewal model for which both the force of infection and the infected removal rates are arbitrary functions of the infection age, \(\tau\), and use the direct Lyapunov method to establish the global asymptotic stability of the equilibrium solutions. In particular, we show that the basic reproduction number, \(R_0\), represents a sharp threshold parameter such that for \(R_0 \leq 1\), the infection-free equilibrium is globally asymptotically stable; whereas the endemic equilibrium becomes globally asymptotically stable when \(R_0 > 1\), i.e. when it exists.

Keywords Global stability · Lyapunov · Renewal · Kermack–McKendrick

Mathematics Subject Classification 92D30 · 34D23 · 34A34 · 37N25

1 Introduction

The classic Kermack–McKendrick paper (Kermack and McKendrick 1927) is a seminal contribution to the mathematical theory of epidemic modelling. Within, the authors formulate a general epidemic model in which the infectiousness of infected individuals and the rate at which they recover or are removed is an arbitrary function of the infection age, \(\tau\); from this, they derive several fundamental results including the
conditions for an epidemic outbreak and the final size equation. As a consequence of
their general formulation, the analysis and conclusions of the Kermack–McKendrick
paper encompass a wide class of epidemic models, including countless incarnations
that have since appeared in the infectious diseases modelling literature (e.g. the SIR
and SEIR models) (Breda et al. 2012).

In this article we revisit the classic Kermack–McKendrick model (Kermack and
McKendrick 1927) and further investigate the system properties and global dynamics
in the presence of demographic influences. Our main result, which is derived in Sect. 3,
is to show that the basic reproduction number \( R_0 \) represents a sharp threshold param-
eter that determines the global stability of the infection-free and endemic equilibria.
Specifically, we find that when \( R_0 \leq 1 \) the infection-free equilibrium point is the
unique equilibrium in the nonnegative orthant and is globally asymptotically stable
within this region. Conversely, when \( R_0 > 1 \) an endemic solution emerges which is
globally asymptotically stable when it exists. Both of these results are proved by the
direct Lyapunov method, that is, by identifying appropriate Lyapunov functionals.

Lyapunov functions have previously been used to establish the global asymptotic
stability properties of SIR, SIS and SIRS models [see e.g. (Korobeinikov 2004)] for
which the population is either constant (Korobeinikov and Wake 2002; O’Regan et al.
2010) or varying (Li and Muldowney 1995; Li et al. 1999). These results have also been
extended to SEIR and SEIS models in (Li and Muldowney 1995; Li et al. 1999; Fan
et al. 2001; McCluskey 2008), and epidemic models with multiple parallel infectious
stages (Korobeinikov 2008) or strains (Bichara et al. 2013). However, these results
have each been established within the context of compartmental-type epidemic models
for which the per-capita flow rates between the stages of infection are assumed to be
constant and infectiousness is fixed for the duration of their infectious period.

Only recently, by using an approach that relied on both the direct Lyapunov method
and semigroup theory, were (Magal et al. 2010) able to determine the global sta-
bility properties of equilibria in infection-age models. This work has since been
expanded (McCluskey 2008, 2009, 2010a) and extended to models with general inci-
dence functions (Huang and Takeuchi 2011; McCluskey 2010b; Chen et al. 2016;
Soufiane and Touaoula 2016) and multiple parallel infectious stages (Wang and Liu
2012) or strains (Martcheva and Li 2013). Here, we provide an alternative treatment
given in terms of the original renewal formulation of the Kermack–McKendrick model
[see also (Diekmann 1977; Metz and Diekmann 1986)].

In the next section we briefly describe the renewal system variables, parameters
and their governing equations, and then discuss the system phase-space. Then, in
Sect. 3, we derive the main result of this article where we introduce a set of Lyapunov
functionals which we use to establish the global asymptotic stability of the infection-
free and endemic equilibria.

## 2 Model description

In the renewal formulation of the Kermack–McKendrick model we need only explicitly
consider a class of susceptible (i.e. infection-naïve) individuals, \( S \), who each experi-
ence a time-dependent force of infection $F(t)$.\footnote{The dynamics of the class of infected individuals, $I$, is implicitly captured through the force of infection, $F(t)$.} By definition, the force of infection is the per-capita rate at which susceptibles become infected. Therefore the incidence at time $t$, $v(t)$, is given by

$$v(t) = F(t)S(t),$$

where $S(t)$ is the number of susceptibles and $v(t)$ describes the rate at which new infected individuals appear at time $t$. Assuming then that individuals who have been infected for $\tau$ units of time on average contribute an amount $A(\tau)$ to the force of infection, we find that the total force of infection at time $t$, $F(t)$, can be written in terms of a renewal equation:

$$F(t) = \int_{0}^{\infty} A(\tau)v(t - \tau) d\tau,$$

$$= \int_{0}^{\infty} A(\tau)F(t - \tau)S(t - \tau) d\tau.$$

Here $v(t - \tau)$ represents the incidence at time $t - \tau$.

In general, the infectivity kernel $A \geq 0$ is an arbitrary function of the infection age $\tau$ whose definition motivates us, in analogy with (Magal et al. 2010), to define

$$\bar{\tau} = \sup \{ \tau \geq 0 : A(\tau) > 0 \},$$

the maximum infection age at which an individual can contribute to the force of infection. Here we assume $\bar{\tau} < \infty$; the case $\bar{\tau} = \infty$ is much more difficult to address, and we do not attempt to cover this case [see (Diekmann et al. 2008; Diekmann and Gyllenberg 2012) for results in this direction]. In this case we need only look back to this maximum infection age to calculate $F(t)$:

$$F(t) = \int_{0}^{\bar{\tau}} A(\tau)v(t - \tau) d\tau,$$

$$= \int_{0}^{\bar{\tau}} A(\tau)F(t - \tau)S(t - \tau) d\tau.$$ \hspace{1cm} (2)

To complete the model description we assume that in addition to removal by infection, individuals are recruited into the susceptible class at a constant rate $\lambda$ and die naturally at a constant per-capita rate $\mu$. Combining these rates, we find that

$$\frac{dS(t)}{dt} = \lambda - \mu S(t) - F(t)S(t),$$

assuming that infection leads to permanent immunity. Given (3), it is straightforward to show that $S(t) > 0$ for $t > 0$ provided it is nonnegative initially.
An important parameter that governs the system trajectory is the basic reproduction number \( R_0 \), defined as the expected number of secondary cases caused by a single (typical) infectious individual in a fully susceptible population. Given the definition of \( A(\tau) \) and the expression for \( F(t) \) [Eq. (2)], the functional form of \( R_0 \) is naturally given by

\[
R_0 = S^0 \int_0^\tau A(\tau) \, d\tau
\]  

(4)

where

\[
S^0 = \frac{\lambda}{\mu}
\]  

(5)

is the steady-state susceptible population in the absence of infection (see below).

Before introducing suitable initial conditions for the system, we emphasize that in order to solve (2) and (3) we must have knowledge of the entire past history of \( F \) and \( S \) over the interval \( \tau \in [\bar{\tau}, 0] \). Therefore, the state of our system \( P = (S, F) \), where \( S \) and \( F \) are functions defined over the interval \( [\bar{\tau}, 0] \) describing the history of susceptibles and the force of infection respectively, belongs to an infinite-dimensional phase-space \( \Omega \). For our application, \( \Omega \) can appropriately be chosen as

\[
\Omega = C^0_+([-\bar{\tau}, 0]) \times L^1_+(-\bar{\tau}, 0)
\]

which is a Banach space with the natural norm

\[
\|(S, F)\| = \sup_{s \in [-\bar{\tau}, 0]} |S(s)| + \int_{-\bar{\tau}}^0 |F(s)| \, ds.
\]

With this choice of state-space, standard arguments show that the model (2)–(3) is well defined.

Given \( \Omega \), a suitable choice of initial conditions is given by

\[
S_0 \in C^0_+([-\bar{\tau}, 0]) \quad \text{and} \quad F_0 \in L^1_+(-\bar{\tau}, 0).
\]

The system Eqs. (2)–(3) induce a continuous semiflow \( \Phi_t : \Omega \to \Omega \) where the trajectory is given by \( (S_t(\cdot), F_t(\cdot)) \in \Omega \) with

\[
S_t(s) = S(t+s), \quad F_t(s) = F(t+s), \quad s \in [-\bar{\tau}, 0].
\]

We point out that in this notation, the pair \( (S_t(0), F_t(0)) = (S(t), F(t)) \) represents the most recent value in the history of a state along the system trajectory at time \( t \),

\[\text{Here, } C^k \text{ is the set of functions whose } k \text{-th derivative is continuous; } L^p \text{ is the space of functions for which the } p \text{-th power of the absolute value is Lebesgue measurable; a subscript ‘+’ implies that the function is nonnegative; and the quantity in the brackets denotes the region over which the function is defined. For example, } C^1_+([-\bar{\tau}, 0]) \text{ is the set of nonnegative functions defined over the interval } [-\bar{\tau}, 0] \text{ whose first derivative is continuous.}\]
namely \((S_t(\cdot), F_t(\cdot)) \in \Omega\). In this case the model equations (2)–(3) can be understood as rules for updating the most recent values of the histories of \(F\) and \(S\) respectively.

**Lemma 1** If the infectivity kernel \(A\) is of bounded variation, that is \(A \in BV([0, \bar{\tau}])\), system trajectories \((S_t(\cdot), F_t(\cdot))\) generated by model equations (2)–(3) that originate in \(\Omega\) are eventually compact. That is, for sufficiently large \(t\), \(\Phi_t : \Omega \to \Omega^c\) where \(\Omega^c \subset \Omega\) is some relatively compact set.

**Proof of Lemma 1** To begin, we rewrite Eq. (2) using the notation introduced above:

\[
F(t) = \int_0^{\bar{\tau}} A(\tau)F_t(-\tau)S_t(-\tau) \, d\tau.
\]

First, consider the interval \(t \in [0, \bar{\tau}]\). Corollary 1 of theorem 2 in (Mikusiński and Ryll-Nardzewski 1951) asserts that the convolution of a function of bounded variation with a bounded function is continuous. Therefore, from (6) we have that \(F(t > 0) \in C^0_+\) for \(F_0 \in L^1_+\) and \(S_0 \in C^1_+\). By substituting this result back into (3), we consequently find that \(S(t > 0) \in C^1_+\) from which it follows that \(\Phi_{t > \bar{\tau}} : \Omega \to C^1_+([-\bar{\tau}, 0]) \times C^0_+([-\bar{\tau}, 0])\).

Consider now the interval \(t > \bar{\tau}\). By theorem 5 of (Mikusiński and Ryll-Nardzewski 1951)—which states that the convolution of a function of bounded variation with a continuous function is absolutely continuous—we have that \(F(t > \bar{\tau}) \in AC_+\). Therefore, \(\Phi_{t > 2\bar{\tau}} : \Omega \to \tilde{\Omega}\) where

\[
\tilde{\Omega} = C^1_+([-\bar{\tau}, 0]) \times AC_+([-\bar{\tau}, 0]).
\]

Finally, by straightforward computations we can show that model extensions generated by Eqs. (2) and (3) are uniformly bounded in the natural topology of \(C^1 \times AC\). Hence, system trajectories originating in \(\Omega\) enter a bounded subset \(\Omega^c \subset \tilde{\Omega} \subset \Omega\) that is relatively compact.

Henceforth we assume that \(A \in BV_+([0, \bar{\tau}])\) such that the system trajectory is eventually compact and the \(\omega\)-limit set of (2)–(3) is non-empty.

In the following, we decompose \(\Omega\) into an “interior” and a “boundary” set \(\hat{\Omega}\) and \(\partial \Omega\), respectively. Here, we do not refer to topological concepts, but rather to the interpretation in view of our application. For all initial values in the interior, the force of infection is non-zero. The elements in the boundary set, in turn, do have a vanishing force of infection and therefore lead to trivial dynamics. To be more precise, we define

\[
\hat{\Omega} = \left\{(S, F) \in \Omega : \exists a \in [0, \bar{\tau}] \text{ s.t. } \int_0^{\bar{\tau}} A(\tau + a)F(-\tau)S(-\tau) \, d\tau > 0 \right\}
\]

and

\[
\partial \Omega = \Omega \setminus \hat{\Omega}.
\]

\(^3\) To show this, we could, for example, consider the infection-age formulation of the system (2)–(3) (which tracks the infected cohort at time \(t\), \(i(t, \tau)\), as opposed to the force of infection, \(F(t)\)) or, alternatively, we could use the Lyapunov functionals introduced below.
Finally, it is easy to verify that the fixed states of the system (2)–(3) are given by

\[ P_0 = (S^0, F^0) = (S^0, F^0) = \left( \frac{\lambda}{\mu}, 0 \right). \]

\[ \tilde{P} = (\tilde{S}, \tilde{F}) = (\tilde{S}, \tilde{F}) = \left( \frac{\lambda}{\mu R_0}, \mu (R_0 - 1) \right). \]  \hspace{1cm} (8)

Importantly, we see that the endemic equilibrium point, \( \tilde{P} \), only exists in the interior region \( \hat{\Omega} \) for \( R_0 > 1 \); for the limiting case \( R_0 = 1 \), the endemic and infection-free equilibria coincide.

Ultimately, our goal will be to establish that (i) when \( R_0 \leq 1 \) all system trajectories of (2)–(3) within \( \Omega \) asymptotically approach the infection-free equilibrium point \( P_0 \in \partial \Omega \) and (ii) when \( R_0 > 1 \) trajectories that originate in \( \Omega \) asymptotically approach the endemic equilibrium \( \tilde{P} \in \hat{\Omega} \), except those that originate in \( \partial \Omega \) which approach \( P_0 \).

### 3 Global stability analysis

#### 3.1 Infection-free equilibrium

**Theorem 1** The infection-free equilibrium point \( P_0 \) of the system (2)–(3) is globally asymptotically stable in \( \Omega \) for \( R_0 \leq 1 \). However, if \( R_0 > 1 \), solutions of (2)–(3) starting sufficiently close to \( P_0 \) in \( \Omega \) leave a neighbourhood of \( P_0 \), except those starting within the boundary region \( \partial \Omega \) which approach \( P_0 \).

**Proof of Theorem 1** To verify theorem 1 we define \( D = \Phi(\Omega) \) where, from (3), we have \( S(0) > 0 \) for \( (S, F) \in D \). Note, \( D \) is forward invariant and any trajectory originating in \( \Omega \) enters \( D \) either at, or before \( t = \bar{\tau} \).

Consider the Lyapunov functional \( U : D \rightarrow \mathbb{R}_+ \) defined by

\[ U(S, F) = g \left( \frac{S(0)}{S^0} \right) + \int_0^{\bar{\tau}} \eta(\tau) F(-\tau) S(-\tau) d\tau \]

where

\[ g(x) = x - 1 - \log x \quad \text{and} \quad \eta(\tau) = \int_{\tau}^{\bar{\tau}} A(s) ds. \]  \hspace{1cm} (9)

In particular we have \( \eta(\bar{\tau}) = 0 \),

\[ \eta(0) = \frac{R_0}{S^0} \quad \text{and} \quad \eta'(\tau) = -A(\tau) \]  \hspace{1cm} (10)

where \( a' \) denotes differentiation with respect to \( \tau \). Importantly, the functional \( U(S, F) \geq 0 \) is well defined since \( S(0) > 0 \), and has a global minimum at the infection-free equilibrium \( P_0 \).
Next, let \((S_t(\cdot), F_t(\cdot))\) be a trajectory of the model (2)–(3) with initial condition in \(D\). With \(S_t(s) = S(t + s)\) and \(F_t(s) = F(t + s)\) we may write

\[
U(S_t(\cdot), F_t(\cdot)) = g \left( \frac{S_t(0)}{S^0} \right) + \int_0^{\bar{t}} \eta(\tau) F_t(-\tau) S_t(-\tau) d\tau,
\]

where in the second line we have substituted in (10) and in the last line we have used \(\lambda\) where in the second line we have substituted in the identity

\[
U = \int_0^{\bar{t}} \eta(\tau) F(t - \tau) S(t - \tau) d\tau.
\]

In order to compute the time derivative of \(U(S_t, F_t)\) we rewrite this as

\[
U(S_t(\cdot), F_t(\cdot)) = g \left( \frac{S(t)}{S^0} \right) + \int_{t-\bar{t}}^{t} \eta(t - s) F(s) S(s) ds. \tag{11}
\]

Differentiating each term in (11) along system trajectories separately, we first have

\[
\frac{d}{dt} \left[ g \left( \frac{S(t)}{S^0} \right) \right] = \left( \frac{1}{S^0} - \frac{1}{S(t)} \right) \frac{dS(t)}{dt},
\]

where in the second line we have substituted in the identity \(\lambda = \mu S^0\).

Next we differentiate the second term in (11) to obtain

\[
\frac{d}{dt} \left( \int_{t-\bar{t}}^{t} \eta(t - s) F(s) S(s) ds \right) = \eta(0) F(t) S(t) - \eta(\bar{t}) F(t - \bar{t}) S(t - \bar{t})
\]

\[
+ \int_{t-\bar{t}}^{t} \frac{d\eta(t - s)}{dt} F(s) S(s) ds,
\]

where in the second line we have substituted in (10) and in the last line we have used the definition of \(F(t)\), Eq. (2).

Finally, combining (12) and (13) yields

\[
\frac{d}{dt} U(S_t, F_t) = -\mu \frac{S_t(0)}{S^0} \left( 1 - \frac{S^0}{S_t(0)} \right)^2 - (1 - R_0) \frac{S_t(0)}{S^0}. \tag{14}
\]
We emphasize that we know for a trajectory \((S, F) \in D \subset \Omega\), that for \(t > \bar{\tau}\) we already have \(F \in C^0([\bar{\tau}, 0])\), such that this expression is well defined and \(U\) is a proper Lyapunov functional on the domain \(D\).

Importantly, for \(R_0 \leq 1\) we have \(dU/dt \leq 0\). The derivative \(\dot{U}(t) = 0\) if and only if \(S(0) = S^0\) and either (a) \(R_0 = 1\) or (b) \(F(0) = 0\). Therefore, the largest invariant subset in \(\Omega\) for which \(\dot{U} = 0\) is the singleton \(\{P_0\}\). By Lemma 1 the orbit is eventually precompact hence, by the infinite-dimensional form of LaSalle’s extension of Lyapunov’s global asymptotic stability theorem (Smith 2010, Theorem 5.17), the infection-free equilibrium point \(P_0\) is globally asymptotically stable in \(\Omega\) for \(R_0 \leq 1\).

Conversely, if \(R_0 > 1\) and \(F(0) > 0\), the derivative \(\dot{U} > 0\) if \(S(t)\) is sufficiently close to \(S^0\). Therefore, solutions starting sufficiently close to the infection-free equilibrium point \(P_0\) leave a neighbourhood of \(P_0\), except those starting in \(\partial \Omega\). Since \(\dot{U} \leq 0\) for solutions starting in \(\partial \Omega\) these solutions approach \(P_0\) through this subspace.

\[\square\]

### 3.2 Endemic equilibrium

**Theorem 2** If \(R_0 > 1\) the endemic equilibrium point \(\check{P}\) is globally asymptotically stable in \(\hat{\Omega}\) (i.e. away from the boundary region \(\partial \Omega\)).

**Proof of Theorem 2** First, in theorem 1 we observed that \(F(t)\) for \(t > 0\) is bounded away from zero when \(R_0 > 1\), such that for \(R_0 > 1\) the semiflow \(\Phi_t : \hat{\Omega} \rightarrow \hat{\Omega}\). Therefore, in analogy with theorem 1 we define \(\check{D} = \Phi_{\bar{\tau}}(\hat{\Omega})\) which is a forward-invariant set for \(R_0 > 1\). Moreover \(S, F > 0\) for \((S, F) \in \check{D}\) and any trajectory originating in \(\hat{\Omega}\) enters \(\check{D}\) at the latest at time \(t = \bar{\tau}\), provided \(R_0 > 1\).

In this case we define \(W : \check{D} \rightarrow \mathbb{R}_+\)

\[
W(S, F) = g\left(\frac{S(0)}{S}\right) + \int_0^{\bar{\tau}} \chi(\tau) g\left(\frac{F(-\tau)S(-\tau)}{FS}\right) d\tau
\]

where \(g(x)\) has been defined previously in (9) and

\[
\chi(\tau) = \bar{F}\bar{S} \int_\tau^{\bar{\tau}} A(s) ds.
\]

---

*(LaSalle Invariance Principle)* Given a Lyapunov functional \(V : D \rightarrow \mathbb{R}_+\), consider the set

\[
E = \{u \in D \mid \dot{V}(u) = 0\}.
\]

If for \(u_0 \in D\) the orbit \(\Phi_t(u_0)\) is precompact (i.e. lies in a compact set of \(D\), then

\[
\lim_{t \to \infty} d(\Phi_t(u_0), M) = 0
\]

where \(M\) is the largest (positively) invariant subset of \(E\) and \(d\) denotes distance.
Immediately we have that $\chi(\bar{\tau}) = 0$,

$$\chi(0) = \bar{F} \quad \text{and} \quad \chi'(\tau) = -\bar{F}\bar{S}A(\tau).$$

Once again, note that $W(S, \mathcal{F})$ is well defined on $\hat{D}$.

Similar to before, we let $(S_t(\cdot), \mathcal{F}_t(\cdot))$ be a trajectory of the model with initial condition in $\hat{D}$ and adopt the notation $S_t(s) = S(t + s)$ and $\mathcal{F}_t(s) = F(t + s)$. We may then write

$$W(S_t(\cdot), \mathcal{F}_t(\cdot)) = g\left(\frac{S(t)}{\bar{S}}\right) + \int_0^{\bar{\tau}} \chi(\tau) g\left(\frac{F(t-\tau)S(t-\tau)}{\bar{F}\bar{S}}\right) d\tau$$

which we at once rewrite as

$$W(S_t(\cdot), \mathcal{F}_t(\cdot)) = g\left(\frac{S(t)}{\bar{S}}\right) + \int_{t-\bar{\tau}}^t \chi(t-s) g\left(\frac{F(s)S(s)}{\bar{F}\bar{S}}\right) ds. \quad (15)$$

Once again we differentiate each term separately. Beginning with the first term in (15) we have

$$\frac{d}{dt} \left[ g\left(\frac{S(t)}{\bar{S}}\right) \right] = \left(\frac{1}{\bar{S}} - \frac{1}{S(t)}\right) \frac{dS(t)}{dt},$$

$$= \frac{\lambda}{\bar{S}} - \frac{S(t)}{\bar{S}} - F(t) \frac{S(t)}{\bar{S}} - \frac{\lambda}{S(t)} + \mu + F(t),$$

$$= \mu \left(2 - \frac{S(t)}{\bar{S}} - \frac{\bar{S}}{S(t)}\right) + \bar{F} \left(1 - \frac{\bar{S}}{S(t)}\right) + F(t) \left(1 - \frac{S(t)}{\bar{S}}\right),$$

$$= -\mu \frac{S(t)}{\bar{S}} \left(1 - \frac{\bar{S}}{S(t)}\right)^2 + \bar{F} \left(1 - \frac{\bar{S}}{S(t)}\right) + F(t) \left(1 - \frac{S(t)}{\bar{S}}\right) \quad (16)$$

where in the third line we have substituted in the identity $\lambda = \mu \bar{S} + \bar{F}\bar{S}$.

Turning to the second term we find

$$\frac{d}{dt} \left[ \int_{t-\bar{\tau}}^t \chi(t-s) g\left(\frac{F(s)S(s)}{\bar{F}\bar{S}}\right) ds \right] = \chi(0) g\left(\frac{F(t)S(t)}{\bar{F}\bar{S}}\right) - \chi(\bar{\tau}) g\left(\frac{F(t-\bar{\tau})S(t-\bar{\tau})}{\bar{F}\bar{S}}\right)$$

$$+ \int_{t-\bar{\tau}}^t \frac{d\chi(t-s)}{ds} g\left(\frac{F(s)S(s)}{\bar{F}\bar{S}}\right) ds,$$

$$= \bar{F} \left(\frac{F(t)S(t)}{\bar{F}\bar{S}}\right) - \bar{F}\bar{S} \int_{t-\bar{\tau}}^t A(t-s) g\left(\frac{F(s)S(s)}{\bar{F}\bar{S}}\right) ds.$$
Substituting in the definition $g(x) = x - 1 - \log x$ and Eq. (2) this expression becomes

$$
\frac{d}{dt} \left[ \int_{t-\bar{\tau}}^{t} \chi(t-s) \ g \left( \frac{F(s)S(s)}{FS} \right) \ ds \right]
$$

$$
= F(t) \left( \frac{S(t)}{S} - 1 \right) - \tilde{F} \left[ \log \left( \frac{F(t)S(t)}{FS} \right) \right]
$$

$$
- \tilde{S} \int_{t-\bar{\tau}}^{t} A(t-s) \log \left( \frac{F(s)S(s)}{FS} \right) \ ds \right].
$$

(17)

The final term in the square brackets can be bounded using Jensen’s inequality$^5$:

$$
\tilde{S} \int_{t-\bar{\tau}}^{t} A(t-s) \log \left( \frac{F(s)S(s)}{FS} \right) \ ds \leq \log \left[ \tilde{S} \int_{t-\bar{\tau}}^{t} A(t-s) \frac{F(s)S(s)}{FS} \ ds \right],
$$

$$
= \log \left( \frac{F(t)}{F} \right).
$$

Importantly, we note that equality between the left- and right-hand sides occurs if and only if $F(t)S(t) = \tilde{F}\tilde{S}$. Substituting this result back into the expression above we find

$$
\log \left( \frac{F(t)S(t)}{FS} \right) - \tilde{S} \int_{t-\bar{\tau}}^{t} A(t-s) \log \left( \frac{F(s)S(s)}{FS} \right) \ ds
$$

$$
\geq \log \left( \frac{F(t)S(t)}{FS} \right) - \log \left( \frac{F(t)}{F} \right),
$$

$$
= \log \left( \frac{S(t)}{S} \right),
$$

$$
\geq 1 - \frac{\tilde{S}}{S(t)}
$$

where in the last line we have used $\log x \geq 1 - \frac{1}{x}$, where equality requires $S(t) = \tilde{S}$. This condition implies that

$$
\frac{d}{dt} \left[ \int_{t-\bar{\tau}}^{t} \chi(t-s) \ g \left( \frac{F(s)S(s)}{FS} \right) \ ds \right] \leq F(t) \left( \frac{S(t)}{S} - 1 \right) - \tilde{F} \left( 1 - \frac{\tilde{S}}{S(t)} \right).
$$

(18)

$^5$ For a concave function $\varphi(\cdot)$ the following inequality holds (Jensen 1906):

$$
\varphi \left( \int_{0}^{\infty} h(t) f(t) \ dt \right) \geq \int_{0}^{\infty} h(t)\varphi \left( f(t) \right) \ dt
$$

where $h(t)$ is a normalized probability distribution.
Finally, combining (16) and (18) yields
\[
\frac{d}{dt} W(S_t, F_t) \leq -\mu S_t(0) \left( 1 - \frac{\bar{S}}{S_t(0)} \right)^2, \\
\leq 0.
\] (19)

From Eq. (19) we see that the largest invariant subset in \( \hat{\Omega} \) for which \( \dot{W} = 0 \) consists only of the endemic equilibrium point \( \bar{P} \). By Lemma 1 the orbit is eventually precompact hence, by LaSalle’s extension of Lyapunov’s asymptotic stability theorem, the endemic equilibrium point \( \bar{P} \) is globally asymptotically stable in \( \hat{\Omega} \). □

### 4 Conclusions

In this article we investigated the global dynamics of the general Kermack–McKendrick model, formulated in terms of a set of renewal equations and supplemented with simple demographic dynamics. First, we demonstrated that when the basic reproduction number \( R_0 \leq 1 \) the infection-free equilibrium point \( P_0 \) is the unique equilibrium in \( \Omega = C_0^0((-\bar{\tau}, 0]) \times L_1^1((-\bar{\tau}, 0]) \). In contrast, when \( R_0 > 1 \), an endemic equilibrium solution emerges in \( \hat{\Omega} \subset \Omega \) for which a positive fraction of the population remains infected. Most significantly, by introducing appropriate Lyapunov functionals we established that the infection-free and endemic equilibria are globally asymptotically stable within \( \Omega \) and \( \hat{\Omega} \) when \( R_0 \leq 1 \) and \( R_0 > 1 \), respectively. These results generalize a number of previous investigations into the global stability of epidemic models.

We emphasize that one of the goals of this article was to promote the use of more general epidemic models beyond ordinary differential equation (compartmental) descriptions—that are ubiquitous in the infectious diseases modelling literature—by providing additional tools and theory relevant to the renewal formulation. However, whilst the model analyzed here considered somewhat general transmission dynamics by allowing the expected contribution to the force of infection at infection-age \( \tau \) to be left as an arbitrary function with only moderate restrictions, the assumptions regarding the demographic properties of the population are more restrictive. In particular, the model that we have analyzed [Eqs. (2) and (3)] assumes that the age distribution of our population is exponential, i.e. that the natural mortality rate \( \mu \) is constant. For many settings, particularly developed countries, this assumption may be unrealistic. Therefore, it would be interesting to investigate whether our analysis could be extended to epidemic models for which the natural mortality rate \( \mu \rightarrow \mu(a) \) is an arbitrary function of an individual’s chronological age, \( a \). Research in this direction dates back to at least (Thieme 1991) [see also (Müller and Kuttler 2015; Thieme 2011)] who found that Hopf bifurcations arise when an individual’s infectivity is dependent on their chronological age. Whether this applies to age-structured models in general is a promising avenue of future research.
References

Bichara D, Iggidr A, Sallet G (2013) Global analysis of multi-strains SIS, SIR and MSIR epidemic models. J Appl Math Comput 44(1):273–292
Breda D, Diekmann O, De Graaf W, Pugliese A, Vermiglio R (2012) On the formulation of epidemic models (an appraisal of Kermack and McKendrick). J Biol Dyn 6(sup2):103–117
Chen Y, Zou S, Yang J (2016) Global analysis of an SIR epidemic model with infection age and saturated incidence. Nonlinear Anal Real World Appl 30:16–31
Diekmann O (1977) Limiting behaviour in an epidemic model. Nonlinear Anal Theory Methods Appl 1(5):459–470
Diekmann O, Gyllenberg M (2012) Equations with infinite delay: blending the abstract and the concrete. J Differ Equ 252(2):819–851
Diekmann O, Getto P, Gyllenberg M (2008) Stability and bifurcation analysis of Volterra functional equations in the light of suns and stars. SIAM J Math Anal 39(4):1023–1069
Fan M, Li MY, Wang K (2001) Global stability of an SEIS epidemic model with recruitment and a varying total population size. Math Biosci 170(2):199–208
Huang G, Takeuchi Y (2011) Global analysis on delay epidemiological dynamic models with nonlinear incidence. J Math Biol 63(1):125–139
Jensen JLV (1906) Sur les fonctions convexes et les inégalités entre les valeurs moyennes. Acta Math 30(1):175–193
Kermack WO, McKendrick AG (1927) A contribution to the mathematical theory of epidemics. Proc R Soc Lond Math Phys Eng Sci 115(772):700–721
Korobeinikov A (2004) Global properties of basic virus dynamics models. Bull Math Biol 66(4):879–883
Korobeinikov A (2008) Global properties of SIR and SEIR epidemic models with multiple parallel infectious stages. Bull Math Biol 71(1):75–83
Korobeinikov A, Wake G (2002) Lyapunov functions and global stability for SIR, SIRS, and SIS epidemiological models. Appl Math Lett 15(8):955–960
Li MY, Muldowney JS (1995) Global stability for the SEIR model in epidemiology. Math Biosci 125(2):155–164
Li MY, Graef JR, Wang L, Karsai J (1999) Global dynamics of a SEIR model with varying total population size. Math Biosci 160(2):191–213
Magal P, McCluskey C, Webb G (2010) Lyapunov functional and global asymptotic stability for an infection-age model. Appl Anal 89(7):1109–1140
Martcheva M, Li XZ (2013) Competitive exclusion in an infection-age structured model with environmental transmission. J Math Anal Appl 408(1):225–246
McCluskey CC (2008) Global stability for a class of mass action systems allowing for latency in tuberculosis. J Math Anal Appl 338(1):518–535
McCluskey CC (2009) Global stability for an SEIR epidemiological model with varying infectivity and infinite delay. Math Biosci Eng 6(3):603–610
McCluskey CC (2010a) Complete global stability for an SIR epidemic model with delay: distributed or discrete. Nonlinear Anal Real World Appl 11(1):55–59
McCluskey CC (2010b) Global stability for an SIR epidemic model with delay and nonlinear incidence. Nonlinear Anal Real World Appl 11(4):3106–3109
Metz JAJ, Diekmann O (1986) The dynamics of physiologically structured populations. Lecture notes in biomathematics, vol. 68. Springer, Berlin
Mikusiński J, Ryll-Nardzewski C (1951) Sur le produit de composition. Studia Mathematica 12:51–57
Müller J, Kuttler C (2015) Methods and models in mathematical biology. Springer, Berlin
O’Regan SM, Kelly TC, Korobeinikov A, O’Callaghan MJ, Pokrovskii AV (2010) Lyapunov functions for SIR and SIRS epidemic models. Appl Math Lett 23(4):446–448
Smith H (2010) An introduction to delay differential equations with applications to the life sciences. Springer, Berlin
Soufiane B, Touaoula TM (2016) Global analysis of an infection age model with a class of nonlinear incidence rates. J Math Anal Appl 434(2):1211–1239
Thieme HR (1991) Stability change of the endemic equilibrium in age-structured models for the spread of S–I–R type infectious diseases. Springer Berlin Heidelberg, Berlin, Heidelberg, pp 139–158
Thieme HR (2011) Global stability of the endemic equilibrium in infinite dimension: Lyapunov functions and positive operators. J Differ Equ 250(9):3772–3801
Wang X, Liu S (2012) Global properties of a delayed SIR epidemic model with multiple parallel infectious stages. Math Biosci Eng 9(3):685–695

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