

Stochastic Models for Epidemic Processes in Open Populations with External Infections

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Abstract

This paper studies stochastic epidemic models for infectious disease transmission in an open population with external infections. We highlight the limitations of classical deterministic epidemic models, particularly the Susceptible-Infected-Recovered (SIR) framework, which assumes a closed population and does not adequately capture the uncertainty and randomness inherent in epidemic spread. Stochastic birth-death models incorporate random fluctuations and provide a more realistic probabilistic description of epidemic dynamics. However, standard birth-death formulations generally do not account for infections entering the population from external sources. To address this limitation, we consider a birth-death process with immigration, where immigration represents infections introduced from outside the population. The paper reviews the unpublished work of Kobayashi, who first proposed this birth-death-immigration framework in the context of COVID-19 modelling. One of the remarkable features of this model is that it admits an exact analytical solution in terms of a generalized negative binomial distribution.

The paper presents the probability distribution, expected values, and variances associated with the model, and discusses two immigration mechanisms: all-state immigration and zero-state immigration. In addition, several extensions involving time-varying infection and immigration rates are investigated to demonstrate how nonhomogeneous assumptions influence epidemic behaviour and intervention outcomes. Since this work is intended as a review and exposition of Kobayashi's unpublished results, the focus is on summarizing the theoretical results, clarifying the probabilistic structure of the model, and discussing possible extensions and applications in epidemic modelling.

Key words: Stochastic models; Infectious disease dynamics; Open populations; External infections; Birth-death-immigration process; Time-nonhomogeneous models.

1. Introduction

In recent years, the limitations of purely deterministic epidemic models have become increasingly apparent, particularly in the context of emerging infectious diseases and globally connected populations. Deterministic compartmental models such as the Susceptible-Infected-Recovered (SIR) model (see a review in Allen *et al.* (2008)), although mathematically tractable and useful for large-scale approximations, often fail to capture the stochastic variability that is naturally present in epidemic transmission. Such variability becomes especially important when infection counts are small, when outbreaks begin through isolated introductions, or when external infections continuously reintroduce disease into a population.

Classical stochastic birth-death models improve upon deterministic formulations by incorporating randomness directly into the infection and recovery mechanisms (Bailey, 1991). These models provide insight into extinction probabilities, variability in outbreak sizes, and the occurrence of rare but significant epidemic events. However, many standard birth-death epidemic models still assume effectively closed populations and therefore do not explicitly model infections entering from outside sources.

The present paper focuses on a birth-death process with immigration, where immigration represents external infections entering the population. The model considered here is based on the unpublished work of Kobayashi (2021b,c), who introduced this framework for modelling COVID-19 transmission dynamics. A particularly striking aspect of the model is that it possesses an exact analytical solution characterized by a generalized negative binomial distribution. This feature makes the model mathematically elegant while also providing a useful probabilistic description of epidemic spread in open populations.

The primary goal of this paper is not to introduce a completely new model, but rather to provide a careful review, exposition, and summary of Kobayashi's unpublished results. In doing so, we preserve the original mathematical structure while presenting the results and interpretations in a unified and accessible manner. The paper also discusses possible extensions, including time-nonhomogeneous formulations in which infection and immigration rates vary over time. Additional details, including derivations and numerical results, can be found in Madushani U.K.A. (2025).

Infectious diseases have posed a persistent threat to human health throughout history, leading to significant morbidity, mortality, and societal disruption, with some escalating into devastating global pandemics. Understanding and predicting the spread of these diseases is paramount for implementing effective public health interventions and mitigating their impact. Mathematical models have become indispensable tools in this endeavour, offering frameworks for analyzing disease transmission dynamics and evaluating potential control strategies. The mathematical study of infectious diseases is considered to have originated with John Graunt in 1662 (see a review of the history in Brauer (2017)). Subsequently, as reviewed by Kretzschmar and Wallinga (2010), a foundational model in epidemiology was formulated in 1927 by Kermack and McKendrick: the deterministic Susceptible-Infected-Recovered (SIR) model. This model, based on dividing the population into compartments representing individuals' disease status, marked the beginning of compartmental modelling and provided a basic deterministic framework for understanding disease spread within a closed population. The introduction of the "Basic Reproduction Number" (R_0) by MacDonald (1952) further enhanced the understanding of disease transmissibility. Over time,

various extensions and modifications of the SIR model, such as SIS (Susceptible - Infected - Susceptible), SEIS (Susceptible - Infected - Exposed - Susceptible), and SVIR (Susceptible - Vaccinated - Infected - Recovered), have been developed to account for different disease characteristics and epidemiological scenarios. However, deterministic models operate under simplifying assumptions, such as fixed infection and recovery rates, which ignore the inherent randomness in disease transmission.

A significant limitation of deterministic models is their inability to account for random variations and the unpredictability of real-world epidemics, thus failing to accurately forecast dynamic disease patterns (Andersson and Britton, 2012). To address these limitations, stochastic models were introduced, incorporating the element of randomness and variability in disease transmission. Stochastic models are crucial for reflecting the unpredictable nature of human interactions and disease progression, capturing the uncertainty inherent in real-world epidemics and accounting for chance occurrences that can significantly alter the course of disease spread. While the foundational concepts of stochastic processes emerged in 1963, their application in epidemiological literature became more prevalent starting in 1978 (see a review in Brauer (2017)). In recent years, Allen (2008, 2010) has significantly contributed to epidemiological research, particularly in the analysis of stochastic epidemic models. Here, we explore the application of birth-death and birth-death-immigration stochastic models to understand the spread of disease, particularly in open populations where external infections can occur. The concept of the birth-death process, initially used for population growth analysis, has been adapted to model infectious disease dynamics, focusing on the number of infected individuals over time. In 1939, Feller highlighted the significance of stochastic processes for understanding population growth by analysing a birth-death process where the birth rate λ and death rate μ are constants. Kendall (1948b) extended Feller's work by considering the birth and death rates as functions of time t denoted as $\lambda(t)$ and $\mu(t)$. Since then, various birth-death models have been developed under different assumptions about the temporal dynamics of model parameters. Bailey (1991) and Andersson and Britton (2012) showed that a birth-death process can also be used to study infectious disease dynamics where the number of individuals infected is the main interest. Allen *et al.* (2017) demonstrated ways to use birth-death models in epidemiology to capture the dynamic behaviour of infected individuals in a system. The immigration concept was introduced to the birth-death process by Kendall (1948a) and birth-death immigration processes were reviewed in the book "The Elements of Stochastic Process with Applications to the Natural Sciences" by Bailey (1991). For modelling infectious diseases, "immigration" can be used to represent the introduction of new infections from external sources, herein called external infections. The concept of external infection was not utilized in COVID-19 epidemiology until Kobayashi (2020) discussed the significance of the birth-death-immigration stochastic model for studying open populations. He introduced stochastic models based on the birth-death process with external infection for homogeneous and non-homogeneous cases, incorporating random variations in infection and recovery/death rates in his unpublished series of papers. He noted that these stochastic models could offer more accurate predictions of epidemic patterns in real-world situations (see Kobayashi (2020, 2021a,b,c)).

The research presented here aims to contribute to a deeper understanding of infectious disease dynamics in open populations by: i) (Section 2) Reviewing the deterministic SIR model and emphasizing its limitations, particularly the closed population assumption and the lack of inherent variability; ii) (Section 3) Examining the application of a basic

stochastic birth-death model in epidemiology and discussing its limitations, such as the absorbing state of disease extinction; iii) (Section 4) Investigating birth-death-immigration models under two distinct policies for external infections: all-state immigration (continuous) and zero-state immigration (reintroduction after extinction); and iv) (Section 5) Analyzing the impact of varying internal and external infection rates on the expected number of infected individuals in a time-nonhomogeneous birth-death all-state immigration model, thereby exploring the effects of intervention measures. By addressing these objectives, this paper aims to underscore the importance of stochastic modelling, particularly birth-death-immigration processes, for gaining valuable insights into the uncertainties and variations inherent in real-world epidemic scenarios.

2. Review of deterministic SIR model

The deterministic SIR model serves as a foundational framework in epidemiology for understanding the spread of infectious diseases within a population. Despite having limitations, it provides essential insights into the basic dynamics of epidemics. The deterministic SIR model divides a population of size N into three compartments: Susceptible (S), Infected (I), and Recovered (R). It assumes a closed population where the total size remains constant $S(t) + I(t) + R(t) = N$. The model tracks the flow of individuals between these compartments based on two key parameters:

- β (Per-capita infection rate): Represents the rate at which susceptible individuals become infected through contact with infected individuals. The rate of new infections is proportional to the product of the number of susceptible and infected individuals $S(t)I(t)$.
- γ (Per-capita recovery rate): Represents the rate at which infected individuals recover from the disease and become immune. This is the inverse of the average infectious period.

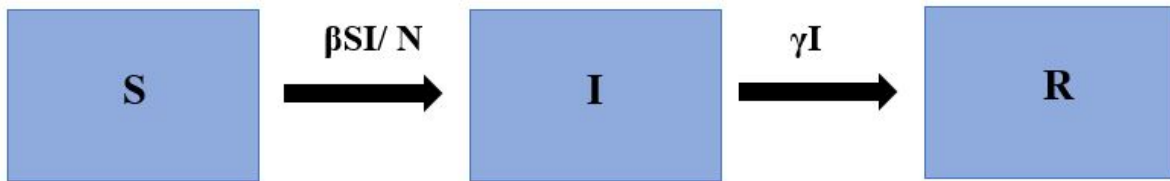


Figure 1: SIR compartmental diagram: Susceptible (S), Infected (I), Recovered (R):

$S(t) + I(t) + R(t) = N$ for all t .

The dynamics of the deterministic SIR model are described by a system of ordinary differential equations:

$$\frac{dS(t)}{dt} = -\frac{\beta S(t)I(t)}{N} \frac{dI(t)}{dt} = \frac{\beta S(t)I(t)}{N} - \gamma I(t) \frac{dR(t)}{dt} = \gamma I(t).$$

These equations describe the rates of change in the number of individuals in each compartment over time.

Two critical quantities derived from the deterministic SIR model are the Basic Reproduction Number R_0 and the Effective Reproduction Number R_t .

- **Basic Reproduction Number R_0 :** Defined as the average number of secondary infections generated by a single infected individual in a completely susceptible population.

$$R_0 = \frac{\beta}{\gamma}.$$

- **Effective Reproduction Number R_t :** Represents the average number of secondary infections produced by an infectious individual at a specific time t .

$$R_t = R_0 \cdot \frac{S(t)}{N}.$$

The **Final Epidemic Size** in the deterministic SIR model refers to the total proportion of the population that becomes infected and recovers by the end of the epidemic. Let s_∞ be the proportion susceptible at the end and let r_∞ be the proportion recovered at the end, then:

$$s_\infty = e^{R_0(s_\infty - 1)}, \quad r_\infty = 1 - s_\infty = 1 - e^{-R_0 r_\infty}.$$

2.1. Example numerical results for the SIR model

In order to compare later to birth-death-models, this section illustrates a numerical solution of the deterministic SIR model. Euler's method was used to explore the dynamic behaviour of each compartment: Susceptible, Infected and Recovered, considering two different scenarios of R_0 : $R_0 < 1$ and $R_0 > 1$. This approach allows us to computationally simulate and analyze infectious disease transmission dynamics within a closed population under specific model parameters. We select a population $N = 100$ and the initial number of infectives $I(0) = 5$ for both cases. Thus, $S(0) = 95$ and $R(0) = 0$. Two figures for $R_0 < 1$ and $R_0 > 1$ are generated for two different sets of parameters to illustrate the dynamic behaviour of each compartment.

In Figure 2(a), the infection rate $\beta = 0.3$ (i.e., on average, each infected individual will transmit the disease to one other individual approximately every 3.33 days) and recovery rate $\gamma = 0.1$ (i.e. the average infectious period is 10 days). In this case, $R_0 = \frac{0.3}{0.1} = 3 > 1$. The number of susceptibles sharply declines as the disease spreads rapidly due to each infected individual transmitting the disease to more than one other person. The number of recovered rises steadily, since all individuals who get infected eventually recover. The number of infected individuals initially increases as more susceptible individuals are available to sustain transmission. Subsequently, the epidemic reaches its peak. Following this, the number of new cases declines and eventually ceases to change as fewer susceptible individuals are available to infect. The number of infections declines to zero over time since no infective individuals are left in the closed population.

On the other hand, in Figure 2(b), $\beta = 0.6$ and $\gamma = 0.8$, so that $R_0 = 0.75 < 1$; the disease rapidly disappears. These two plots demonstrate the impact of R_0 on disease dynamics.

In summary, when $R_0 > 1$, the disease initially spreads rapidly, peaks as the number of susceptible individuals decreases due to infection, and eventually dies out as there will be no infective individuals left in the closed population. Conversely, when $R_0 < 1$, the infection naturally diminishes over time as each infected individual spreads the disease to less than one other person.

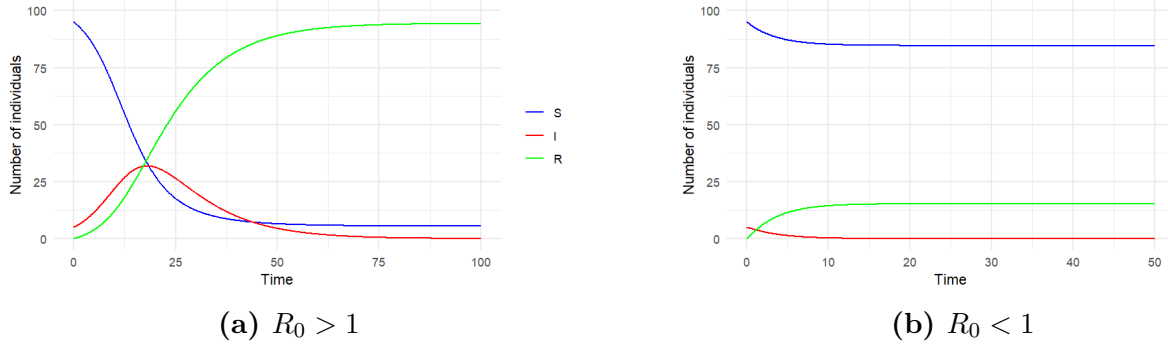


Figure 2: Numerical solutions for deterministic SIR model using Euler's method for the parameters: (a) $\beta = 0.3$ and $\gamma = 0.1$ (for $R_0 > 1$) and (b) $\beta = 0.6$ and $\gamma = 0.8$ ($R_0 \leq 1$) with $N = 100$, $I_0 = 5$. The resulting plots illustrate the susceptible, infectious, and recovery populations by blue, red and green curves, respectively.

2.2. Limitations of deterministic SIR models

While the deterministic SIR model provides a valuable initial framework for understanding epidemic dynamics, it suffers from several limitations. These limitations highlight the need for more sophisticated modelling approaches, such as stochastic models, especially when dealing with real-world scenarios. Key limitations of the SIR model include:

Closed Population Assumption: The deterministic SIR model assumes a constant, closed population, neglecting the movement of individuals into or out of it. In reality, populations are often open, with migration and the introduction of external infections significantly impacting disease spread. For example, international travel played a crucial role in the rapid global spread of COVID-19.

Deterministic Nature: The model is deterministic, meaning that given initial conditions and parameters, the outcome is exactly predictable at any given time. This fails to account for the inherent randomness and variability in biological and social processes that influence disease transmission. Real-world epidemics exhibit unpredictable patterns due to chance events in individual contacts and disease progression.

Homogeneous Mixing: The assumption of homogeneous mixing, where every individual has an equal probability of contact with every other individual, is a simplification of complex social interaction patterns. Heterogeneities in contact structures can significantly affect disease spread.

Nonlinearity: The nonlinear nature of the differential equations can make analytical solutions difficult to obtain for more complex variations of the model.

Ignoring Stochastic Effects: Deterministic models are inadequate for describing the initial stages of an epidemic when the number of infected individuals is small, where stochastic

tic fluctuations can play a significant role in determining whether an outbreak occurs or fades out. They also fail to capture the possibility of disease extinction even when the conditions for spread ($R_0 > 1$) are met, due to random variations.

Inability to Explain Variability in Real-World Epidemics: Different regions or countries can experience significantly different epidemic patterns for the same disease even with seemingly similar conditions, a phenomenon that deterministic models struggle to explain due to their lack of inherent randomness.

These limitations of the deterministic SIR model underscore the necessity of employing stochastic models to provide a more realistic and comprehensive understanding of infectious disease dynamics, particularly in open populations influenced by external factors. It should be noted, however, that deterministic spatial models have been developed that allow movement of individuals between multiple populations (see for example Chen *et al.* (2020) and references therein). In this paper we focus only on the single population scenario.

3. Simple linear birth-death process in epidemiology

In a simple linear birth-death process applied to epidemiology, it is often assumed that the susceptible population is large enough to be considered infinite, resulting in an open population model where only the dynamics of the infected population are explicitly tracked. The rates of birth (infection) and death (recovery/death) are assumed to be linearly dependent on the current number of infected individuals n :

- **Birth rate (λ_n):**

$$\lambda_n = n\lambda$$

where λ is the per-capita infection rate. This implies that the total rate of new infections is proportional to the number of currently infected individuals.

- **Death rate (μ_n):**

$$\mu_n = n\mu$$

where μ is the per-capita recovery/death rate. This implies that the total rate of removal of infected individuals is proportional to the number of currently infected individuals.

When the number of infected individuals, n , reaches zero, the birth rate $\lambda_0 = 0$, indicating that state 0 is an absorbing state. Once the process reaches this state, the infection becomes extinct and the number of infected individuals remains at zero indefinitely. This process has been well studied by others (see Allen (2010); Bailey (1991); Kobayashi (2020)) and we give a brief summary of what is known along with example simulations in the remainder of this section.

3.1. Probability generating function and time-dependent distribution

The time-dependent probability mass function $P_n(t) = \mathbb{P}(I(t) = n \mid I(0) = I_0)$ gives the probability of having n infected individuals at time t given an initial number of

infections I_0 . These state probabilities satisfy the forward Kolmogorov differential equations:

$$\frac{dP_n(t)}{dt} = \lambda(n-1)P_{n-1}(t) + \mu(n+1)P_{n+1}(t) - (\lambda + \mu)nP_n(t), \quad n \geq 1. \quad (1)$$

When $n = 0$, $\lambda_0 = 0$ because $\lambda_n = n\lambda$ and we have:

$$\frac{dP_0(t)}{dt} = \mu P_1(t), \quad n = 0.$$

These are differential-difference equations and can be explicitly solved using the probability or moment-generating functions (PGF) approach. The PGF $G(z, t)$ is defined as:

$$G(z, t) = \sum_{n=0}^{\infty} z^n P_n(t).$$

For the simple linear birth-death process with $I(0) = I_0$, the PGF, $G(z, t)$, is given by (see Allen (2010); Bailey (1991); Kobayashi (2020)):

$$G_{\text{BD}:I_0}(z, t) \equiv G(z, t) = \left(\frac{\frac{\mu(e^{at}-1)}{\lambda e^{at}-\mu} + \frac{1-(\mu(e^{at}-1))}{\lambda e^{at}-\mu} - \frac{\lambda(e^{at}-1)}{\lambda e^{at}-\mu} z}{1 - \left(\frac{\lambda(e^{at}-1)}{\lambda e^{at}-\mu} \right) z} \right)^{I_0}, \quad (2)$$

where $a = \lambda - \mu$.

From this PGF, the time-dependent probability mass function $P_n(t)$ can be derived as:

$$P_n(t) = \sum_{j=0}^{\min(I_0, n)} \binom{I_0}{j} \binom{I_0 + n - j - 1}{I_0 - 1} \alpha(t)^{I_0-j} \beta(t)^{n-j} (1 - \alpha(t) - \beta(t))^j$$

for $n \geq 1$, where

$$\alpha(t) = \frac{\mu(e^{at} - 1)}{\lambda e^{at} - \mu}, \quad \beta(t) = \frac{\lambda(e^{at} - 1)}{\lambda e^{at} - \mu}.$$

The probability of extinction is:

$$P_0(t) = \alpha(t)^{I_0}.$$

3.2. Expected value and variance

$$E[I(t)] = \begin{cases} I_0 e^{at}, & \text{if } \lambda \neq \mu \\ I_0, & \text{if } \lambda = \mu. \end{cases} \quad (3)$$

The variance $\text{Var}(I(t))$ is given by:

$$\text{Var}(I(t)) = \begin{cases} I_0 \cdot \frac{\lambda+\mu}{\lambda-\mu} \cdot e^{(\lambda-\mu)t} \cdot (e^{(\lambda-\mu)t} - 1) & \text{if } \lambda \neq \mu \\ 2I_0\lambda t & \text{if } \lambda = \mu. \end{cases}$$

The variance demonstrates that the stochastic fluctuations around the expected value can be significant, especially over time.

3.3. Simulation of the birth-death process

To visualize the dynamic behaviour of the number of infected individuals over time within the stochastic framework, simulation of sample paths is crucial. This involves simultaneously computing the transition probabilities and the holding times in each state. The holding time in state n is exponentially distributed with rate $n(\lambda + \mu)$.

When a transition occurs, it is either a:

- **Birth (infection)** with probability $\lambda/(\lambda + \mu)$, or a
- **Death (recovery/death)** with probability $\mu/(\lambda + \mu)$.

Simulations reveal that individual sample paths can exhibit significant variability around the expected trajectory. Importantly, simulations demonstrate that even when $\lambda > \mu$ and the expected number of infected individuals grows exponentially, there is always a positive probability of extinction (reaching the absorbing state 0) in the stochastic model. See Figure 3.

This highlights a key difference from the deterministic model, where extinction is impossible if the birth rate exceeds the death rate.

3.4. Limitations of the linear birth-death model

While the linear birth-death model incorporates stochasticity, it also has limitations. The most significant limitation in the context of long-term epidemic dynamics is the absorbing state at zero. Once the number of infected individuals reaches zero, the disease becomes permanently extinct in the model, and no further infections can occur. This is unrealistic for many real-world diseases that can be reintroduced into a population through external sources or re-emergence. This limitation motivates the exploration of birth-death-immigration models, which account for the introduction of new infections from outside the population.

4. Birth-death immigration models

To overcome the limitation of the simple birth-death model regarding disease extinction, birth-death-immigration models incorporate the introduction of new infections from external sources, termed “immigration”. This section examines two types of birth-death-immigration models relevant to infectious disease dynamics in open populations: all-state immigration [Kobayashi (2020)] and zero-state immigration [Shiny and Viswanath (2022)].

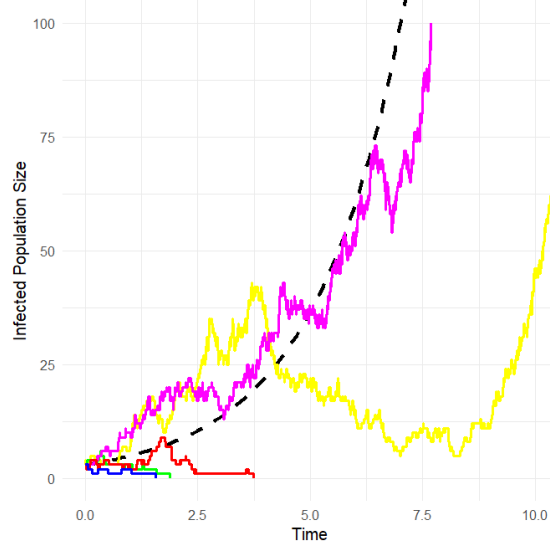


Figure 3: The stochastic mean (black dashed curve) and five sample paths of the birth and death model are plotted for $\lambda = 2$, $\mu = 1.5$, and $I_0 = 3$. Three curves (red, blue, green) from five sample paths quickly reach absorption while the remaining two curves (pink, yellow) continue.

4.1. Birth-death model with external infections via all-state immigration

The birth-death all-state immigration model extends the linear birth-death process by including a constant rate ν at which infected individuals enter the population from outside, regardless of the current number of infected individuals in the system. This model is characterized by three parameters:

- λ (Per-capita internal infection rate): As in the birth-death model, contributing to new infections within the population.
- μ (Per-capita recovery/death rate): Representing the removal of infected individuals.
- ν (External infection rate): A constant rate at which new infected individuals immigrate into the population per unit time.

The total infection rate when there are n infected individuals becomes $\lambda_n = n\lambda + \nu$, and the total recovery/death rate remains $\mu_n = n\mu$. This model allows for the persistence of the disease in the population, as new infections can continuously be introduced even when the number of internally generated infections is low.

4.1.1. Probability generating function and time-dependent distribution

Let $I_0 = I(0)$ denote the initial number of infected individuals in the system and $P_n(t)$ be the time-dependent probability mass function of the process $I(t)$:

$$P_n(t) = P(I(t) = n \mid I(0) = I_0), \quad n = 0, 1, 2, \dots \quad \text{and} \quad t \geq 0,$$

with initial condition $P_n(0) = \delta_{n,I_0}$, where δ_{n,I_0} is the Kronecker delta function. The state probabilities, $P_n(t)$, in the birth-death immigration model satisfy the following Forward Kolmogorov equations:

$$\begin{aligned} \frac{dP_0(t)}{dt} &= -\nu P_0(t) + \mu P_1(t), \quad n = 0, \\ \frac{dP_n(t)}{dt} &= [(n-1)\lambda + \nu]P_{n-1}(t) - [n(\lambda + \mu) + \nu]P_n(t) + (n+1)\mu P_{n+1}(t), \quad n = 1, 2, 3, \dots \end{aligned} \quad (4)$$

with initial condition $I(0) = I_0$, i.e., $P_n(0) = \delta_{n,I_0}$ for $n \in \{0, 1, 2, 3, \dots\}$.

The time-dependent PGF for the birth-death all-state immigration model with $I(0) = I_0$ is given by:

$$G(z, t) = G_{\text{BDI:0}}(z, t) \cdot G_{\text{BD:I}_0}(z, t),$$

where $G_{\text{BDI:0}}(z, t)$ is the PGF for the model with $I(0) = 0$, and $G_{\text{BD:I}_0}(z, t)$ is the PGF for the simple birth-death model with initial number of infections I_0 as in equation (2).

For the case with $I(0) = 0$, the PGF is:

$$G_{\text{BDI:0}}(z, t) = \left(\frac{\mu(e^{at} - 1)}{\lambda e^{at} - \mu} + \frac{\nu(e^{at} - 1)}{\lambda e^{at} - \mu} \cdot \frac{1 - z}{\lambda - \mu z} \right)^{-\nu/\lambda}$$

where $a = \lambda - \mu$. This corresponds to a generalized negative binomial distribution.

The probability mass function for $I(0) = 0$ is:

$$P_n(t) = \binom{n+r-1}{n} (1 - \beta(t))^r \beta(t)^n,$$

where $r = \nu/\lambda$ and $\beta(t) = \frac{\lambda(e^{at}-1)}{\lambda e^{at}-\mu}$.

4.1.2. Expected value and variance

The expected number of infected individuals for the birth-death all-state immigration model with $I(0) = I_0$ is:

$$\bar{I}(t) = \mathbb{E}[I(t)] = \begin{cases} I_0 e^{at} + \frac{\nu}{a}(e^{at} - 1), & \text{if } a \neq 0 \\ I_0 + \nu t, & \text{if } a = 0 \end{cases}$$

where $a = \lambda - \mu$.

The variance is:

$$\begin{aligned} \text{Var}(I(t)) &= \text{Var}_{\text{BD}}(I(t)) + \text{Var}_{\text{BDI:0}}(I(t)) \\ &= I_0 \cdot \frac{\lambda + \mu}{\lambda - \mu} e^{(\lambda-\mu)t} (e^{(\lambda-\mu)t} - 1) + \frac{\nu}{\lambda - \mu} e^{(\lambda-\mu)t} (e^{(\lambda-\mu)t} - 1), \quad \text{for } \lambda \neq \mu. \end{aligned}$$

For $I(0) = 0$, the variance simplifies to:

$$\text{Var}_{BDI:0}(I(t)) = \frac{\nu}{\lambda - \mu} e^{(\lambda - \mu)t} (e^{(\lambda - \mu)t} - 1).$$

A key feature of this model is that the variance of the number of infected individuals is often much higher than the mean, especially when $r = \nu/\lambda$ is small, leading to a high coefficient of variation ($\text{CV} = \frac{\sqrt{\text{Var}[I(t)]}}{E[I(t)]} > 1$). This indicates significant over-dispersion and a greater degree of unpredictability in the epidemic patterns compared to what might be expected from a deterministic model or a process with lower variability. The generalized negative binomial distribution, which describes the probability distribution, has a long tail, implying a non-negligible probability of observing very large numbers of infected individuals. See Tables 1 ($r = \frac{\nu}{\lambda} = \frac{0.5}{0.6} \approx 0.83$) and 2 ($r = \frac{\nu}{\lambda} = \frac{0.1}{0.6} \approx 0.16$).

Table 1: The mean, variance and coefficient of variation for linear $BD : 1$, $BDI : 0$ and $BDI : 1$, where per-capita infectious rate $\lambda = 0.6$, per-capita death/recovery rate $\mu = 0.4$, immigration rate $\nu = 0.5$, $a = \lambda - \mu = 0.2$.

	BD: I_0	BDI:0	BDI: I_0
$E[I(t)]$	54.5982	133.9954	188.5935
$\text{Var}[I(t)]$	14631.8	21679.71	36311.51
$\text{CV}[I(t)]$	2.2155	1.0988	1.0104

Table 2: The mean, variance, and CV for $BD : 1$, $BDI : 0$, and $BDI : 1$ for $\lambda = 0.6$, $\mu = 0.4$, $\nu = 0.1$.

	BD: I_0	BDI:0	BDI: I_0
$E[I(t)]$	54.5982	26.780	81.3972
$\text{Var}[I(t)]$	14631.8	4335.942	18967.74
$\text{CV}[I(t)]$	2.2155	2.4571	1.6920

4.1.3. Simulation results and percentile curves

Simulations of the birth-death all-state immigration model, as illustrated in figure 4, show considerable variability between individual sample paths, even under the same parameter values and initial conditions. This reinforces the idea that chance can play a significant role in the actual progression of an epidemic. The long tail of the probability distribution implies that extreme scenarios, with much higher numbers of infections than the expected value, have a non-zero probability of occurring.

Percentile curves, derived from the probability distribution and simulation results (Figures 5 and 6), further highlight this variability. These curves show the range within which a certain percentage of the simulated epidemic trajectories are expected to fall over time. The wide spread between the percentile curves, especially at later times when exponential growth (if $\lambda > \mu$) becomes dominant, emphasizes the uncertainty in predicting the exact number of infections. The median of the distribution often deviates significantly from the expected value, further indicating the skewness and the limitations of relying solely on the mean for predictions.

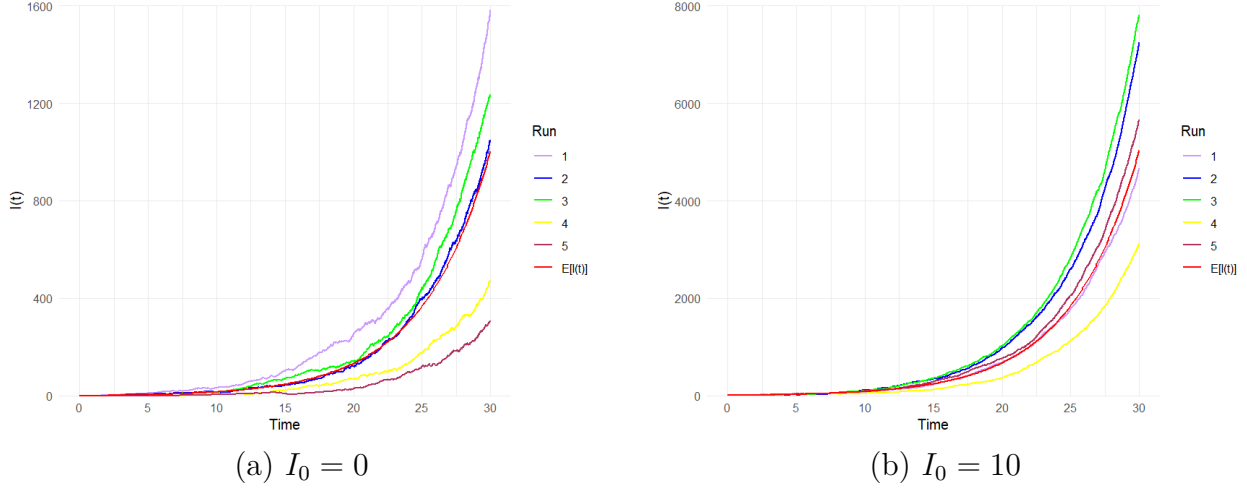


Figure 4: Five sample paths for $I(t)$ versus t corresponding to the birth-death all-state immigration model. The expected number of infected individuals (red) is shown for (a) with $I_0 = 0$ and (b) with $I_0 = 10$. Parameters: $\lambda = 0.6$, $\mu = 0.4$, $\nu = 0.5$.

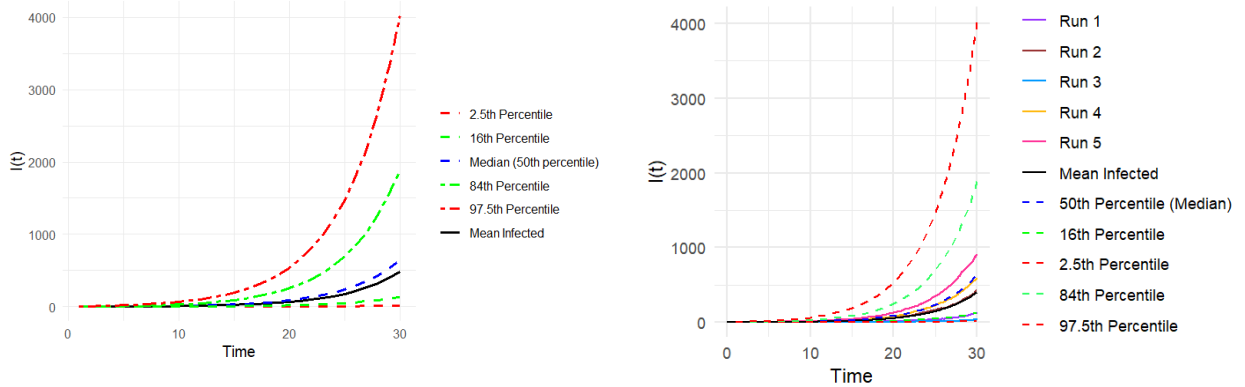


Figure 5: Percentile curves of $P_n(t)$ for $I(t)$ when $I_0 = 0$, $\lambda = 0.6$, $\mu = 0.4$, and $\nu = 0.5$ along with $\bar{I}(t)$.

Figure 6: Percentile curves of $P_n(t)$ for simulated $I(t)$ when $I_0 = 0$, $\lambda = 0.6$, $\mu = 0.4$, and $\nu = 0.5$ along with $\bar{I}(t)$.

4.1.4. Stationary probability distribution

When $\lambda \leq \mu$ (i.e., $a \leq 0$), the birth-death all-state immigration model reaches a stationary probability distribution:

$$\pi_n = \lim_{t \rightarrow \infty} P_n(t).$$

This distribution is also a generalized negative binomial distribution given by:

$$\pi_n = \binom{n+r-1}{n} \left(\frac{a}{\lambda}\right)^r \left(1 - \frac{a}{\lambda}\right)^n \quad (5)$$

where $r = \nu/\lambda$ and $a = \mu - \lambda$. The mean of this stationary distribution is:

$$\mathbb{E}[I] = \frac{\nu}{\mu - \lambda}, \quad \text{for } \lambda < \mu.$$

Simulations shown in Figure 7 illustrate how the number of infected individuals fluctuates around this stationary mean when $\lambda \leq \mu$.

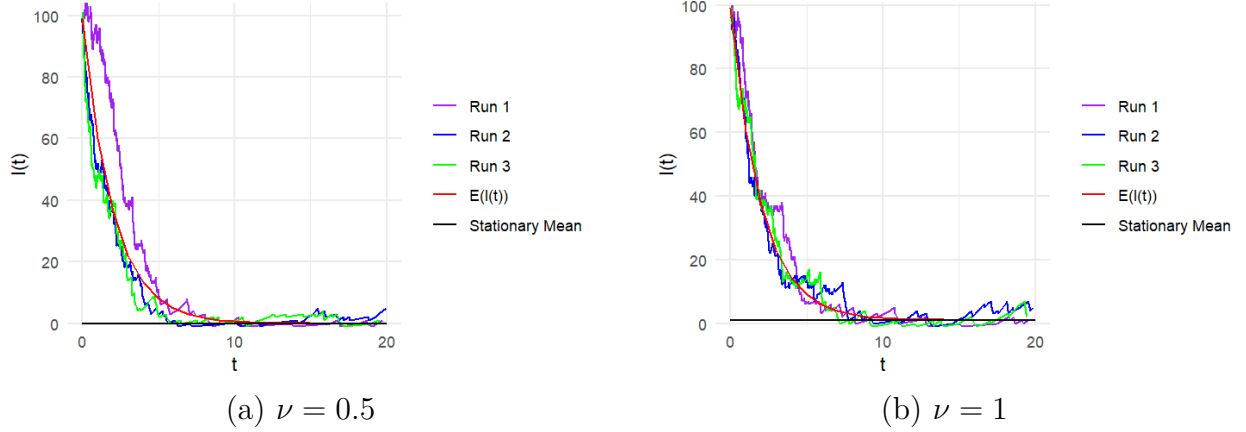


Figure 7: Three sample paths of $I(t)$ versus time t for the birth-death all-state immigration model along with the expected value $E[I(t)] = \bar{I}(t)$ (red-line) when $I(0) = 100$, $\lambda = 0.5$, $\mu = 1$, and (a) $\nu = 0.5$, (b) $\nu = 1$; the respective mean values of the stationary distribution are 1, 2 (horizontal lines).

4.1.5. Associated processes of birth-death model with immigration

In this section, we study the deterministic mean behaviour of the associated processes in the Birth-Death model with immigration. In addition to the process describing the number of infected individuals, $I(t)$, $t \geq 0$, we introduce the following processes:

1. The process $\{A(t), t \geq 0\}$ denotes the cumulative number of infectious individuals entering the population from outside by time t . We assume that $A(t)$ is a Poisson process with rate ν (individuals per unit time).
2. The process $\{B(t), t \geq 0\}$ denotes the cumulative number of internally generated infections by time t . Secondary infections are assumed to occur at rate λ (persons/unit time/infectious person).
3. The process $\{R(t), t \geq 0\}$ denotes the cumulative number of recovered or deceased individuals by time t . Such departures are assumed to occur at rate μ (persons/unit time/infectious person).

Let $I_0 = I(0)$ represent the initial number of infected individuals. Then, the number of infected individuals in the population at time t is given by

$$I(t) = I(0) + A(t) + B(t) - R(t). \quad (6)$$

The external arrival process $(A(t), t \geq 0)$ is a Poisson process with rate ν and is therefore well characterized. However, the processes $(B(t), t \geq 0)$ and $(R(t), t \geq 0)$ are more difficult

to analyze. Nevertheless, expressions for their expected values can be obtained from the mean behavior of the infection process $(I(t), t \geq 0)$.

Using the probability generating function associated with $(I(t), t \geq 0)$, the ordinary differential equation governing the mean number of infected individuals, $\bar{I}(t) = E[I(t)]$, is obtained as

$$\frac{d\bar{I}(t)}{dt} = (\lambda - \mu)\bar{I}(t) + \nu. \quad (7)$$

Solving the above differential equation yields

$$\bar{I}(t) = E[I(t)] = I_0 e^{at} + \frac{\nu}{a}(e^{at} - 1), \quad I_0 = I(0) \quad \text{and} \quad a = \lambda - \mu, \quad (8)$$

where $I_0 = I(0)$.

Since $A(t)$ is a Poisson process with rate ν , the expected number of external infectious arrivals by time t is

$$E[A(t)] = \bar{A}(t) = \nu t, \quad t \geq 0. \quad (9)$$

Because infected individuals generate secondary infections at rate λ , the expected cumulative number of internal infections, $\bar{B}(t)$, satisfies

$$\frac{d\bar{B}(t)}{dt} = \lambda \bar{I}(t). \quad (10)$$

Integrating the above equation and using the expression for $\bar{I}(t)$ gives:

$$\begin{aligned} E[B(t)] = \bar{B}(t) &= \lambda \int_0^t \bar{I}(u) du, \\ &= \frac{\lambda I_0 (e^{at} - 1)}{a} + \frac{\lambda \nu (e^{at} - 1)}{a^2} - \frac{\lambda \nu t}{a}, \\ &= \frac{\lambda}{a} (\bar{I}(t) - I_0 - \bar{A}(t)). \end{aligned} \quad (11)$$

Similarly, since the infected population recovers/dies at the rate of μ , the expected number of recovered or deceased individuals $\bar{R}(t)$, satisfies

$$\frac{d\bar{R}(t)}{dt} = \mu \bar{I}(t). \quad (12)$$

Integrating both sides and using Equation (8), we obtain the solution for the ODE (12):

$$\begin{aligned} E[R(t)] = \bar{R}(t) &= \mu \int_0^t \bar{I}(u) du, \\ &= \frac{\mu I_0 (e^{at} - 1)}{a} + \frac{\mu \nu (e^{at} - 1)}{a^2} - \frac{\mu \nu t}{a}, \\ &= \frac{\mu}{a} (\bar{I}(t) - I_0 - \bar{A}(t)). \end{aligned} \quad (13)$$

Although explicit expressions for the expected values of the associated processes can be obtained, a detailed characterization of the stochastic behaviour of the processes $(B(t), t \geq 0)$ and $(R(t), t \geq 0)$ remains an open problem and is currently under investigation. In particular, the process $(R(t), t \geq 0)$ resembles the departure process of a queueing system, and such processes are generally difficult to characterize analytically.

4.2. Birth-death model with immigration only at zero (zero-state immigration)

The birth-and-death zero-state immigration model presents an alternative scenario in which external infections (immigration at a rate ν) are introduced only when the number of infected individuals in the system reaches zero. This can model situations where strict control measures, such as travel bans, are implemented as soon as infections are detected and are only lifted when the infection is eradicated.

The infinitesimal transition probabilities for this model differ from the all-state immigration model, particularly at state 0:

$$\begin{aligned} P[I(t + \Delta t) = 1 \mid I(t) = 0] &= \nu\Delta t + o(\Delta t) \\ P[I(t + \Delta t) = n + 1 \mid I(t) = n] &= n\lambda\Delta t + o(\Delta t), \quad n \geq 1 \\ P[I(t + \Delta t) = n - 1 \mid I(t) = n] &= n\mu\Delta t + o(\Delta t), \quad n \geq 1. \end{aligned}$$

The differential-difference equations for $P_n(t)$ in this model are more complex. $P_n(t) = P(I(t) = n \mid I(0) = I_0)$ satisfies the following differential-difference equations:

$$\begin{aligned} \frac{dP_0(t)}{dt} &= -\nu P_0(t) + \mu P_1(t), \quad n = 0, \\ \frac{dP_1(t)}{dt} &= \nu P_0(t) - (\lambda + \mu)P_1(t) + 2\lambda P_2(t), \quad n = 1, \\ \frac{dP_n(t)}{dt} &= (n - 1)\lambda P_{n-1}(t) - n(\lambda + \mu)P_n(t) + (n + 1)\mu P_{n+1}(t), \quad n \geq 2. \end{aligned} \tag{14}$$

Assume that $I(0) = 1$ and hence $P_1(0) = 1$. Using the probability-generating function approach, we can transform the Kolmogorov forward equations into a partial differential equation:

$$\frac{\partial G(z, t)}{\partial t} = (z - 1) \left[(\lambda z - \mu) \frac{\partial G(z, t)}{\partial z} + \nu P_0(t) \right], \tag{15}$$

with initial condition:

$$G(z, 0) = z, \quad G(1, t) = 1.$$

Finding an exact solution to this equation is not straightforward due to the presence of the $P_0(t)$ term. However, one can find,

$$G(z, t) = \int_0^\zeta \nu \frac{\phi(u)}{1 - \phi(u)} \left(1 - \frac{\mu}{\lambda} \right) p_0(u) du + \zeta.$$

and

$$P_0(t) = \int_0^t \nu \frac{\phi(u)}{1 - \phi(u)} \left(1 - \frac{\mu}{\lambda}\right) P_0(u) du + \frac{1}{1 - \frac{\lambda}{\mu} \exp((\lambda - \mu)t)} - \frac{\mu}{\lambda}. \quad (16)$$

where

$$\phi(\zeta) = \exp((\mu - \lambda)\zeta) \left(\frac{\zeta - 1}{\zeta - \frac{\mu}{\lambda}} \right).$$

This can be used to find the expected value and the variance of the system.

Deriving the mean and variance using the probability generating function is straightforward, and they are given by:

$$E[I(t)] = \exp((\lambda - \mu)t) \left(\int_0^t \nu \exp((\mu - \lambda)u) P_0(u) du + 1 \right);$$

$$\text{Var}[I(t)] = \exp(2(\lambda - \mu)t) \left(\int_0^t (\nu P_0(u)(1 - 2E(u)) + (\lambda + \mu)E(u)) \exp(2(\mu - \lambda)u) du \right).$$

Both expressions involve $P_0(t)$ and cannot be computed exactly; one has to use numerical techniques for Volterra-type integral equations to calculate $P_0(t)$ (see Shiny and Viswanath (2022)). However, a stationary probability distribution exists for this model if $\lambda < \mu$, given by:

$$\pi_0 = \frac{1}{1 - \frac{\nu}{\mu} \ln \left| 1 - \frac{\lambda}{\mu} \right|} \quad (17)$$

$$\pi_n = \frac{\nu}{n\mu} \left(\frac{\lambda}{\mu} \right)^{n-1} \pi_0, \quad n = 1, 2, 3, \dots \quad (18)$$

This model, while also incorporating external infections, captures a different dynamic where the disease can experience periods of extinction followed by reintroduction, making it relevant for understanding the persistence and re-emergence of certain infectious diseases.

5. Model extension: Time-nonhomogeneous parameters

Real-world epidemic scenarios are often characterized by changes in the rates of infection, recovery, and external influences over time due to factors such as public health interventions, changes in population behaviour, and variations in the influx of infected individuals. To better reflect these dynamic conditions, the birth-death all-state immigration model can be extended to incorporate time-nonhomogeneous parameters: $\lambda(t)$, $\mu(t)$, and $\nu(t)$.

For this non-homogeneous model, the expected number of infected individuals at time t , given $I(0) = I_0$, is given by Kobayashi's formula:

$$\bar{I}(t) = I_0 \cdot e^{s(t)} + e^{s(t)} \cdot N(t),$$

where

$$s(t) = \int_0^t [\lambda(u) - \mu(u)] du, \quad N(t) = \int_0^t \nu(u) \cdot e^{-s(u)} du.$$

This extension allows for the analysis of how time-varying rates impact the expected epidemic trajectory. This section next explores three specific scenarios under this framework with constant recovery rate μ : varying λ with constant ν ; varying ν with constant λ ; varying λ and ν .

5.1. Analysis with time-nonhomogeneous infection rate $\lambda(t)$

This scenario considers a situation where the per-capita infection rate $\lambda(t)$ changes over time while the recovery rate μ and immigration rate ν remain constant. This is particularly relevant for modeling the impact of interventions aimed at reducing transmission, such as social distancing measures or lock downs.

This section reproduces an example from Kobayashi, where $\lambda(t)$ is modeled as a decreasing function over a period of time d , representing the time it takes for the public to fully respond to and implement government regulations aimed at reducing contact rates:

$$\lambda(t) = \begin{cases} \lambda_0, & t < t_1 \\ \lambda_1 + (\lambda_0 - \lambda_1) \cdot \frac{1 - \cos(\pi \cdot \frac{t-t_1}{d})}{2}, & t_1 \leq t \leq t_1 + d \\ \lambda_1, & t > t_1 + d, \end{cases}$$

where t_1 is the time at which the intervention is announced, λ_0 is the initial infection rate, and λ_1 is the reduced infection rate. See Figure 8.

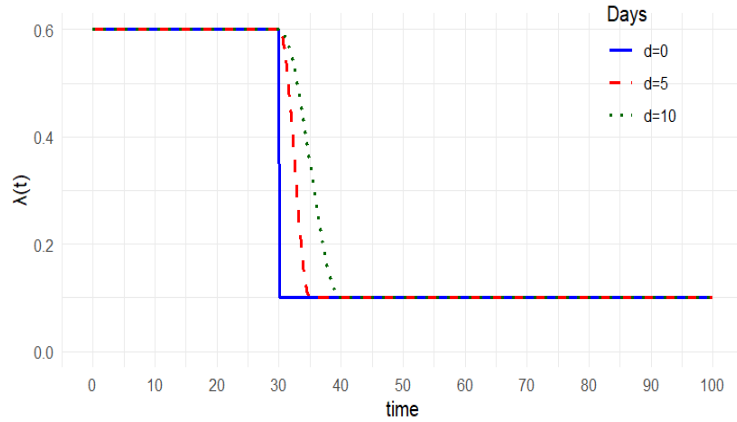


Figure 8: A plot of $\lambda(t)$ from time $t = 0$ to $t = 100$ and d takes values of 0, 5, and 10.

Analysis of the expected number of infected individuals under such a time-varying $\lambda(t)$ demonstrates the significant impact of the delay d in the public's response. See Figure 9. A longer delay leads to a more rapid initial spread and a higher peak in the number of infections. This underscores the critical importance of prompt and effective implementation of control

measures to mitigate the impact of an outbreak. To better reflect realistic scenarios, our model could be extended to implement a finite duration $D > d$ for the control measures, for example by resetting $\lambda(t) = \lambda_0$ for $t \geq t_1 + D$. We expect that infections would rise again after the control period is over, however, perhaps not to the original level, reflecting the impact of the initial controls. This is left for future work.

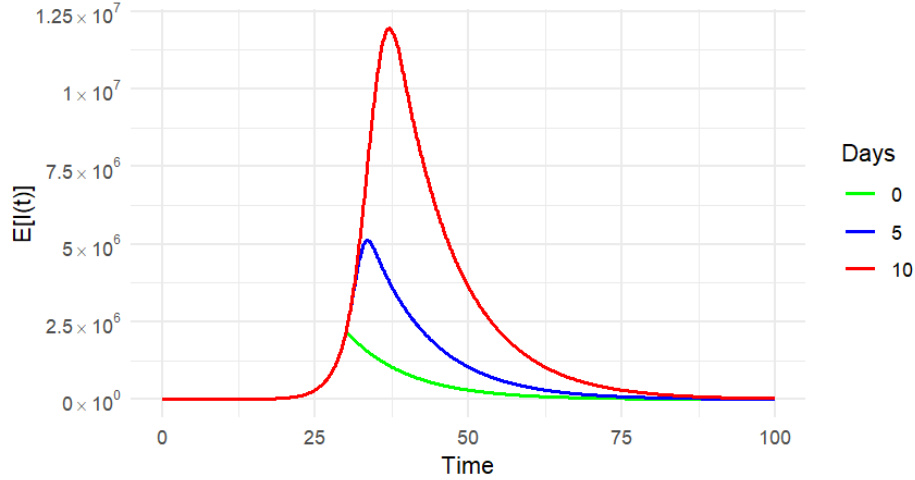


Figure 9: The expected number of infected individuals versus time under the conditions of $\lambda(t)$, $I_0 = 1$, $\nu = 5$, $\mu = 0.2$. The results for different d values are represented by three colours: blue for $d = 0$, green for $d = 5$, and red for $d = 10$.

5.2. Analysis with time-nonhomogeneous immigration rate $\nu(t)$

This scenario examines the effect of a time-varying external infection rate $\nu(t)$ while keeping λ and μ constant. This is relevant for understanding the impact of changing travel patterns, border controls, or the emergence of new external sources of infection.

This section considers $\nu(t)$ modeled by a step function and a piecewise function representing a gradual decline in immigration (see Figure 10):

$$\nu(t) = \begin{cases} \nu_0, & 0 \leq t < t_1 \\ \nu_1, & t \geq t_1 \end{cases} \quad \text{and} \quad \nu(t) = \begin{cases} \nu_0, & t < t_1 \\ \nu_0 + (\nu_1 - \nu_0) \cdot \frac{1 - \cos(\pi \cdot \frac{t - t_1}{d})}{2}, & t_1 \leq t \leq t_1 + d \\ \nu_1, & t > t_1 + d. \end{cases}$$

Analysis of the expected number of infected individuals under these scenarios highlights how changes in the rate of external infections can influence the overall epidemic trajectory. See Figure 11. A sudden reduction in $\nu(t)$ leads to a flattening of the curve and a lower equilibrium level of infection (if $\lambda \leq \mu$). Similar to the case with $\lambda(t)$, delays in implementing measures to reduce external infections have a detrimental effect on controlling the spread. Similarly, our model could be extended to implement a finite duration $D > d$ for travel restrictions, for example by resetting $\nu(t) = \nu_0$ for $t \geq t_1 + D$. Since external infections will rise after the restriction period is over, we also expect the number of infections to rise. This is left for future work.

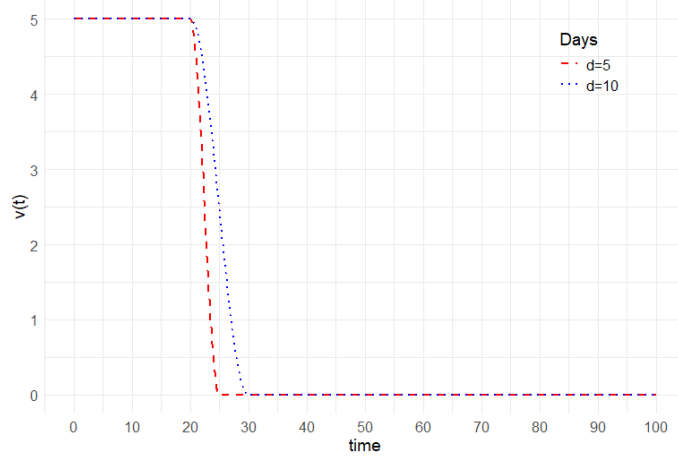


Figure 10: The function $\nu(t)$ transitions from $\nu_0 = 5$ to $\nu_1 = 0.001$ over the interval from $t_1 = 20$ to $t_1 + d = 20 + d$, where d takes values of 5 and 10. The results for different values of d are represented in three colors: red for $d = 5$, and blue for $d = 10$. Note that for $d = 0$, $\nu(t)$ is a step function which drops from $\nu_0 = 5$ to $\nu_1 = 0.001$ at $t = 20$.

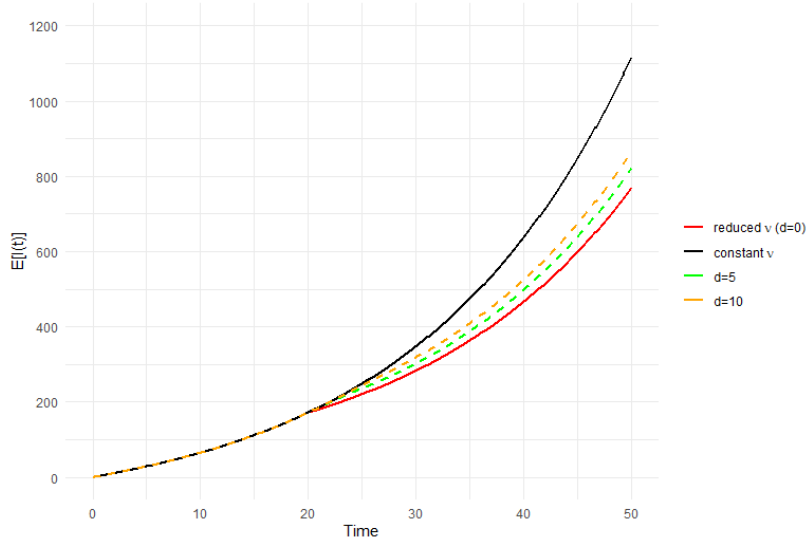


Figure 11: The expected number of infected individuals versus time in the system under the conditions of $\nu(t)$, $I_0 = 0$, $\lambda = 0.65$, $\mu = 0.6$. The results for different d values are represented in three colors: solid red for $d = 0$, dashed green for $d = 5$, and dashed orange for $d = 10$. The solid black curve represents the case where the transmission rate ν is constant at 5 for the entire time.

5.3. Analysis with time-nonhomogeneous $\lambda(t)$ and $\nu(t)$

Finally, in this section, we investigate the scenario where both the per-capita infection rate $\lambda(t)$ and the external infection rate $\nu(t)$ vary over time, while the recovery rate μ remains constant. This provides a more comprehensive model that accounts for the simultaneous effects of internal transmission control measures and changes in external exposures.

Using the same functional forms for $\lambda(t)$ and $\nu(t)$ as in the previous analyses, the expected number of infected individuals is calculated using the general formula for $\bar{I}(t)$. See Figure 12. Comparison of the results with the case where only $\lambda(t)$ is time-varying (Figure 9) suggests that while altering both rates simultaneously has an impact, reducing the internal infection rate $\lambda(t)$ has a more substantial effect on controlling the expected number of infections. This implies that focusing on measures that reduce transmission within the population may be more effective than solely focusing on reducing external introductions.

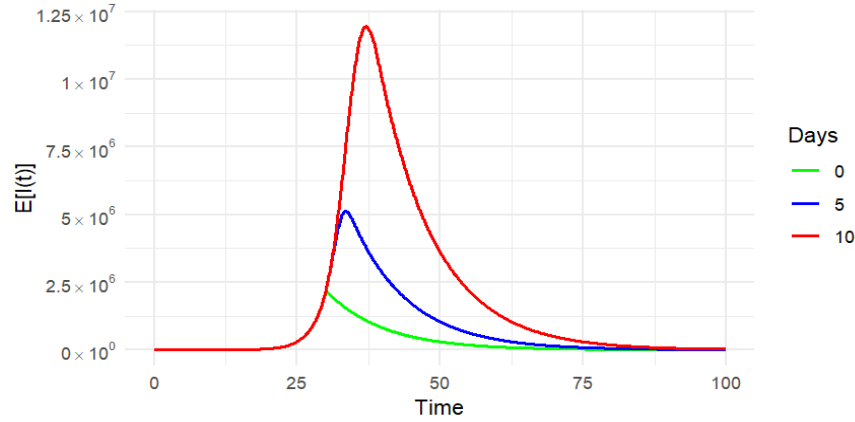


Figure 12: The expected number of infected individuals versus time under the conditions of $I_0 = 0$, $\lambda(t)$, $\nu(t)$, $\mu = 0.2$. The results for different d values are represented in three colors: blue for $d = 0$, green for $d = 5$, and red for $d = 10$.

6. Discussion and conclusion

This paper has explored the application of stochastic models, particularly birth-death and birth-death-immigration processes, to understand the dynamics of infectious disease spread in an open population, emphasizing the role of external infections. The limitations of the foundational deterministic SIR model, including its closed population assumption and inability to capture inherent randomness, necessitate the use of stochastic frameworks for more realistic epidemic modelling. Note that we focus on a single open population and assume it is so large that the number of susceptible individuals can be assumed to be unbounded. We do not consider multiple populations where migration of susceptible individuals could have a significant impact on the epidemic dynamics. Stochastic network models are expected to be suitable for modelling this scenario but that is beyond the scope of the models presented here.

The simple linear birth-death model, while incorporating stochasticity, suffers from the limitation of an absorbing state at disease extinction, which does not reflect the possibility of re-introduction in open populations. The birth-death all-state immigration model addresses this by allowing for a continuous influx of external infections, leading to a probability distribution of the number of infected individuals that follows a generalized negative binomial distribution with a long tail and high variability. This inherent variability, demonstrated through simulations and percentile curves, highlights the unpredictable nature of epidemic patterns and can explain why real-world outbreaks often deviate significantly from

deterministic predictions.

Introducing time-nonhomogeneous parameters into the birth-death all-state immigration model provides a powerful tool for analyzing the impact of changing conditions and interventions on epidemic dynamics. The analysis of varying internal infection rates $\lambda(t)$ underscores the crucial role of prompt and effective public health measures in mitigating outbreaks. Similarly, changes in the external infection rate $\nu(t)$, representing factors like travel restrictions, can significantly influence the epidemic trajectory. Notably, the research suggests that reducing the internal infection rate has a more profound impact on controlling the expected number of infections compared to solely focusing on reducing external introductions.

Much work remains to be done regarding applying birth-death-immigration models to epidemics. **Parameter Estimation:** Develop and apply statistical methods, such as maximum likelihood estimation or the Expectation-Maximization (EM) algorithm, to estimate the parameters of the birth-death all-state immigration stochastic model from real-world epidemiological datasets. This would allow for data-driven predictions of future epidemic behaviour. **Analysis of Cumulative Processes:** Conduct more detailed theoretical studies of the cumulative internal infection process $B(t)$ and the cumulative recovery/death process $R(t)$ within the birth-death-immigration framework to gain further insights into the overall dynamics of the epidemic. **Evaluation of Public Health Interventions:** Extend the time-nonhomogeneous models to explicitly incorporate and evaluate the impact of various public health techniques, such as quarantine strategies and different vaccination approaches, on disease spread and control. **Incorporating Heterogeneity:** Develop stochastic models that account for heterogeneity in population mixing, susceptibility, and infectivity to provide even more realistic representations of disease transmission dynamics. **Application to Specific Diseases:** Apply the developed stochastic models with external infection to analyze the dynamics of specific infectious diseases, utilizing real-world data to validate the models and generate disease-specific predictions.

In conclusion, this paper emphasizes the importance of stochastic modelling, particularly birth-death-immigration models with the capacity to incorporate time-varying parameters, for gaining a deeper understanding of infectious disease dynamics in an interconnected world where external infections are a significant factor. These models offer a more realistic framework for predicting epidemic behavior, assessing the impact of interventions, and informing public health strategies aimed at controlling and mitigating the spread of infectious diseases.

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Conflict of interest

The authors do not have any financial or non-financial conflict of interest to declare for the research work included in this article.

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