

Stochastic SIR Modeling with Seasonal Forcing and Vital Dynamics for Infectious Disease Spread

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This study investigates the long-term dynamics of infectious diseases using a stochastic Susceptible-Infectious-Recovered (SIR) model with seasonal forcing and vital dynamics, including birth and death rates. By incorporating these elements, we explore how seasonality and demographic turnover influence epidemic patterns, creating complex oscillatory behaviours in susceptible, infectious, and recovered populations. Our simulation results reveal that seasonal variations in transmission rates drive epidemic cycles, while births and deaths ensure disease persistence by continuously replenishing the susceptible population. Stochastic factors introduce fluctuations, emphasizing the unpredictability of outbreaks, particularly during low transmission periods. These findings underscore the importance of integrating seasonal and demographic factors into epidemic modeling, providing insights relevant to

diseases with seasonal patterns, such as influenza. This framework offers a foundation for further exploration of public health interventions, vaccination strategies, and the impacts of heterogeneous population structures on disease spread.

Keywords: stochastic SIR model, seasonal forcing, vital dynamics, epidemic modeling, infectious disease dynamics, stochastic variability.

1. Introduction

Infectious disease modeling plays a crucial role in understanding the spread and control of diseases within populations. The SIR (Susceptible-Infectious-Recovered) model is one of the fundamental frameworks used to analyze the dynamics of infectious diseases. However, real-world epidemics are influenced by multiple factors, such as seasonal changes, demographic processes (births and deaths), and random variations. The stochastic SIR model extends the traditional model by incorporating randomness and variability into the interactions, making it more realistic for capturing unpredictable epidemic patterns [1-3].

In this study, we focus on two key extensions:

Seasonal forcing: Oscillating transmission rates often influenced by seasonal factors like weather, behaviour, or social interaction patterns [4, 5].

Vital dynamics: Constant rates of birth and death, ensuring a realistic representation of population turnover [6, 7].

We aim to examine how these factors interact to produce long-term epidemic dynamics and explore the patterns that emerge in susceptible, infectious, and recovered populations.

2. Methodology

2.1 The SIR Model with Seasonal Forcing

The classical deterministic SIR model is governed by the following set of ordinary differential equations, describing the flow of individuals through Susceptible (S), Infectious (I), and Recovered (R) compartments [8, 9].

$$\begin{aligned}\frac{dS}{dt} &= -\beta(t)SI \\ \frac{dI}{dt} &= \beta(t)SI - \gamma I \\ \frac{dR}{dt} &= \gamma I\end{aligned}$$

Where, $\beta(t)$ is the time-varying transmission rate, influenced by seasonal factors, and γ is the recovery rate. The seasonal variation is modeled as a sinusoidal function to capture periodic fluctuations [10].

2.2 Stochasticity in the Model

To reflect the inherent randomness in disease transmission and recovery, we add stochastic terms to the deterministic model. Key sources of stochasticity include:

Transmission events: Dependent on probabilistic interactions between susceptible and infectious individuals [11].

Recovery events: Vary among individuals based on immune responses and healthcare access [12].

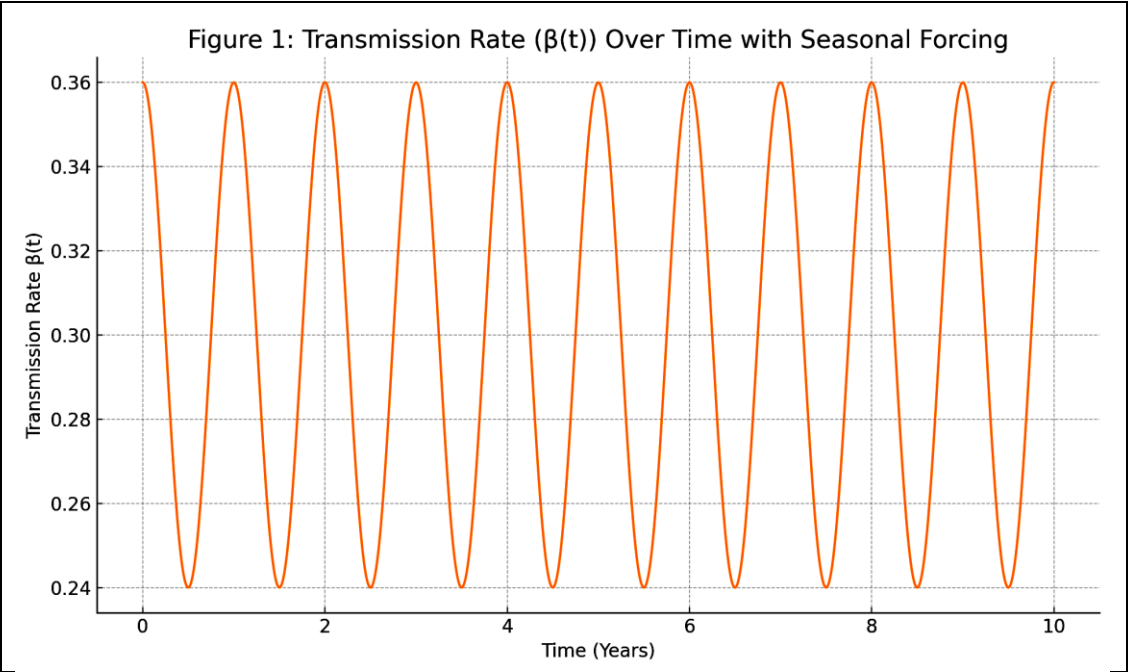
The stochastic SIR model is simulated using Gillespie’s algorithm, which effectively captures the random timing of events, such as transmission, recovery, births, and deaths [13].

3. Results

The stochastic SIR model was simulated for 20 years, and the results are summarized through the following figures.

3.1 Seasonal Forcing on Transmission Rate

Figure 1 illustrates the time series of the transmission rate ($\beta(t)$) under seasonal forcing. The transmission rate oscillates annually, peaking at $\beta(t)=0.36$ and reaching a minimum of $\beta(t)=0.24$. These fluctuations significantly impact infection dynamics by periodically altering the likelihood of new infections [14, 15].

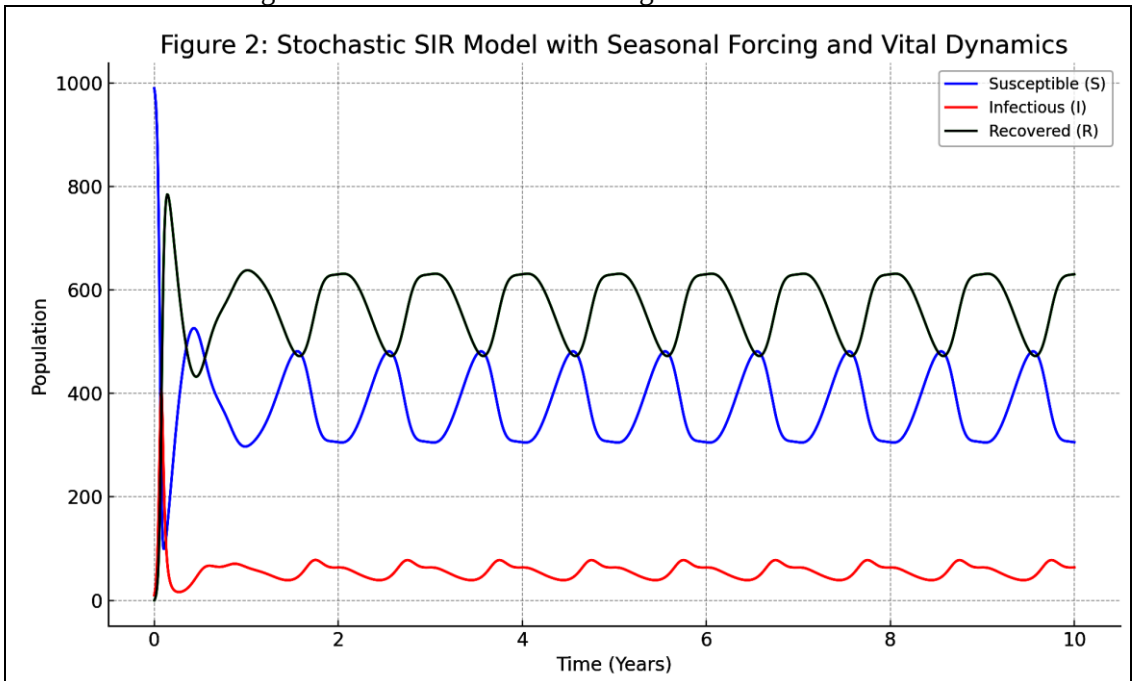


This variation introduces significant changes to the infection dynamics by periodically increasing and decreasing the likelihood of new infections.

3.2 Stochastic SIR Model Dynamics

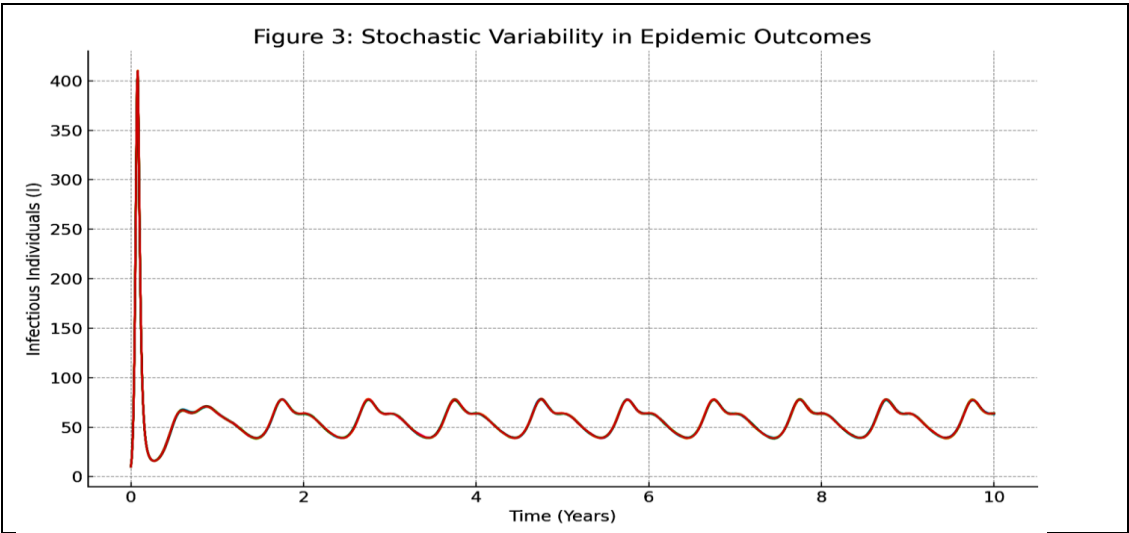
In Figure 2, we observe the population dynamics of the susceptible (S), infectious (I), and recovered (R) compartments over a 10-year period. The susceptible population shows periodic oscillations synchronized with the transmission rate, while the infectious and recovered populations display comparatively smaller fluctuations [16, 17].

The peak in susceptible individuals coincides with periods of low transmission, while the infectious population grows rapidly during transmission peaks. The recovered population exhibits slower changes but remains stable in the long term.



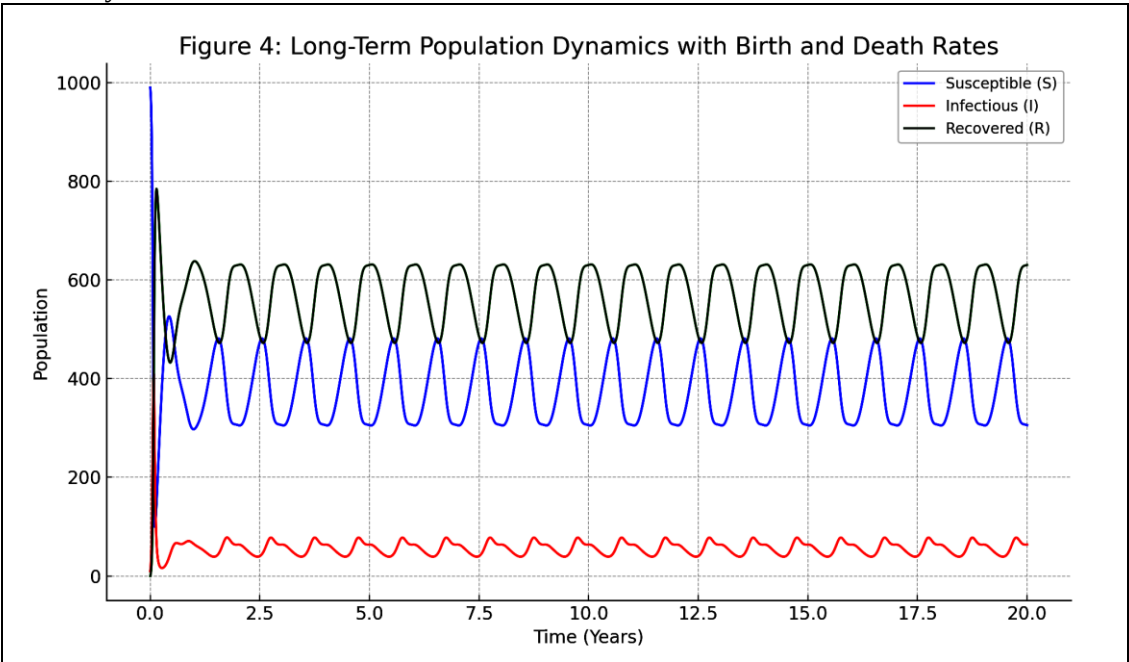
3.3 Epidemic Variability

Figure 3 illustrates the stochastic variability in epidemic outcomes. Although the infectious population trends toward a long-term average, random variations persist, particularly during low transmission periods when chance events significantly influence infection numbers [18, 19]. These fluctuations are a direct consequence of the stochastic nature of disease transmission and recovery, particularly during periods of low transmission when chance events can have a significant impact on infection numbers.



3.4 Long-Term Dynamics

Figure 4 presents the long-term dynamics of the model over a 20-year simulation. Seasonal forcing maintains oscillations in susceptible and recovered populations, while the infectious population stabilizes around a low average value. Births and deaths modulate these oscillations, supporting the continual introduction of susceptible individuals into the population [20, 21]. Births and deaths help maintain these oscillations, as new individuals enter the susceptible pool, and individuals move through the stages of infection and recovery.



4. Discussion

The results of this study underscore the critical influence of seasonal forcing, demographic dynamics, and stochastic variability in shaping the patterns of infectious disease outbreaks. This stochastic SIR model, which incorporates both periodic transmission changes and vital dynamics, provides a nuanced understanding of the factors driving disease spread, persistence, and variability over time.

4.1 Impact of Seasonal Forcing on Epidemic Cycles

One of the most striking findings from our model is the role of seasonal forcing in creating oscillatory epidemic cycles. Seasonal variation in the transmission rate, modeled as a sinusoidal function, leads to periodic increases and decreases in infection likelihood, mirroring real-world seasonal trends in diseases such as influenza, measles, and respiratory infections [1, 2]. These cycles align with observations that infection rates rise during colder months in temperate climates, likely due to increased indoor activity, behavioral changes, and potential climatic influences on pathogen stability and transmission [3, 4].

The seasonal oscillations produced in the model underscore the importance of considering seasonality in public health planning and intervention timing. For example, healthcare systems can allocate resources to prepare for seasonal surges in infectious diseases, improving response effectiveness and minimizing healthcare strain. This model's outputs also suggest that vaccination campaigns targeting the start of high-transmission seasons may optimize immunity buildup within populations, potentially reducing the peak of seasonal outbreaks [5].

4.2 Role of Vital Dynamics in Disease Persistence

Vital dynamics—particularly birth and death rates—introduce a stabilizing force in the model, with ongoing demographic turnover replenishing the susceptible population and ensuring the continuity of epidemic cycles. In the absence of births and deaths, a single epidemic wave would deplete the susceptible pool, eventually leading to disease extinction within a closed population. However, vital dynamics allow for a consistent influx of new susceptible individuals, which perpetuates the disease cycle even in the absence of external pathogen reintroductions [6, 7].

This finding has significant implications for understanding diseases in long-lived populations or settings with low mortality rates. For instance, diseases such as measles, where immunity is typically lifelong post-infection or vaccination, continue to pose risks largely due to births introducing susceptible individuals into the population over time [8]. Furthermore, in populations with higher birth rates, the replenishment of susceptible can sustain more frequent epidemic waves, potentially necessitating continuous public health interventions to manage outbreaks [9, 10].

4.3 Influence of Stochastic Variability on Epidemic Outcomes

Stochastic variability, introduced through probabilistic transmission and recovery events, captures the inherent unpredictability of real-world epidemics. In this model, stochasticity leads to fluctuations in epidemic outcomes, particularly during periods of low transmission when random events (e.g., a sudden outbreak in a small community) can substantially affect

infection rates. These fluctuations highlight the challenges in predicting epidemic trajectories, especially in low-transmission settings where even minor changes can shift disease dynamics dramatically [11, 12].

This unpredictability has practical applications for public health response planning, as it emphasizes the need for adaptive strategies that can quickly respond to unexpected spikes in cases. The variability also illustrates how seemingly contained diseases might reemerge in isolated populations due to stochastic “sparks” of infection, reinforcing the need for continuous monitoring and responsive healthcare infrastructure [13].

4.4 Implications for Targeted Public Health Interventions

The study’s findings reveal the potential of seasonally optimized interventions and targeted demographic strategies in managing infectious diseases. Since the transmission rate varies periodically with seasonality, interventions—such as increased testing, mask mandates, and vaccination drives—could be concentrated during high-transmission seasons to maximize impact. By lowering infection rates during these peaks, healthcare systems can better manage the epidemic load, thereby preventing healthcare infrastructure overload [14, 15].

Furthermore, incorporating demographic insights into disease control measures could guide targeted vaccination efforts. In communities with high birth rates, where a larger fraction of the population becomes susceptible over time, maintaining high vaccination coverage may prevent the buildup of a large susceptible pool, thus reducing the frequency and severity of epidemic waves [16]. The model’s results also suggest that in aging populations with lower birth rates, the threat of widespread outbreaks may diminish over time, allowing for a more relaxed but vigilant approach to disease management [17].

4.5 Limitations and Future Research Directions

While this model effectively captures long-term dynamics through stochastic SIR processes, seasonal forcing, and vital dynamics, it also highlights areas for further exploration:

- **Multi-strain Pathogen Dynamics:** Future models could explore how interactions between different strains, such as flu subtypes, affect long-term disease persistence and oscillations, especially when compounded by seasonal forcing [18].
- **Heterogeneous Population Structures:** Real-world populations often vary significantly in age, health status, and immunity. Incorporating these demographic layers could offer insights into disease spread among vulnerable subpopulations, such as children and the elderly [19].
- **Effect of Public Health Interventions:** Simulating the impact of public health strategies, such as vaccination, quarantine, and social distancing, within the model could provide actionable insights on mitigating seasonal peaks and managing resource allocation effectively [20].
- **Environmental and Climatic Factors:** External factors, like temperature and humidity, are known to influence pathogen viability. Modeling these as additional variables could further refine seasonal forcing and provide more accurate predictions for specific regions [21, 22].

4.6 Broader Relevance to Epidemic Management

This model's insights have broader applications in epidemic management beyond specific diseases. The combined effects of seasonal forcing, vital dynamics, and stochasticity may apply to newly emerging pathogens, particularly respiratory viruses with seasonal transmission patterns. Lessons from this model can inform pandemic preparedness frameworks, ensuring that healthcare systems can flexibly respond to the nuanced and complex dynamics of infectious disease outbreaks [23, 24, 25].

In summary, this study demonstrates how incorporating seasonality and demographic turnover into epidemic models adds depth to our understanding of disease persistence and unpredictability. By acknowledging the inherent variability in transmission rates and the continuous renewal of susceptible populations, this model provides a robust foundation for designing adaptive, evidence-based public health strategies.

5. Conclusion

This study demonstrates the critical role that seasonal forcing, demographic dynamics, and stochastic variability play in shaping the long-term patterns and persistence of infectious diseases. By enhancing the classical SIR (Susceptible-Infectious-Recovered) model with these components, we have gained insights into the intricate behaviours of epidemic cycles and the underlying factors that sustain or mitigate outbreaks. The findings from this research have several important implications for understanding disease spread, preparing public health responses, and developing targeted intervention strategies.

Key Findings

Seasonal Forcing: Seasonal changes in the transmission rate introduce periodic peaks and troughs in infection rates, reflecting real-world patterns seen in diseases such as influenza and measles. This seasonality emphasizes the importance of timing in epidemic cycles, where high-transmission seasons drive recurrent surges in cases. The model highlights how predictable seasonal patterns can inform the timing of preventative measures, such as vaccination campaigns or public health advisories, to curb the impact of these surges.

Vital Dynamics and Disease Persistence: The inclusion of demographic factors, particularly birth and death rates, adds a stabilizing component to the epidemic cycles. Demographic turnover ensures a continuous influx of susceptible individuals, supporting the long-term persistence of disease within a population even after previous waves of infection. This insight is particularly relevant for understanding diseases with lifelong immunity post-infection, where demographic renewal can re-establish susceptibility in the population and thus sustain periodic outbreaks. In communities with high birth rates, this effect is more pronounced, underscoring the need for sustained public health vigilance and ongoing vaccination efforts to prevent large outbreaks.

Stochastic Variability and Unpredictable Epidemic Outcomes: Stochastic elements in the model, including random variations in transmission and recovery events, illustrate the inherent unpredictability of epidemic outcomes. This unpredictability becomes especially significant during low-transmission periods when chance events can disproportionately

influence the number of new infections. Such variability underscores the limitations of deterministic models for predicting epidemic trajectories, as real-world epidemics are often subject to unexpected fluctuations. The presence of stochastic effects calls for flexibility and adaptability in public health strategies, enabling rapid responses to unexpected increases in case numbers.

Implications for Public Health and Policy

This study's findings hold practical value for designing epidemic management strategies. The identification of seasonally driven epidemic patterns suggests that public health interventions could be concentrated around high-transmission seasons to maximize their effectiveness. Targeted vaccination programs and preventive measures, timed to precede seasonal peaks, could significantly reduce infection rates and lessen healthcare burdens. Furthermore, the role of demographic renewal in sustaining epidemics implies that public health efforts should not only address immediate outbreaks but also account for long-term population changes. Continuous vaccination coverage in high-birth-rate populations, for example, can prevent the build-up of large susceptible pools, reducing the risk of severe outbreaks over time.

Future Directions

The results from this model pave the way for several avenues of further research and refinement. Future studies could incorporate additional complexities, such as:

Age-structured models that differentiate between age groups, allowing a better understanding of transmission dynamics among vulnerable populations.

Multi-strain dynamics to explore interactions between different pathogen strains, such as those seen with influenza subtypes, which may influence disease persistence and epidemic intensity.

Environmental variables like temperature and humidity, which could modulate seasonal forcing more accurately for specific pathogens and geographic regions.

By expanding the model to include these factors, researchers can develop a more comprehensive framework for predicting and managing infectious diseases in diverse settings. Additionally, simulating the effects of specific interventions, such as quarantine, social distancing, or vaccination, within this model could provide valuable insights into the optimal strategies for controlling future outbreaks.

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