

A Novel Characterization of the Incidence Term in Epidemic Models: Interpretation of the Resulting Basic Reproduction Number in Terms of Healthcare Accessibility

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Abstract: We propose a novel characterization of the incidence term in mathematical epidemic models. This new characterization takes into consideration the fact that at any given time, only a proportion κ ($0 \leq \kappa \leq 1$), of infectives have access to healthcare. Infectives without access remain untreated and therefore, become reservoirs for new transmissions. The proposed incidence term is a decreasing function of κ , the associated recovery term is an increasing function of κ , while the disease-induced death term is a decreasing function of κ . These observations indicate that, increasing κ reduces new infection cases, speeds up recovery and reduces disease-induced deaths. Furthermore, we prove that the resulting basic reproduction number has a maximum value at $\kappa = 0$, and a minimum value at $\kappa = 1$. The parameter κ thus, exemplifies the role of healthcare accessibility in effectively controlling the spread of infectious diseases. Illustrations using a modified Kermack-McKendrick, SIR, SEIR and Vector-Host epidemic models, with some numerical simulations, are discussed.

Keywords: Epidemic Models, Incidence Term, Recovery Term, Disease-Induced Death Term, Basic Reproduction Number, Healthcare Accessibility Parameter.

Introduction

There are many infectious diseases for which there is a cure yet they remain endemic worldwide.

Low-Income and Middle-Income countries (LMICs) bear a disproportionately high burden of these infectious diseases, when compared with wealthier countries. This is due to a combination of factors, including poor sanitation, limited access to healthcare, inadequate vaccination coverage, presence of specific vectors, poverty and malnutrition (Guerin, 2023).

Africa carries over 20% of the global burden of disease (Niohuru, 2023).

For effective control of the spread of any infectious disease, healthcare accessibility is as important as the existence of a cure. Geographical accessibility to healthcare is an important component of infectious disease dynamics (Hierink et al., 2021). Proximity to a healthcare facility is an essential aspect of the fight against infectious diseases, as it ensures timely intervention, and prevents diseases from spreading to more people.

In areas with few public health facilities and low accessibility, the probability of detecting a case is also a

function of the distance between an infected person and the “observer” (e.g. a health centre) (Nelli et al., 2020). When the outbreak of an infectious disease occurs in communities without adequate healthcare facilities, the delay in detection will cause the infection to spread to more people, and eventually, overwhelm the healthcare system.

Apart from spatial accessibility, another hindrance to healthcare is physician-to-population ratio. The World Health Organization (WHO) recommends at least 1 physician per 1,000 people. Many countries, especially in Africa, and the rest of the developing world, fall short of this ratio (World Bank, 2026).

Some examples of endemic infectious diseases for which there is a cure, yet remain endemic include:

- Malaria (Oke et al., 2020; Packard, 2021; Collins and Duffy, 2022; Niohuru, 2023; Appiah et al., 2024; World Health Organization, 2025)
- HIV/AIDS: HIV does not have a cure. Anti-Retroviral Therapy (ART) is the main treatment used to control HIV. ART helps those living with HIV to live longer (Yusuf and Benyah, 2012; Makusha et al., 2020; Pillay and Johnson, 2021)
- Tuberculosis (TB) (Heaton, 2022; Liyew et al., 2025; Osei-Wusu et al., 2025)

- Measles (Poletti et al., 2018; Alemneh and Belay, 2023; Pokharel et al., 2024; Alnafisah and Sohaly, 2025)
- Influenza (flu) (Lofgren et al., 2007; Neumann and Kawaoka, 2022; Carlos et al., 2024)

Every year, Malaria, HIV/AIDS and Tuberculosis kill over 2.8 million people, and this burden is felt disproportionately by less wealthy countries (Guerin, 2023).

The term incidence, refers to the rate at which new cases of an infectious disease occur within a population over a specific time period.

In mathematical models, the incidence term,

$$\lambda(t)S(t), \quad (1)$$

typically involves a product of the force of infection $\lambda(t)$, and the susceptible population $S(t)$, (Anderson and May, 1991; Diekmann and Heesterbeek, 2000; Brauer and Castillo-Chavez, 2001; Martchev, 2015).

Among the most common incidence terms are the bilinear incidence term, and the standard incidence term, given respectively, by

$$\lambda_1(t)S(t) = \beta I(t)S(t), \quad (2a)$$

with force of infection $\lambda_1(t) = \beta I(t)$ and $I(t)$ is the prevalence;

$$\lambda_2(t)S(t) = \frac{\beta I(t)}{N(t)}S(t), \quad (2b)$$

with corresponding force of infection $\lambda_2(t) = \frac{\beta I(t)}{N(t)}$, and $\frac{I(t)}{N(t)}$ is the prevalence. Here, $S(t)$ and $I(t)$ represent respectively, the susceptible and infectious populations; $N(t)$ is the total population, β is the transmission rate.

Some examples of epidemic models which use the incidence term in Eq. (2b) are given in Eq. (3-5).

The Kermack-McKendrick Model

The classic Kermack-McKendrick SIR model was first developed by Kermack and McKendrick (1927), elaborated upon by by Anderson and May (1991), and now, forms the basis for many modern-day models of epidemics, (White and Enright, 2010).

The model assumes, inter alia, that the host population is constant, and that, the disease offers permanent immunity to those who recover from it, and rules out disease-induced deaths. The model also assumes that demographic turnover happens at a much slower time scale than the disease transmission, therefore, does not include births and deaths.

Table 1: Parameters for the Kermack-McKendrick Model

β	transmission rate
$\frac{\beta I(t)}{N(t)}$	force of infection
γ	recovery rate

The parameters for the Kermack-McKendrick Model are given in Table (1). The Transfer Diagram for the classic Kermack-McKendrick model is given in Fig. (1).

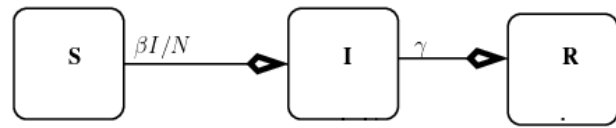


Fig. 1: A Transfer Diagram for the classic Kermack-McKendrick model

The classic Kermack-McKendrick model (Martchev, 2015; Brauer et al., 2019; Watmough, 2021; Teboh-Ewungkem and Akumah-Ngwa, 2021), is given in Eq. (3).

$$\begin{aligned} \frac{dS}{dt} &= -\frac{\beta I(t)}{N(t)}S(t), \\ \frac{dI}{dt} &= \frac{\beta I(t)}{N(t)}S(t) - \gamma I(t), \\ \frac{dR}{dt} &= \gamma I(t). \end{aligned} \quad (3)$$

where $S(t)$, $I(t)$ and $R(t)$ represents respectively, the susceptible, infected and infectious and the recovered (or removed) population at time t . The total population is $N(t) = S(t) + I(t) + R(t)$.

This model is also referred to as an SIR model without vital dynamics.

The SIR Model With Vital Dynamics

This model is used for an infectious disease which persists in a population for long periods of time, and therefore, births, deaths and migration must be taken into consideration. The model assume a natural death rate of μ so that $1/\mu$ is the life expectancy in each compartment. In addition to natural death, an infectious host may also die from the disease, at the rate of δ .

The SIR model provides a simple yet powerful abstraction of disease progression, in spite of its several limiting assumptions, such as, homogeneity of the population, as well as instantaneous disease transmission. In fact, the SIR model has become the quintessential framework, from which other models in mathematical epidemiology has emerged.

The parameters for the SIR model with Vital Dynamics are given in Table (2).

Table 2: Parameters for the SIR model

Λ	recruitment rate
β	transmission rate
$\frac{\beta I(t)}{N(t)}$	force of infection
γ	recovery rate
μ	natural death rate
δ	disease-induced death rate

These assumptions lead to the system of equations for the SIR Model with Vital Dynamics (Martchev, 2015; Brauer et al., 2019; Watmough, 2021; Teboh-Ewungkem and Akumah-Ngwa, 2021), given in Eq. (4).

$$\begin{aligned}\frac{dS}{dt} &= \Lambda - \frac{\beta I(t)}{N(t)} S(t) - \mu S(t), \\ \frac{dI}{dt} &= \frac{\beta I(t)}{N(t)} S(t) - (\gamma + \delta + \mu) I(t), \\ \frac{dR}{dt} &= \gamma I(t) - \mu R(t)\end{aligned}\quad (4)$$

The total population is $N(t) = S(t) + I(t) + R(t)$.

The SEIR Model With Vital Dynamics

An SEIR model is a variant of the SIR model, with an exposed class, E. The exposed (latent) class consists of individuals who have been infected, but not yet infectious. An infectious host may die from the disease or recover with immunity against re-infection.

The transfer diagram and parameters for the SEIR model are given in Fig. (3) and Table (3) respectively.

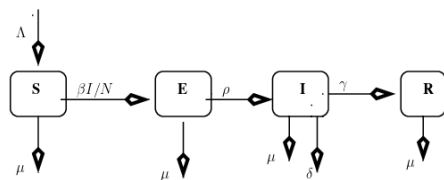


Fig. 3: A Transfer Diagram for an SEIR model

Table 3: Parameters for the SEIR model

Λ	recruitment rate
β	transmission rate
$\frac{\beta I(t)}{N(t)}$	force of infection
γ	recovery rate
μ	natural death rate
δ	disease-induced death rate
ρ	progression rate from exposed to infectious

These assumptions lead to the following system of equations for the SEIR epidemic model (Martchev, 2015; Brauer et al., 2019; Watmough, 2021; Teboh-Ewungkem and Akumah-Ngwa, 2021), in Eq. (5).

$$\begin{aligned}\frac{dS}{dt} &= \Lambda - \frac{\beta I(t)}{N(t)} S(t) - \mu S(t) \\ \frac{dE}{dt} &= \frac{\beta I(t)}{N(t)} S(t) - (\mu + \rho) E(t) \\ \frac{dI}{dt} &= \rho E(t) - (\mu + \gamma + \delta) I(t) \\ \frac{dR}{dt} &= \gamma I(t) - \mu R(t)\end{aligned}\quad (5)$$

where $N(t) = S(t) + E(t) + I(t) + R(t)$.

Comments on the Standard Incidence Term

In each of the epidemic models given in Eqs. (3-5), the standard incidence term is given as:

$$\frac{\beta I(t)}{N(t)} S(t) \quad (6)$$

where $\frac{\beta I(t)}{N(t)}$ is the force of infection, which is essentially, a proportion of $I(t)$. This characterization assumes implicitly, that all infectives, $I(t)$, transmit the disease equally to susceptibles.

Other examples of epidemic models which use this incidence terms are White and Enright (2010), Vargas-De-León (2011), Parsamanesh and Erfanian (2018) and Li and Guo (2024).

In Kiseleva et al. (2025), the authors pointed out that not all infectives contribute equally to disease-spread. According to them, some infective sub-populations may recover quickly or exhibit mild symptoms, while others remain infectious for extended periods, exerting disproportionate influence on transmission. They contend that such heterogeneity can arise from differences in immune response, spatial localization, presence or absence of preventive measures, and policy-driven isolation or exposure levels. Unfortunately, the authors ended up representing the incidence term as a proportions of $I(t)$.

Instead of representing incidence as a proportion of $I(t)$, we will first, classify the total infective population into two disjoint sub-population namely, those who have access to healthcare, and those who don't access to healthcare.

We posit, that the latter sub-population remain untreated and infectious, and thus contribute to the incidence through contacts with susceptibles. In the next section, we will quantify these two sub-populations of $I(t)$, in terms of a healthcare accessibility parameter, referring to availability and affordability, which we denote by κ .

Comments on the Recovery Term γI

In each of the epidemic models given in Eqs. (3), (4), (5), (White and Enright, 2010; Vargas-De-León, 2011; Parsamanesh and Erfanian, 2018), the recovery term is given as

$$\gamma I(t), \quad (7)$$

where γ is the per-capita recovery rate, and $I(t)$ is the total infective population.

This characterization of the recovery term implicitly, assumes that treatment is available to all infected individuals. In reality, recovery occurs only for those who have access to healthcare. In Appiah et al. (2024), the authors used the recovery term $\gamma \kappa I(t)$, where $\kappa I(t)$ ($0 \leq \kappa \leq 1$), represents the number (or proportion) of infectives who have access to healthcare. In their simulations, they demonstrated that increasing κ -values reduce the total infectious population.

Comments on the Disease-Induced Term δI

The disease-induced death terms in Eqs. (3-5) (White and Enright, 2010; Vargas-De-León, 2011; Parsamanesh and Erfanian, 2018) are given as:

$$\delta I(t), \quad (8)$$

where δ is the per-capita death rate, and $I(t)$ is the total infective population. Instead of the above representation, as a proportion of $I(t)$, we contend that disease-induced deaths occur only in the sub-population of $I(t)$ who don't have access to healthcare.

In each of the papers cited above, the incidence term, recovery term and disease-induced death term are all represented as proportions of $I(t)$. As we have pointed out above, recovery occurs only in the sub-population of $I(t)$, who have access to healthcare, while disease-induced deaths occur in the sub-population of $I(t)$, who don't have access to healthcare. In fact, recovery and disease-induced deaths occur at opposite halves of the infectious population spectrum.

In the next section, we will show from our classification, how the resulting incidence term, recovery term, disease-induced death term, as well as the basic reproduction number, expressed in terms of κ , give a profound insight into the role of healthcare accessibility, in effectively controlling the spread of infectious diseases.

A Novel Characterization of the Incidence Term

We consider a novel characterization of the incidence term, in terms of a healthcare accessibility parameter κ . This characterization, also results in corresponding characterizations of the recovery term, the disease-induced death term, as well as the basic reproduction number.

The total infective population $I(t)$, at time t , can be expressed as:

$$I(t) \equiv \kappa I(t) + (1 - \kappa)I(t), \quad 0 \leq \kappa \leq 1. \quad (9)$$

Remark 1

1. The first term, $\kappa I(t)$, on the right-hand side of Eq. (9) represents the sub-population of $I(t)$ who have access to healthcare.
2. The second term, $(1 - \kappa)I(t)$, represents the sub-population of $I(t)$, who don't have access to healthcare and therefore, remain untreated and infectious.

This classification is crucial, as it characterizes the incidence term, the associated recovery term, disease-induced death term, as well as the basic reproduction number, in terms of the healthcare accessibility parameter, κ .

Proposition 1 (A Novel Standard Incidence Term)

Let $I(t)$ be the total number (or proportion) of infectives in a population at time t , and let $\kappa I(t)$ ($0 \leq \kappa \leq 1$) be the number (or proportion) of infectives with access to healthcare. Then,

1. the proposed standard incidence term is given by:

$$\lambda_{\kappa}(t)S(t) = \frac{\beta(1 - \kappa)I(t)}{N(t)}S(t), \quad (10)$$

where,

1. $\lambda_{\kappa}(t) = \frac{\beta(1 - \kappa)I(t)}{N(t)}$, is the force of infection,
2. $S(t)$ is the susceptible population.

2. the recovery term, (for infectives with access to healthcare, at a per-capita recovery rate of γ), is

$$\gamma \kappa I(t), \quad (11)$$

3. the disease-induced death term, (for infectives without access to healthcare, at a per-capita death rate of δ), is given by:

$$\delta_{\kappa}I(t) \triangleq \delta(1 - \kappa)I(t). \quad (12)$$

Proof

1. From the second term on the right-hand side of (9), and the second part of Remark (1), the prevalence of the disease is

$$\frac{(1 - \kappa)I(t)}{N(t)}, \quad (13a)$$

with corresponding force of infection given by

$$\lambda_{\kappa}(t) = \frac{\beta(1 - \kappa)I(t)}{N(t)}, \quad (13b)$$

where β is the transmission rate.

The proposed novel standard incidence term is, therefore, given by:

$$\lambda_{\kappa}(t)S(t) = \frac{\beta(1 - \kappa)I(t)}{N(t)}S(t),$$

as stated in Eq. (10).

2. By hypothesis, the first term on the right hand side of (9), $\kappa I(t)$, represents the sub-population of $I(t)$ who have access to healthcare. So, at a per-capita recovery rate of γ , the recovery term is given by

$$\gamma \kappa I(t) = \gamma \kappa I(t).$$

3. Disease-induced death occur only in the sub-population $(1 - \kappa)I(t)$. Hence, at a per-capita disease-induced death rate of δ , the disease-induced death term is given by:

$$\delta_{\kappa}I(t) \triangleq \delta(1 - \kappa)I(t).$$

Note that δ_{κ} satisfies the following,

$$\begin{cases} \delta_{\kappa} = \delta, & \text{when } \kappa = 0 \\ \delta_{\kappa} < \delta, & \text{when } 0 < \kappa < 1 \\ \delta_{\kappa} = 0, & \text{when } \kappa = 1. \end{cases}$$

Remark 2

Proposition (1) as well as its proof, in the case of bilinear incidence is similar.

Corollary 1

1. The proposed new standard incidence term (10), is a decreasing function of κ , which implies that increasing κ reduces new cases.
2. the recovery term (11), is an increasing function of κ , which means that increasing κ speeds up recovery.
3. the disease-induced death term (12) is a decreasing function of κ , that is, increasing κ reduces disease-induced deaths.

The message from Corollary (1) is clear. That is, increasing healthcare accessibility reduces new infection cases, speeds up recovery, and reduces disease-induced deaths.

Remark 3

We have demonstrated in Proposition (1) that:

1. incidence and disease-induced deaths on the one hand, occur in the sub-population $(1 - \kappa)I(t)$, of infectives who don't have access to healthcare.
2. recovery on the other hand, occurs in the opposite sub-population $\kappa I(t)$, of infectives who have access to healthcare.

Instead of representing all three categories, viz. incidence, recovery and disease-induced deaths as proportions of $I(t)$, we instead, represent them in terms of the classification given in Eq. (9). This novel representation brings to the fore, the all-important role of healthcare accessibility in controlling the spread of infectious diseases.

Furthermore, in Theorem (1), we state and prove the main attributes of the basic reproduction number resulting from this new characterization.

Theorem 1

Let $\mathcal{R}_0^{(\kappa)}$ be the basic reproduction number of the system resulting from Proposition (1). Then,

1. $\mathcal{R}_0^{(\kappa)}$ is a strictly decreasing function of κ .
2. $\mathcal{R}_0^{(1)} \leq \mathcal{R}_0^{(\kappa)} \leq \mathcal{R}_0^{(0)}$, for $0 \leq \kappa \leq 1$,

where $\mathcal{R}_0^{(0)}$ and $\mathcal{R}_0^{(1)}$ are obtained from $\mathcal{R}_0^{(\kappa)}$, with $\kappa = 0$, and $\kappa = 1$, respectively.

Proof

1. Compute the basic reproduction number $\mathcal{R}_0^{(\kappa)}$, using the next-generation matrix approach.

Express $\mathcal{R}_0^{(\kappa)}$ as $\frac{A(1 - \kappa)}{B(\gamma\kappa + \delta_\kappa + \mu)}$, where the factors A , $B > 0$, and independent of κ . Then,

$$\begin{aligned} \frac{d}{d\kappa} [\mathcal{R}_0^{(\kappa)}] &= \frac{d}{d\kappa} \left[\frac{A(1 - \kappa)}{B(\gamma\kappa + \delta_\kappa + \mu)} \right] \\ &= \frac{A}{B} \frac{d}{d\kappa} \left[\frac{(1 - \kappa)}{(\gamma\kappa + \delta_\kappa + \mu)} \right] \\ &= \frac{A}{B} \left[\frac{-(\gamma + \mu)}{(\gamma\kappa + \delta_\kappa + \mu)^2} \right] \\ &= - \left[\frac{A(\gamma + \mu)}{B(\gamma\kappa + \delta_\kappa + \mu)^2} \right] < 0. \end{aligned}$$

This proves that $\mathcal{R}_0^{(\kappa)}$ is a strictly decreasing function of κ .

2. Hence, the maximum and minimum values of $\mathcal{R}_0^{(\kappa)}$ denoted respectively, by $\mathcal{R}_0^{(0)}$ and $\mathcal{R}_0^{(1)}$, on the interval $[0, 1]$. occur at $\kappa = 0$, and $\kappa = 1$, respectively. Therefore,

$$\mathcal{R}_0^{(1)} \leq \mathcal{R}_0^{(\kappa)} \leq \mathcal{R}_0^{(0)}, \quad 0 \leq \kappa \leq 1. \quad (14)$$

Remark 4

Observations from Theorem (1) show that:

1. when the accessibility parameter $\kappa = 0$, (implying, that a typical infective has no access to healthcare), the basic reproduction number attains its maximum possible value, with obvious implications for the effective control of the disease.
2. on the other hand, when $\kappa = 1$, (implying, that a typical infective has access to healthcare), the corresponding basic reproduction number has its minimum possible value.
3. $\mathcal{R}_0^{(\kappa)}$ being a strictly decreasing function of κ implies, increasing κ reduces the size of the basic reproduction number, and as a result, makes it easier to control, and possibly, eradicate an infectious disease.

In the next three sections, we apply Proposition (1) to a selection of typical epidemic models, compute the basic reproduction number, and verify Theorem (1) in each case.

Modified Kermack-McKendrick Model Based on Proposition (1)

The classic Kermack-McKendrick model given in Eq. (3), excludes disease-induced deaths, in spite of the fact that fatalities do occur when infectives do not have access to healthcare and effective treatment.

We re-formulate the classic Kermack-McKendrick model using:

1. the proposed standard incidence term $\frac{\beta(1 - \kappa)I(t)}{N(t)}S(t)$,
2. the recovery term $\gamma\kappa I(t)$, and
3. the disease-induced death term $\delta_\kappa I(t) \triangleq \delta(1 - \kappa)I(t)$,

from Proposition (1). The Transfer Diagram is given in Fig. (4).

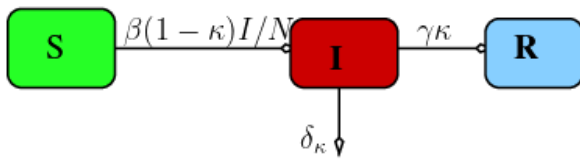


Fig. 4: A Transfer Diagram for the modified Kermack-McKendrick model

The parameters for the modified Kermack-McKendrick Model are given in Table (4).

Table 4: Parameters for the modified Kermack-McKendrick Model

β	transmission rate
$0 \leq \kappa \leq 1$	a constant
$\frac{\beta(1-\kappa)I(t)}{N(t)}$	force of infection
$\gamma\kappa$	recovery rate
δ_κ	disease-induced death rate

From the foregoing, we obtain the following modified Kermack-McKendrick model,

$$\begin{aligned} \frac{dS}{dt} &= -\frac{\beta(1-\kappa)I(t)}{N(t)}S(t), \\ \frac{dI}{dt} &= \frac{\beta(1-\kappa)I(t)}{N(t)}S(t) - (\gamma\kappa + \delta_\kappa)I(t), \\ \frac{dR}{dt} &= \gamma\kappa I(t), \end{aligned} \quad (15)$$

where $S(t)$, $I(t)$ and $R(t)$ represents respectively, the susceptible, infectious and the recovered (or removed) population at time t . The total population is $N(t) = S(t) + I(t) + R(t)$.

We compute the basic reproduction number using the next-generation matrix approach developed by Diekmann et al. (1990) and van der Driessche and Watmough (2002). The next generation matrix G , is given by:

$$G = FV^{-1}$$

where

$$F = \left[\frac{\partial \mathcal{F}_i}{\partial x_j}(E^0) \right], \quad V = \left[\frac{\partial \mathcal{V}_i}{\partial x_j}(E^0) \right],$$

and E^0 denotes the disease-free equilibrium, $\mathcal{F}_i$ is the rate of appearance of new infections in compartment i , and $\mathcal{V}_i$ is the rate of transfer of infections from compartment i .

The model in (15) has one disease class namely, $I(t)$, and so with $S(t) = N(t)$, at time $t = 0$ we obtain:

$$\begin{aligned} \mathcal{F}_1 &= \frac{\beta(1-\kappa)IS}{N} \Rightarrow F = \frac{\beta(1-\kappa)S}{N} = \beta(1-\kappa), \\ \mathcal{V}_1 &= (\gamma\kappa + \delta_\kappa)I \Rightarrow V = (\gamma\kappa + \delta_\kappa) \Rightarrow V^{-1} = \frac{1}{\gamma\kappa + \delta_\kappa} \\ \mathcal{R}_0^{(\kappa)} &= FV^{-1} = \frac{\beta(1-\kappa)}{\gamma\kappa + \delta_\kappa} \end{aligned} \quad (16)$$

Verification of Theorem (1)

1. It can be verified that

$$\frac{d}{d\kappa} \left[\frac{\beta(1-\kappa)}{\gamma\kappa + \delta_\kappa} \right] = \frac{-\beta\gamma}{(\gamma\kappa + \delta_\kappa)^2} < 0,$$

which confirms Part 1 of Theorem (1).

2. For Part 2 of Theorem (1), we obtain:

$$0 = \mathcal{R}_0^{(1)} \leq \mathcal{R}_0^{(\kappa)} \leq \mathcal{R}_0^{(0)} = \frac{\beta}{\delta} \quad (17)$$

An SIR Epidemic Model With Vital Dynamics, Based on Proposition (1)

A re-formulation of the SIR model given in Eq. (2), using the standard incidence term, recovery term and disease-induced death term in Eqs. (10), (11) and (12) respectively, from Proposition (1).

The transfer diagram and parameters for the SIR model are given in Fig. (5) and Table (5) respectively.

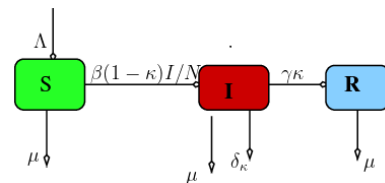


Fig. 5: A Transfer Diagram for the SIR model based on Proposition (1)

Table 5: Parameters for the SIR model based on Proposition (1)

Λ	recruitment rate
μ	natural death rate
β	transmission rate
$0 \leq \kappa \leq 1$	a constant
$\frac{\beta(1-\kappa)I(t)}{N(t)}$	force of infection
$\gamma\kappa$	recovery rate
δ_κ	disease-induced death rate

The dynamics of the SIR model vital dynamics, using Proposition (1), is given by:

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \frac{\beta(1-\kappa)I(t)}{N(t)}S(t) - \mu S(t), \\ \frac{dI}{dt} &= \frac{\beta(1-\kappa)I(t)}{N(t)}S(t) - (\gamma\kappa + \delta_\kappa + \mu)I(t), \\ \frac{dR}{dt} &= \gamma\kappa I(t) - \mu R(t). \end{aligned} \quad (18)$$

The total population is $N(t) = S(t) + I(t) + R(t)$.

Dividing equation (18) through by the carrying capacity, Λ/μ , and putting $x_1 = \frac{S}{\Lambda/\mu}$, $x_2 = \frac{I}{\Lambda/\mu}$, $x_3 = \frac{R}{\Lambda/\mu}$, gives the equivalent system:

$$\begin{aligned} \frac{dx_1}{dt} &= \mu - \beta(1-\kappa)x_2x_1 - \mu x_1, \\ \frac{dx_2}{dt} &= \beta(1-\kappa)x_2x_1 - \gamma\kappa x_2 - \delta_\kappa x_2 - \mu x_2, \\ \frac{dx_3}{dt} &= \gamma\kappa x_2 - \mu x_3, \end{aligned} \quad (19)$$

where $x_1 + x_2 + x_3 = 1$.

The system in (19) has a unique disease-free equilibrium point given by:

$$E^0 = [x_1^0 = 1, \quad x_2^0 = 0, \quad x_3^0 = 0],$$

and an endemic equilibrium point given by:

$$E^* = \left[x_1^* = \frac{\gamma\kappa + \delta_\kappa + \mu}{\beta(1 - \kappa)}, \right. \\ x_2^* = \frac{\mu(\beta\kappa + \gamma\kappa - \beta\kappa + \delta_\kappa + \mu)}{\beta(\kappa - 1)(\gamma\kappa + \delta_\kappa + \mu)} \\ \left. x_3^* = \frac{\gamma\kappa(\beta\kappa + \gamma\kappa - \beta + \delta_\kappa + \mu)}{\beta(\kappa - 1)(\gamma\kappa + \delta_\kappa + \mu)} \right].$$

The basic reproduction number, using the next-generation matrix approach developed by Diekmann et al. (1990) and van der Driessche and Watmough (2002), is given by:

$$G = FV^{-1}$$

where

$$F = \left[\frac{\partial \mathcal{F}_i}{\partial x_j}(E^0) \right], \quad V = \left[\frac{\partial \mathcal{V}_i}{\partial x_j}(E^0) \right],$$

and E^0 denotes the disease-free equilibrium, $\mathcal{F}_i$ is the rate of appearance of new infections in compartment i , and $\mathcal{V}_i$ is the rate of transfer of infections from compartment i .

We denote the basic reproduction number by

$$\mathcal{R}_0^{(\kappa)} = \rho(G) = \rho(FV^{-1}) \quad (20)$$

The model in (19) has one disease class namely, x_2 , and so,

$$\mathcal{F}_1 = \beta(1 - \kappa)x_2x_1 \Rightarrow F = \beta(1 - \kappa)x_1 = \beta(1 - \kappa), (x_1^0 = 1).$$

$$\mathcal{V}_1 = (\gamma\kappa + \delta_\kappa + \mu)x_2 \Rightarrow V = (\gamma\kappa + \delta_\kappa + \mu),$$

$$\text{and so } V^{-1} = \frac{1}{\gamma\kappa + \delta_\kappa + \mu}.$$

Therefore,

$$\mathcal{R}_0^{(\kappa)} = FV^{-1} = \frac{\beta(1 - \kappa)}{\gamma\kappa + \delta_\kappa + \mu} \quad (21)$$

Verification of Theorem (1)

1. It can be verified that

$$\frac{d}{d\kappa} \left[\frac{\beta(1 - \kappa)}{\gamma\kappa + \delta_\kappa + \mu} \right] = -\frac{\beta(\mu + \gamma)}{(\gamma\kappa + \delta_\kappa + \mu)^2} < 0,$$

which confirms Part 1 of Theorem (1).

2. Part 2 of Theorem (1) becomes,

$$0 = \mathcal{R}_0^{(1)} \leq \mathcal{R}_0^{(0)} \leq \mathcal{R}_0^{(0)} = \frac{\beta}{\mu + \delta} \quad (22)$$

We also note that $\kappa = 0$, means that a typical infective does not have access to healthcare and

treatment. So, from (22) we see that if they survive disease-induced death, (or, if the disease is non-fatal ($\delta = 0$)) then, they will live with the infection throughout their life.

In terms of $\mathcal{R}_0^{(\kappa)}$, the endemic equilibrium point can be expressed as:

$$E^* = \left[x_1^* = \frac{1}{\mathcal{R}_0^{(\kappa)}} \right. \\ x_2^* = \frac{\mu}{\gamma\kappa + \delta_\kappa + \mu} \left[1 - \frac{1}{\mathcal{R}_0^{(\kappa)}} \right], \\ \left. x_3^* = \frac{\gamma\kappa}{\gamma\kappa + \delta_\kappa + \mu} \left[1 - \frac{1}{\mathcal{R}_0^{(\kappa)}} \right] \right]$$

An SEIR Epidemic Model Based on Proposition (1)

We formulated an SEIR epidemic model given in Eq. (5), using Eqs. (10), (11), and (12) from Proposition (1). The Transfer Diagram is given in Fig. (6). The parameters of the model are given in Table (6).

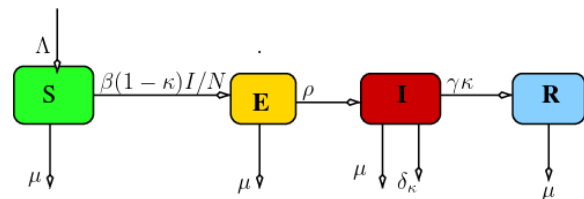


Fig. 6: A Transfer Diagram for an SEIR model based on Proposition (1)

Table 6: Parameters for the SEIR model based on Proposition (1)

Λ	the recruitment rate
β	the transmission rate
μ	the natural death rate
$0 \leq \kappa \leq 1$	a constant
$\frac{\beta(1-\kappa)I(t)}{N(t)}$	force of infection
$\gamma\kappa$	recovery rate
δ_κ	disease-induced death rate
ρ	progression from exposed to infectious

The dynamics of an SEIR epidemic model, using Proposition (1), is given in (23).

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \frac{\beta(1 - \kappa)I(t)}{N(t)}S(t) - \mu S(t) \\ \frac{dE}{dt} &= \frac{\beta(1 - \kappa)I(t)}{N(t)}S(t) - (\mu + \rho)E(t) \\ \frac{dI}{dt} &= \rho E(t) - (\gamma\kappa + \delta_\kappa + \mu)I(t) \\ \frac{dR}{dt} &= \gamma\kappa I(t) - \mu R(t) \end{aligned} \quad (23)$$

where $N(t) = S(t) + E(t) + I(t) + R(t)$.

Dividing through each differential equation by the carrying capacity Λ/μ , and setting $x_1 = \frac{S}{\Lambda/\mu}$, $x_2 = \frac{E}{\Lambda/\mu}$, $x_3 = \frac{I}{\Lambda/\mu}$, $x_4 = \frac{R}{\Lambda/\mu}$, transforms the system (23) into the equivalent system:

$$\begin{aligned}\frac{dx_1}{dt} &= \mu - (1 - \kappa)x_3x_1 - \mu x_1 \\ \frac{dx_2}{dt} &= (1 - \kappa)x_3x_1 - (\mu + \rho)x_2 \\ \frac{dx_3}{dt} &= \rho x_2 - (\gamma\kappa + \delta_\kappa + \mu)x_3 \\ \frac{dx_4}{dt} &= \gamma\kappa x_3 - \mu x_4\end{aligned}\quad (24)$$

where $x_1 + x_2 + x_3 + x_4 = 1$.

The system (24) has a unique disease-free and endemic equilibrium points. The disease-free equilibrium is given by:

$$E^0 = [x_1^0 = 1, x_2^0 = 0, x_3^0 = 0, x_4^0 = 0]$$

The Basic Reproduction Number

The SEIR model has two disease states, $x_2(t)$ and $x_3(t)$. We use the next-generation approach to compute the basic reproduction number $\mathcal{R}_0^{(\kappa)}$

The vectors $\mathcal{F}_i$ and $\mathcal{V}_i$, $i = 1, 2$, are given respectively by

$$\mathcal{F} = \begin{bmatrix} \beta(1 - \kappa)x_3x_1 \\ 0 \\ (\mu + \rho)x_2(t) \\ -\rho x_2(t) + (\gamma\kappa + \delta_\kappa + \mu)x_3(t) \end{bmatrix}, \quad \mathcal{V} =$$

The matrices F and V are given respectively by

$$F = \begin{bmatrix} 0 & \beta(1 - \kappa) \\ 0 & 0 \end{bmatrix}, \text{ when } x_1^0 = 1.$$

Now the matrices F and V are given respectively by

$$F = \begin{bmatrix} 0 & \beta(1 - \kappa) \\ 0 & 0 \end{bmatrix}, \text{ when } x_1^0 = 1.$$

Now

$$V = \begin{bmatrix} (\mu + \rho) & 0 \\ -\rho & (\gamma\kappa + \delta_\kappa + \mu) \end{bmatrix},$$

and so

$$V^{-1} = \begin{bmatrix} \frac{1}{\mu + \rho} & 0 \\ \frac{\rho}{(\mu + \rho)(\gamma\kappa + \delta_\kappa + \mu)} & \frac{1}{\gamma\kappa + \delta_\kappa + \mu} \end{bmatrix}$$

The matrix G is now given by:

$$G = FV^{-1} = \begin{bmatrix} \frac{\rho\beta}{(\mu + \rho)(\gamma\kappa + \delta_\kappa + \mu)} & \frac{\beta(1 - \kappa)}{(\gamma\kappa + \delta_\kappa + \mu)} \\ 0 & 0 \end{bmatrix}$$

The resulting basic reproduction number, which we denote by $\mathcal{R}_0^{(\kappa)}$, is given by:

$$\mathcal{R}_0^{(\kappa)} = \frac{\rho\beta}{(\mu + \rho)} \frac{(1 - \kappa)}{(\gamma\kappa + \delta_\kappa + \mu)}. \quad (25)$$

Verification of Theorem (1)

1. It can be verified that

$$\frac{d}{d\kappa} \left[\frac{\rho\beta}{(\mu + \rho)} \frac{(1 - \kappa)}{(\gamma\kappa + \delta_\kappa + \mu)} \right] = -\frac{\rho\beta}{(\mu + \rho)} \frac{(\mu + \gamma)}{(\gamma\kappa + \delta_\kappa + \mu)^2} < 0,$$

which confirms Part 1 of Theorem (1).

2. Part 2 of Theorem (1) becomes,

$$0 = \mathcal{R}_0^{(1)} \leq \mathcal{R}_0^{(\kappa)} \leq \mathcal{R}_0^{(0)} = \frac{\rho}{(\mu + \rho)} \frac{\beta}{(\mu + \delta)} \quad (26)$$

We also note that $\kappa = 0$, means that a typical infective does not have access to healthcare and treatment. So, from (26) we see that if they survive disease-induced death, (or, if the disease is non-fatal ($\delta = 0$)) then, they will live with the infection throughout their life.

In terms of $\mathcal{R}_0^{(\kappa)}$, the endemic equilibrium point can be expressed as:

$$\begin{aligned}E^* &= \left[x_1^* = \frac{1}{\mathcal{R}_0^{(\kappa)}}, x_2^* = \frac{\mu}{(\mu + \rho)} \left[1 - \frac{1}{\mathcal{R}_0^{(\kappa)}} \right], \right. \\ x_3^* &= A \left[1 - \frac{1}{\mathcal{R}_0^{(\kappa)}} \right], x_4^* = B \left[1 - \frac{1}{\mathcal{R}_0^{(\kappa)}} \right] \end{aligned},$$

where

$$\begin{aligned}A &= \frac{\mu\rho}{(\mu + \rho)(\gamma\kappa + \mu + \delta_\kappa)}, \\ B &= \frac{\gamma\kappa\rho}{(\mu + \rho)(\gamma\kappa + \mu + \delta_\kappa)}\end{aligned}$$

A Vector-Host Epidemic Model, Based on Proposition (1)

We consider an SIRS-SI Vector-host epidemic model (Martchev, 2015; Brauer et al., 2019; Watmough, 2021; Teboh-Ewungkem and Akumah-Ngwa, 2021; Appiah et al., 2024), with an SIRS epidemic model for the host population, S_h, I_h, R_h denoting respectively, susceptible, infected and recovered hosts (with partial immunity). An SI model is used for vector population, with S_v and I_v denoting respectively, the susceptible and infected vectors. It is assumed that infected vectors remain infected for life, and that the infection is not harmful to the vectors.

We note that for a vector-host epidemic model, there are two incidence terms namely,

- incidence from infective vectors to susceptible hosts, and

- incidence from untreated infective hosts to susceptible vectors

From Proposition (1), the two incidence terms, the recovery term and the disease-induced death term, for a Vector-host epidemic model, are given by:

$$\frac{\beta_h I_v(t)}{N_v(t)} S_h(t), \quad (27a)$$

$$\frac{\beta_v(1-\kappa)I_h(t)}{N_h(t)} S_v(t), \quad (27b)$$

$$\gamma\kappa I_h(t); \quad (28)$$

$$\delta_\kappa I_h(t) \triangleq \delta(1-\kappa)I_h(t). \quad (29)$$

The parameters for the model are given in Table (7).

Table 7: Parameters for the Vector-Host Model based on Proposition (1)

β_h	transmission rate from vectors to hosts
β_v	transmission rate from hosts to vectors
$\frac{\beta_h I_v(t)}{N_v(t)}$	force of infection from vectors to hosts
$0 \leq \kappa \leq 1$	a constant
$\frac{\beta_v(1-\kappa)I_h(t)}{N_h(t)}$	force of infection from hosts to vectors
Λ_h	hosts recruitment rate
Λ_v	vectors recruitment rate
$\gamma\kappa$	recovery rate
δ_κ	disease-induced death rate
μ_h	natural death rate for hosts
μ_v	natural death rate for vectors
α	prevention rate
ω	per-capita loss of immunity

The Transfer Diagram is shown in Fig. (7).

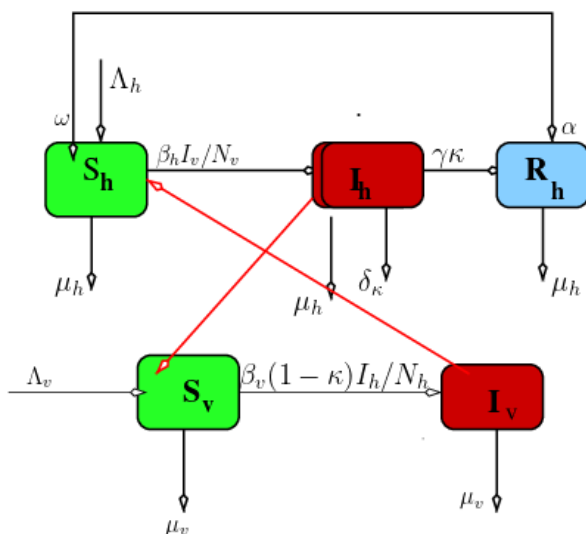


Fig. 7: A Transfer Diagram for SIRS-SI Vector-Host Model, based on Proposition (1)

The dynamics of the resulting Vector-host epidemic model, based on Proposition (1), are given in Eq. (28).

$$\begin{aligned} \dot{S}_h &= \Lambda_h - \frac{\beta_h I_v(t)}{N_v(t)} S_h(t) - (\mu_h + \alpha) S_h(t) + \omega R_h(t) \\ \dot{I}_h &= \frac{\beta_h I_v(t)}{N_v(t)} S_h(t) - (\gamma\kappa + \delta_\kappa + \mu_h) I_h(t) \\ \dot{R}_h &= \gamma\kappa I_h(t) + \alpha S_h(t) - (\mu_h + \omega) R_h(t) \\ \dot{S}_v &= \Lambda_v - \frac{\beta_v(1-\kappa)I_h(t)}{N_h(t)} S_v(t) - \mu_v S_v(t) \\ \dot{I}_v &= \frac{\beta_v(1-\kappa)I_h(t)}{N_h(t)} S_v(t) - \mu_v I_v(t) \end{aligned} \quad (28)$$

where

$$S_h(t) + I_h(t) + R_h(t) = N_h(t), \quad \text{and} \quad S_v(t) + I_v(t) = N_v(t).$$

Dividing through the first three equations in (28) by the carrying capacity for the host population, Λ_h/μ_h , and the last two equations by the carrying capacity for the vector population, Λ_v/μ_v , and setting

$$\begin{aligned} x_1 &= \frac{S_h}{\Lambda_h/\mu_h}, \\ x_2 &= \frac{I_h}{\Lambda_h/\mu_h}, \\ x_3 &= \frac{R_h}{\Lambda_h/\mu_h}, \\ x_4 &= \frac{S_v}{\Lambda_v/\mu_v}, \\ x_5 &= \frac{I_v}{\Lambda_v/\mu_v} \end{aligned}$$

gives the equivalent system:

$$\begin{aligned} \dot{x}_1 &= \mu_h - \beta_h x_5 x_1 - (\mu_h + \alpha) x_1 + \omega x_3 \\ \dot{x}_2 &= \beta_h x_5 x_1 - (\gamma\kappa + \delta_\kappa + \mu_h) x_2 \\ \dot{x}_3 &= \gamma\kappa x_2 + \alpha x_1 - (\mu_h + \omega) x_3 \\ \dot{x}_4 &= \mu_v - \beta_v(1-\kappa) x_2 x_4 - \mu_v x_4 \\ \dot{x}_5 &= \beta_v(1-\kappa) x_2 x_4 - \mu_v x_5 \end{aligned} \quad (29)$$

where $x_1 + x_2 + x_3 = 1$, and $x_4 + x_5 = 1$.

The system (29) has unique disease-free and endemic equilibrium points. The disease-free equilibrium is given by:

$$\begin{aligned} E^0 &= \left[x_1^0 = \frac{\mu_h + \alpha}{\mu_h + \alpha + \omega}, \right. \\ &x_2^0 = 0, \\ &x_3^0 = \frac{\omega}{\mu_h + \alpha + \omega}, \\ &\left. x_4^0 = 1, x_5^0 = 0 \right] \end{aligned}$$

The Basic Reproduction Number

The model in (29) has two disease states, $x_2(t)$ and $x_5(t)$. We use the next-generation approach to compute

the basic reproduction number.

The vectors $\mathcal{F}_i$ and $\mathcal{V}_i$, $i = 1, 2$ are given respectively by

$$\mathcal{F} = [\beta_h x_5 x_1 \quad \beta_v (1 - \kappa) x_2 x_4]^T, \quad \mathcal{V} = [(\mu_h + \gamma\kappa + \delta_\kappa) x_2 \quad \mu_v x_5]^T$$

The matrices F and V are given respectively by

$$F = \begin{bmatrix} 0 & \beta_h x_1 \\ \beta_v (1 - \kappa) x_4 & 0 \end{bmatrix}, \text{ substituting for } x_1^0, x_4^0$$

gives

$$F = \begin{bmatrix} 0 & \frac{\beta_h(\mu_h + \alpha)}{\mu_h + \alpha + \omega} \\ \beta_v(1 - \kappa) & 0 \end{bmatrix}$$

$$V = \begin{bmatrix} (\gamma\kappa + \delta_\kappa + \mu_h) & 0 \\ 0 & \mu_v \end{bmatrix}, \quad V^{-1} = \begin{bmatrix} \frac{1}{(\gamma\kappa + \delta_\kappa + \mu_h)} & 0 \\ 0 & \frac{1}{\mu_v} \end{bmatrix}.$$

The matrix G is given by:

$$G = FV^{-1} = \begin{bmatrix} 0 & \frac{\beta_h(\mu_h + \alpha)}{(\alpha + \omega + \mu_h)\mu_v} \\ \frac{\beta_v(1 - \kappa)}{(\gamma\kappa + \delta_\kappa + \mu_h)} & 0 \end{bmatrix}$$

The resulting basic reproduction number is given by:

$$\mathcal{R}_0^{(\kappa)} = \sqrt{\frac{\beta_h \beta_v (\mu_h + \omega) (1 - \kappa)}{\mu_v (\alpha + \omega + \mu_h) (\gamma\kappa + \delta_\kappa + \mu_h)}}. \quad (30)$$

and

$$[\mathcal{R}_0^{(\kappa)}]^2 = \frac{\beta_h \beta_v (\mu_h + \omega) (1 - \kappa)}{\mu_v (\alpha + \omega + \mu_h) (\gamma\kappa + \delta_\kappa + \mu_h)}$$

Verification of Theorem (1)

1. It can be verified that

$$\frac{d}{d\kappa} \left[[\mathcal{R}_0^{(\kappa)}]^2 \right] = - \frac{\beta_h \beta_v (\mu_h + \omega) (\gamma + \mu_h)}{\mu_v (\alpha + \omega + \mu_h) (\gamma\kappa + \delta_\kappa + \mu_h)^2} < 0,$$

which confirms Part 1 of Theorem (1).

2. Part 2 of Theorem (1) becomes,

$$0 = [\mathcal{R}_0^{(1)}]^2 \leq [\mathcal{R}_0^{(\kappa)}]^2 \leq [\mathcal{R}_0^{(0)}]^2 = \frac{\beta_h \beta_v (\mu_h + \omega)}{\mu_v (\alpha + \omega + \mu_h) (\delta + \mu_h)} \quad (31)$$

The endemic equilibrium point E^* , in terms of the basic reproduction number $[\mathcal{R}_0^{(\kappa)}]^2$, is given by:

$$E^* = [x_1^*, x_2^*, x_3^*, x_4^*, x_5^*]$$

where

$$x_1^* = \frac{C_4}{C_1 \beta_h + C_2} \left[1 + \left(\frac{1}{\mathcal{R}_0^{(\kappa)}} \right)^2 C_3 \right],$$

$$x_2^* = \frac{C_5}{C_1 \beta_h + C_2} \left[1 - \left(\frac{1}{\mathcal{R}_0^{(\kappa)}} \right)^2 \right],$$

$$x_3^* = \frac{1}{C_1 \beta_h + C_2} \left[C_7 + \left[\left(\frac{1}{\mathcal{R}_0^{(\kappa)}} \right)^2 \right] C_8 C_9 \right],$$

$$x_4^* = \frac{(1 + C_3) \left(\frac{1}{\mathcal{R}_0^{(\kappa)}} \right)^2}{C_6}$$

$$x_5^* = \frac{1}{C_6} \left[1 - \left(\frac{1}{\mathcal{R}_0^{(\kappa)}} \right)^2 \right]$$

where

$$C_1 = (\mu_h^2 + (\gamma\kappa + \omega + \delta_\kappa) \mu_h + \omega \delta_\kappa)$$

$$C_2 = \mu_h (\alpha + \omega + \mu_h) (\gamma\kappa + \delta_\kappa + \mu_h)$$

$$C_3 = \frac{\mu_h \beta_h C_1}{C_2}$$

$$C_4 = \mu_h (\gamma\kappa + \delta_\kappa + \mu_h) (\omega + \mu_h)$$

$$C_5 = \mu_h \beta_h (\mu_h + \omega)$$

$$C_6 = \left[1 + \left(\frac{1}{\mathcal{R}_0^{(\kappa)}} \right)^2 C_3 \right]$$

$$C_7 = \mu_h (\alpha (\gamma\kappa + \delta_\kappa + \mu_h) + \gamma\kappa \beta_h)$$

$$C_8 = \frac{\beta_h (\mu_h + \omega)}{(\alpha + \omega + \mu_h)}$$

$$C_9 = (\gamma\kappa \mu_h + \alpha \delta_\kappa + \alpha \mu_h)$$

Simulations With Modified Kenmark-McKendrick & Vector-Host Models

Our simulations were performed on the modified Kenmark-McKendrick model given in Eq. (15), and the Vector-host model given in Eq. (28). All computations and plots were obtained using Maple (Version 2025) from Maplesoft.

Simulations of the Modified Kermack-McKendrick Model in Eq. (15)

The data given in Table (8) was taken from Influenza in a Boarding School, (Anonymous, 1978). This dataset is a classic example used in numerous Mathematical Biology Textbooks and Lectures. A plot of the time-series data is shown in Fig. (8).

In January 1978, 763 students returned to a boarding school after Mid-term break. One student was diagnosed with a flu, followed by two others the next day. by the

end of the month nearly half of the boarding school's population were sick with flu. Most of the students had been affected by the time the epidemic was over. A swab taken from some of the boys revealed that they were infected with *H1N1* influenza A virus.

Table 8: Daily number of influenza-infected boys

Day	1	2	3	4	5	6	7	8	9	10	11	12	13	14
No. Infected	3	6	25	84	222	294	258	237	191	129	72	45	35	22

We performed a least-squares fit of the data in Table (8) to our modified Kermack-McKendrick model given in (15). The parameter-values obtained are $\beta = 3.3$, $\kappa = 0.4$, $\gamma = 1.2$, and $\delta = 0.000018$. The plot is displayed in Fig. (9).

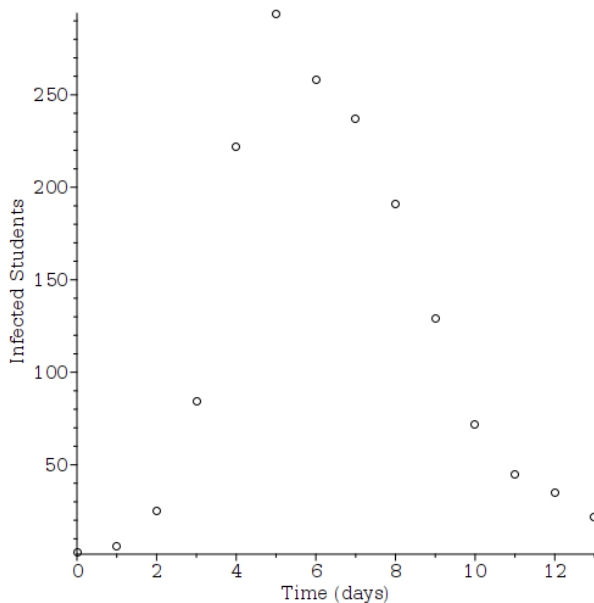


Fig. 8: Plot of the Time-Series Data from Table (8)

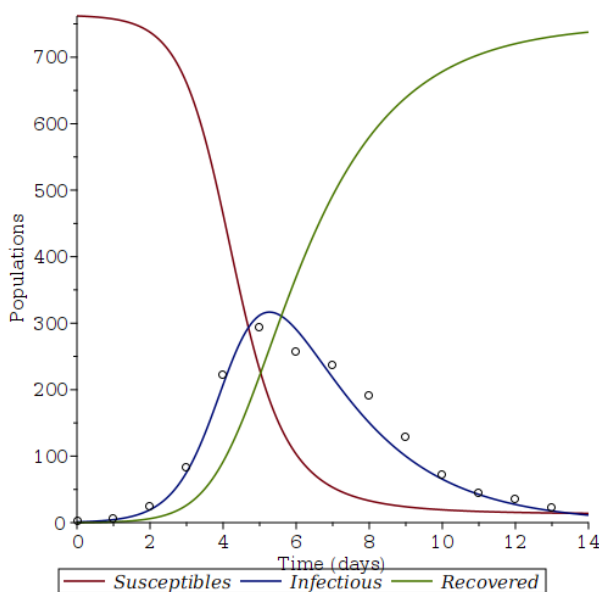


Fig. 9: Modified Kermack-McKendrick Data Fitting Plot

Simulations of the Vector-Host Model in Section (6)

Solutions to the System in (28) Plots of solutions to the system in (28) with initial conditions given in Table (9), and parameter values given in Table (10) are shown in Figs. (10-12).

Table 9: Initial Conditions for the System in (6)

$S_h(0)$	27500000
$I_h(0)$	500000
$R_h(0)$	0
$S_v(0)$	110000000
$I_v(0)$	1000000

The estimated demographic data, as well as parameters values given in Table (10), were taken from Appiah et al. (2024).

Table 10: Parameters Values from Demographic Data and from Least-Squares Best Fit.

Parameters	Units (day^{-1})	Values	Sources
Λ_h	day^{-1}	1200	Estimated from Data
Λ_v	day^{-1}	12000.6970	Estimated from data
β_h	day^{-1}	0.61844195	Estimated from data
β_v	day^{-1}	0.62695935	Estimated from data
μ_h	day^{-1}	$1/(65 \times 365)$	WFB, WHO
μ_v	day^{-1}	0.025	Estimated from data
δ	day^{-1}	0.00143	Estimated from data
γ	day^{-1}	0.2	treatment rate
α	day^{-1}	0.05	prevention rate
ω	day^{-1}	0.00100000	Estimated from data

Figs. (10-12) respectively, display plots of the solution of the system in (28).

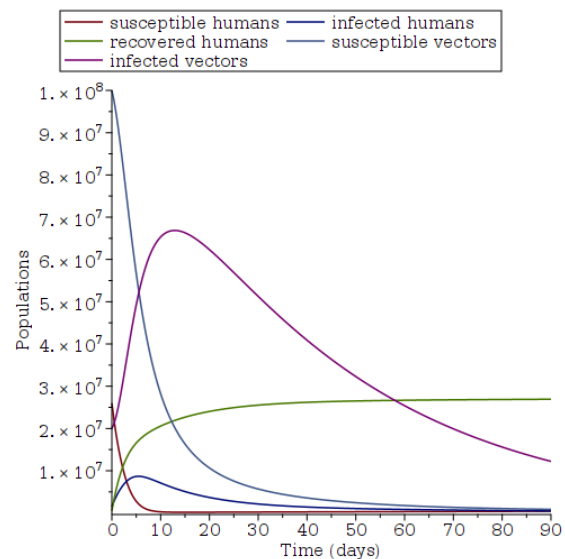


Fig. 10: Plot of All Populations from (28)

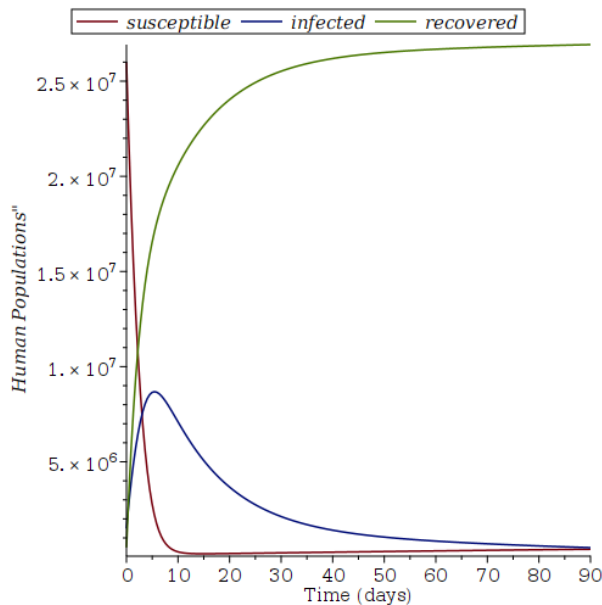


Fig. 11: Plot of Human Populations from (28)

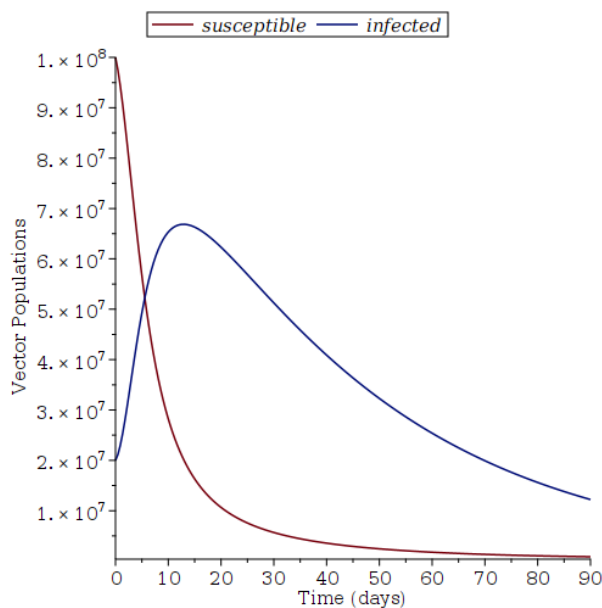


Fig. 12: Plot of Vector Populations from (28)

Simulations to verify Corollary (1). The following set of simulations were performed to ascertain the effects of increasing κ values on the various sub-populations stated in Corollary (1).

The proposed incidence term, $\frac{\beta_v(1-\kappa)I_h(t)}{N_h(t)}S_v(t)$, is a decreasing function of κ .

Table (11) shows that the incidence (from untreated infectious hosts, $(1 - \kappa)I_h(t)$, to susceptible vectors, $S_v(t)$), decrease with increasing κ -values.

The resulting infectious vector populations also decrease with increasing κ -values, as shown in Table (12).

Table 11: Effect of increasing κ -values on incidence

κ	$\frac{\beta_v(1-\kappa)I_h(t)}{N_h(t)}S_v(t)$
0.0	0.00001543763215
0.2	0.00001286989570
0.4	$9.905752224 \times 10^{-6}$
0.6	$6.445727364 \times 10^{-6}$
0.8	$2.354025870 \times 10^{-6}$
0.9	$1.465419577 \times 10^{-8}$
1.0	$Float(\infty)$

Table 12: Effect of increasing κ -values on Infectious Vectors

κ	$I_v(t)$
0.0	0.4318218786
0.2	0.3599970824
0.4	0.2770839782
0.6	0.1803000662
0.8	0.06584687742
0.9	0.0004099075734
$\kappa = 1.0$	$Float(\infty)$

Fig. (13) displays a decreasing sequence of infected vector populations, with increasing κ -values.

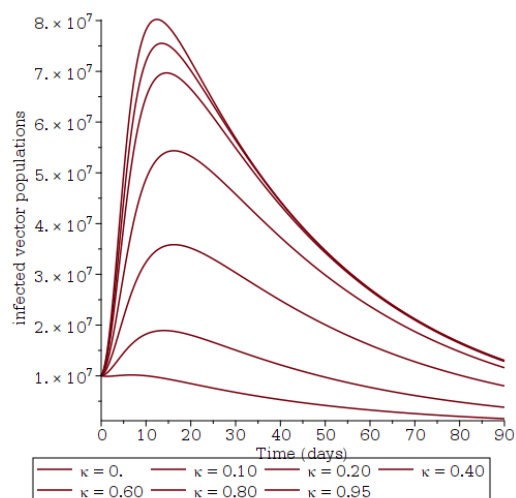


Fig. 13: Infected vector populations, with increasing κ values.

Table (13) display a corresponding decreasing sequence of infectious hosts, with increasing κ -values.

Table 13: Effect of increasing κ -values on Infectious Humans

κ	$I_h(t)$
0.0	0.02878831257
0.2	0.02663318547
0.4	0.02419738236
0.6	0.02082941476
0.8	0.01335005923
0.9	0.0001553316919
1.0	$Float(\infty)$

The resulting effect on infected host populations, shown in Fig. (14), also confirms a decreasing sequence with increasing κ -values.

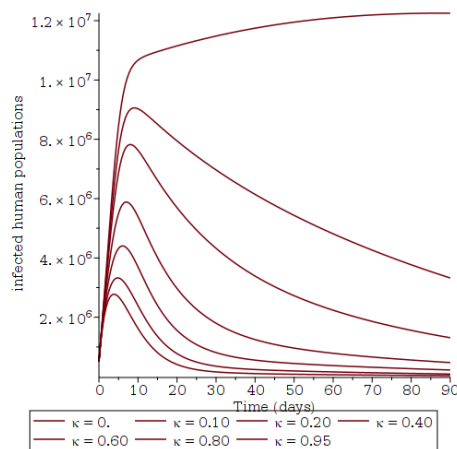


Fig. 14: Infected human populations, with increasing κ -values

The decreasing sequence of infected host populations is the direct result of decreasing infected vector populations.

The recovery term, $\gamma\kappa I_h(t)$, is an increasing function of κ .

Table (14) demonstrates that recovery increases with increasing κ -values.

Table 14: Effect of increasing κ -values on Recovery

κ	$R(t)$
0.0	0.001297834348
0.2	0.2515569965
0.4	0.4773048762
0.6	0.6799221002
0.8	0.8533652378
0.9	0.9082813369
1.0	<i>Float</i> (∞)

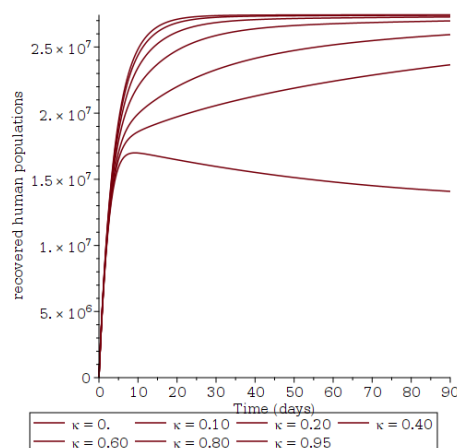


Fig. 15: Recovered human populations, with increasing κ values

Fig. (15) shows a plot of increasing recovery populations, with increasing κ -values.

The disease-induced death term $\delta_\kappa I_h(t) \triangleq \delta(1 - \kappa)I_h(t)$, is a decreasing function of κ .

Table (15) shows that increasing κ reduces disease-induced deaths.

Table 15: Effect of increasing κ -values on disease-induced deaths

κ	$\delta(1 - \kappa)I_h(t)$
0	0.00004116728698
0.2	0.00003046836418
0.4	0.00002076135406
0.6	0.00001191442524
0.8	$3.818116940 \times 10^{-6}$
0.9	$2.221243194 \times 10^{-8}$
1.0	<i>Float</i> (∞)

The following Simulations verify Theorem (1).

Table (16) confirms the statement of Theorem (1). That is, $\mathcal{R}_0^{(\kappa)}$ is a strictly decreasing function of κ , with maximum and minimum values occurring at $\kappa = 0$, and $\kappa = 1$, respectively.

Table 16: Effect of increasing κ values on $\mathcal{R}_0^{(\kappa)}$

κ	$\mathcal{R}_0^{(\kappa)}$
0.0	35.09815108
0.2	5.935705298
0.4	3.667791787
0.6	2.452645848
0.8	1.50422990
0.9	1.003331986
1.0	0.1

Remark 5

We observe that in each of the Tables (11-15), the last row, which is evaluated at $\kappa = 1$, is undefined. This is expected because the endemic equilibrium point, expressed in terms of $(\frac{1}{\mathcal{R}_0^{(\kappa)}})^2$, has a factor $(1 - \kappa)$ in the denominator. So, $\kappa = 1$, makes it undefined.

The epidemiological interpretation is that when $\kappa = 1$, then a typical infective has access to effective healthcare and affordable treatment. This implies that the instantaneous incidence is zero when $\kappa = 1$, and so endemic equilibrium does not exist, when $\kappa = 1$.

Results and Discussion

We have proposed, in Proposition (1), a novel characterization of the standard incidence term, recovery term, and disease-induced death term in terms of a healthcare accessibility parameter, κ . Here, accessibility

refers to availability of healthcare and affordability of treatment.

1. the proposed standard incidence term is given by:

$$\lambda_{\kappa}(t)S(t) = \frac{\beta(1-\kappa)I(t)}{N(t)}S(t), \quad (32)$$

with force of infection given by:

$$\lambda_{\kappa}(t) = \frac{\beta(1-\kappa)I(t)}{N(t)}, \text{ and prevalence of the disease given by } \frac{(1-\kappa)I(t)}{N(t)}.$$

2. the recovery term is

$$\gamma\kappa I(t), \quad (33)$$

3. the disease-induced death term is given by:

$$\delta_{\kappa}I(t) \triangleq \delta(1-\kappa)I(t). \quad (34)$$

Hitherto, the incidence term, recovery term, and disease-induced death term were all expressed as proportions of $I(t)$. From our characterizations in Proposition (1), the role of healthcare accessibility in effectively reducing incidence, disease-induced deaths, as well as speeding up recovery, is brought to the fore.

We have also shown that:

- the proposed novel incidence term is a decreasing function of κ , which implies that increasing healthcare accessibility reduces new cases.

Our simulations, using a vector-host model given in Eq. (28) shows, in Table (11) that, increasing healthcare accessibility reduces the incidence from untreated infectious hosts to susceptible vectors. In Table (12), and Fig. (13) we observe a decreasing sequence of infected vector populations, with increasing healthcare accessibility. Then, in Table (13), and Fig. (14), we observe a corresponding sequence of decreasing infected human populations, with increasing healthcare accessibility.

- the associated recovery term, $\gamma\kappa I_h(t)$, is an increasing function of κ . This means that increasing healthcare accessibility speeds up recovery.

Table (14), shows an increasing sequence of recovered human populations, as healthcare accessibility increases. The plots in Fig. (15) show an increasing sequence of recovered hosts with increasing accessibility

- the disease-induced death term $\delta_{\kappa}I_h(t) \triangleq \delta(1-\kappa)I_h(t)$, is a decreasing function of κ .

That is, increasing healthcare accessibility reduces disease-induced deaths.

Table (15) confirms that increasing healthcare accessibility reduces disease-induced deaths.

We have proved that the basic reproduction number, $\mathcal{R}_0^{(\kappa)}$, resulting from these novel characterizations, is a strictly decreasing function of κ , satisfying,

$$\mathcal{R}_0^{(1)} \leq \mathcal{R}_0^{(\kappa)} \leq \mathcal{R}_0^{(0)}, \quad \text{for } 0 \leq \kappa \leq 1. \quad (35)$$

where, $\mathcal{R}_0^{(0)}$ and $\mathcal{R}_0^{(1)}$ are respectively, the values of $\mathcal{R}_0^{(\kappa)}$ when $\kappa = 0$ and $\kappa = 1$.

The main observations from Eq. (35) are,

1. when the accessibility parameter $\kappa = 0$ (implying that a typical infective has no access to healthcare), the basic reproduction number attains its maximum possible value. Such a scenario results in having the maximum number of untreated cases becoming reservoirs for further transmission. The resulting increase in new cases makes it more difficult to control the spread of the disease.
2. On the other hand, when $\kappa = 1$, (implying that a typical infective has access to proper and effective treatment), the corresponding basic reproduction number has its minimum possible value; thus, making it easier to control the disease.
3. Since $\mathcal{R}_0^{(\kappa)}$ is a decreasing function of κ , increasing healthcare accessibility reduces the size of the basic reproduction number; thus, making it easier to control, and possibly, eradicate an infectious disease. Our simulations, shown in Table (16), shows a decreasing sequence of $\mathcal{R}_0^{(\kappa)}$ values, with increasing κ -values.

Conclusion

The essential role of healthcare accessibility in effectively controlling, and possibly eradicating, infectious diseases cannot be overemphasized. Our novel characterizations of the incidence term, recovery term, disease-induced death term as well as the basic reproduction number, in terms of a healthcare accessibility parameter, κ , demonstrates the effect of increasing healthcare accessibility on reducing incidence, speeding up recovery, reducing disease-induced deaths, as well as reducing the size of the basic reproduction number. We contend that healthcare accessibility is as important as the existence of a cure, for the effective control of any infectious disease.

This research has thus, provided a profound insight into the reasons, (and possibly, answers to) why some infectious diseases which have a cure still remain endemic worldwide.

Recommendations

Healthcare accessibility has two components namely, availability and affordability.

To increase availability, Public Health Authorities must ensure equitable distribution of healthcare facilities, especially, outside the major city centres.

In addition, proper incentives must be provided in order to maintain a reasonable doctor-to-patient ratio in remote communities, which are often, the epicenters of disease outbreaks.

To ensure affordability, treatment costs especially, for endemic diseases such as malaria, must be highly, (better still, fully) subsidized. Every untreated case becomes a reservoir for new transmissions, resulting in a snowball effect on treatment costs.

Future Work

Future research includes the application of Proposition (1) to the study of 6-state, and 7-state vector-host epidemic models, epidemic models with non-linear incidence, and multi-strain epidemic models.

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Dedication

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Ethics

The author declares a no conflict of interest. Moreover, the work is an original research that has not been published previously, and not under consideration for publication elsewhere.

The Next-Generation Matrix Method for $\mathcal{R}_0$

The basic reproduction number is one of the most important threshold quantities used in epidemiology. it is denoted by $\mathcal{R}_0$, and is defined as the average number of secondary infections produced when one infective is

introduced into a host population where everyone is susceptible.

The next generation matrix G (Diekmann et al., 1990; van der Driessche and Watmough, 2002) is given by:

$$G = FV^{-1}$$

where:

$$F = \left[\frac{\partial \mathcal{F}_i(E^0)}{\partial x_j} \right], \quad V = \left[\frac{\partial \mathcal{V}_i(E^0)}{\partial x_j} \right],$$

and E^0 denotes the disease-free equilibrium.

The (i, j) entry of the infection matrix F , is the number of new infections at stage j caused by contacts with diseased individuals in stage i ; while the (i, j) entry of the transition matrix V , is the rate individuals in stage j progress to stage i .

The (i, j) entry of V^{-1} is the expected time spent in compartment i by an individual initially in compartment j over the course of its infection.

The basic reproduction number $\mathcal{R}_0$, is the dominant eigenvalue of G denoted by $\rho(G)$. That is

$$\mathcal{R}_0 = \rho(G) = \rho(FV^{-1}) \quad (36)$$

The (i, j) entries of the matrix G give the rate at which infected individuals of state j generate new infections of type i .

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