

Hybrid deterministic-stochastic modeling of typhoid fever using jump diffusion for epidemic control and decision support

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ABSTRACT

A hybrid deterministic-stochastic (jump-diffusion) model for typhoid fever dynamics, addressing the limitations of classical deterministic models in capturing random environmental fluctuations and sudden outbreak events is presented. The model extends a deterministic seven-compartment framework by introducing both Brownian motion and discrete (Poisson jump) stochastic perturbations in the infected, treated, and recovered compartments. Analytical results establish the positivity and well-posedness of solutions, derive explicit expressions for disease-free and endemic equilibria, conditions for reducible nonlinear jump-endemic model. The basic reproduction numbers for both deterministic (R_0) and stochastic (R_{jp}) systems were established. Sensitivity analysis using normalized indices and Partial Rank Correlation Coefficient (PRCC) methods identifies the symptomatic transmission rate (β_1), vaccination rate (ν), and recovery/treatment rates (γ_1, δ) as the most influential parameters affecting R_0 . PRCC results show β_1 and α have the strongest positive impact on R_0 , while γ_1 and ν have strong negative effects, highlighting the importance of vaccination and treatment in disease control. Numerical simulations demonstrate that increasing ν or γ_1 can reduce R_0 below unity, leading to disease eradication, while higher β_1 or positive jump intensities can sustain outbreaks even under control measures. The stochastic reproduction number R_{jp} is shown to decrease with stronger diffusion but increase with higher jump intensity, emphasizing the need for adaptive, non-constant interventions. The jump-diffusion framework thus provides a robust tool for epidemic decision support under uncertainty.

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Key words: Typhoid fever; Jump-diffusion process; Stochastic differential equation; Reproduction Number; Disease control under Uncertainty

1 Introduction

Communicable diseases ([11, 12, 18, 21, 23, 66]) remain significant threats to human populations, with profound consequences for public health and socioeconomic well-being. Coronavirus disease ([12]), typhoid fever ([33]), tuberculosis ([61]), and HIV/AIDS ([47]) are among the infectious diseases that have adversely affected human lives. Typhoid fever, in particular, remains a major global public health concern. Caused by the bacterium *Salmonella enterica* serovar Typhi (*Salmonella* Typhi), the disease poses a substantial threat, particularly in low- and middle-income countries where access to safe drinking water and adequate sanitation is limited [22, 24, 30, 63]. Typhoid fever is associated with considerable morbidity and mortality [40, 49, 54] and is transmitted predominantly through the consumption of food and water contaminated with *S. Typhi*. Despite advances in antimicrobial treatment and vaccination, periodic outbreaks continue to occur, driven by environmental, socioeconomic, behavioural, and infrastructural factors ([17, 64]).

Mathematical modelling has emerged as an effective tool for investigating the transmission dynamics of typhoid fever ([29, 35, 41]) and evaluating the effectiveness of disease-control interventions. Deterministic epidemiological models ([7, 26]) provide a systematic framework for analysing disease dynamics through epidemiological compartments such as susceptible, exposed, infected, and recovered populations. These models generally assume continuous population dynamics and parameter values, thereby providing useful insights into long-term disease trends and facilitating the evaluation of public health policies. However, disease transmission in real-world settings is influenced by inherent randomness, abrupt outbreak events, and unforeseen external factors. In the case of typhoid fever, for example, sudden contamination of food or water sources, population movement, changes in human behaviour, and environmental variability can produce substantial deviations from the average transmission dynamics predicted by deterministic models.

Stochastic modelling provides a natural framework for incorporating such uncertainties into the analysis of infectious diseases. By explicitly accounting for random and probabilistic factors, stochastic models can provide a more realistic representation of temporal variations in epidemic dynamics. In particular, jump-diffusion models, which combine continuous Gaussian noise driven by Brownian motion with discontinuous non-Gaussian jump processes, provide a suitable framework for representing both gradual random fluctuations and sudden changes in disease transmission. The diffusion component can capture small, continuous variations arising from environmental and behavioural uncertainties, whereas the jump component can represent abrupt increases in infection resulting from events such as environmental contamination, population movement, or failures in disease-control measures. Integrating deterministic and stochastic components can therefore provide a more comprehensive understanding of typhoid fever dynamics and enhance the assessment of intervention strategies [16, 34, 67].

Numerous studies ([16, 24, 32, 34, 43, 56, 59, 60, 67]) have investigated the transmission dynamics of infectious diseases using deterministic mathematical models. In the context of typhoid fever, these models have been widely employed to examine disease transmission and identify effective strategies for reducing its burden. Despite these efforts, typhoid fever continues to pose a substantial threat to public health. Lawal et al. (2024) [33] and Tijani et al. (2023) [58], for example, developed deterministic compartmental optimal-control models incorporating hygiene and environmental factors, including environmental sanitation, personal hygiene, and treatment. Their findings indicated that combining these interventions was more effective than implementing individual control measures. Building on the need for broader intervention strategies, Asamoh et al. (2025) [14] incorporated five control measures—vaccination, booster vaccination, hygiene, treatment, and bacterial sterilisation—into a deterministic typhoid fever model. Their results demonstrated that a combined intervention strategy was more effective than the use of a single intervention.

Fractional-order deterministic models have also been employed to investigate the transmission dynamics of typhoid fever ([6, 27, 46, 55]). The mathematical formulations developed in these studies have been investigated using various analytical and numerical approaches, with numerical validation reported in [6, 19, 25]. These studies demonstrate the considerable development of deterministic approaches to modelling typhoid fever and provide a strong foundation for further extensions incorporating stochastic effects.

Although deterministic models of typhoid fever are well established in the existing literature, stochastic approaches remain comparatively limited. Existing stochastic infectious-disease models can broadly be classified into three categories. The first comprises agent-based stochastic simulation models, such as [53], which represent individual-level interactions and can capture random infection events, heterogeneity in transmission, and stochastic disease persistence. The second comprises stochastic differential-equation models, such as [45], in which stochasticity is incorporated into the disease dynamics through transition probabilities and Markovian processes. The third category consists of hybrid artificial-intelligence-based stochastic computational models, such as [51, 52], which combine mathematical or stochastic models with artificial intelligence techniques for predictive purposes.

Typhoid fever differs from diseases that typically exhibit a single, isolated epidemic episode because its transmission can persist endemically, particularly in regions where inadequate sanitation, limited access to safe water, and environmental contamination remain prevalent. Consequently, recurrent outbreaks and localised infection spikes may arise from sudden contamination events and other environmental or behavioural factors ([37, 42, 57]). While stochastic jump-diffusion approaches have been increasingly applied to the modelling of COVID-19 ([56]), malaria ([5]), and financial systems ([3, 31, 38]), their application to the transmission dynamics of typhoid fever remains limited. To the best of our knowledge, there is a paucity of studies that explicitly incorporate jump-diffusion processes into compartmental models of typhoid fever transmission. This gap motivates the development of a modelling framework capable of representing both continuous stochastic fluctuations and abrupt epidemiological shocks in typhoid transmission.

In this study, we develop a hybrid deterministic-jump-diffusion epidemiological model for typhoid fever that incorporates continuous random fluctuations and sudden outbreak events. The Brownian-motion component represents small, continuous variations in disease dynamics, whereas the jump component represents abrupt epidemiological shocks associated with environmental contamination events, such as sudden contamination of water sources. Stochastic perturbations are incorporated into the infected, treated, and recovered compartments to account for variability in disease progression and intervention outcomes. The deterministic component captures the average behaviour of the disease-transmission process, while the stochastic jump-diffusion component accounts for random fluctuations and sudden outbreak events. By simulating the model under standard control measures, including vac-

cination and treatment, the effectiveness of these interventions is evaluated under both deterministic and stochastic settings. Unlike optimal-control formulations, the present study focuses on the intrinsic effects of stochastic perturbations on disease dynamics and the resulting implications for disease persistence and control.

The study contributes to the literature on infectious-disease modelling by integrating deterministic and stochastic approaches within a unified framework for analysing typhoid fever transmission. The proposed model provides a mechanism for assessing the effects of continuous random fluctuations, sudden outbreak events, and variability in intervention effectiveness on disease transmission and control. Such a framework can provide useful insights for the development of adaptive public health strategies in settings characterised by environmental and epidemiological uncertainty.

Specifically, the study investigates the influence of deterministic and stochastic factors on typhoid fever transmission dynamics and examines the roles of vaccination, treatment, and recovery in disease control under both deterministic and jump-diffusion settings. It further evaluates the effects of outbreak-induced perturbations and variations in intervention effectiveness on the long-term behaviour of the disease. In addition, local stability analysis based on the Jacobian matrix is employed to establish conditions associated with disease eradication and persistence.

To achieve these objectives, a seven-compartment deterministic-jump-diffusion model is formulated by incorporating both Gaussian noise, represented by Brownian motion, and non-Gaussian jump noise, represented by a Poisson process, into the infected, treated, and recovered compartments. The Gaussian component captures continuous random fluctuations, whereas the jump component represents sudden outbreak events associated with environmental contamination and other epidemiological shocks. For the deterministic subsystem, the positivity, feasibility, and well-posedness of the model are established in the absence of stochastic perturbations.

Furthermore, the disease-free and endemic equilibrium states are derived, and the conditions governing the existence and uniqueness of the endemic equilibrium are established. The basic reproduction number and local stability properties of the model are determined to assess the conditions governing disease persistence and extinction. In the presence of stochastic variability arising from Brownian fluctuations and jump perturbations in the infected, treated, and recovered compartments, conditions under which the nonlinear stochastic jump-diffusion typhoid model can be reduced are established. This reduction facilitates the transformation of the nonlinear stochastic differential system into an equivalent linear form. Subsequently, an explicit analytical solution of the resulting linearised jump-diffusion system is derived using Itô's formula. Numerical simulations are then conducted under two distinct scenarios to illustrate and validate the theoretical results.

The remainder of this paper is organised as follows. Section 2 presents the materials and methods, including the formulation of the deterministic and stochastic models and their analytical properties. Section 3 presents the sensitivity analysis of the model parameters. Section 4 presents the numerical simulations and discusses the results, while Section 5 provides the main conclusions and implications of the study.

2 Materials and Methods

2.1 Formulation of Deterministic Typhoid Model

The deterministic SVEIATR model divides the population into seven compartments: Susceptible individuals (S), Vaccinated individuals (V), Exposed individuals (E), Infected individuals (I), Asymptomatic infected individuals (A), Treated individuals (T), and Recovered individuals (R). The total population is $N = S + V + E + I + A + T + R$. The model maintain the intuition of SEIR with an open population in which recruitment and exist is been allowed into the population over time with the idea of system involving Ordinary differential Equations (ODEs) and mass action incidence (Bilinear). In this model, Susceptible compartment S increases through the recruitment rate Λ and quantities ϕR , αV , where ϕ is rate of immunity loss and α is vaccine wane rate. The compartment decreased due to the forces of infection denoted by λS , also by the quantities νS and μS , where λ is the force of infection, ν , is vaccination rate and μ , the natural death rate. Hence, the rate of change involving the susceptible sub-population is given by

$$\frac{dS}{dt} = \Lambda - (\lambda + \mu + \nu)S + \alpha V + \phi R \quad (2.1)$$

The sub-population of vaccinated individuals is increased by the quantities νS , while it is decreased by the quantity μV and αV , where α is vaccine wane rate. Hence, the rate of change of sub-population of vaccinated individuals is given by

$$\frac{dV}{dt} = \nu S - (\alpha + \mu)V. \quad (2.2)$$

The sub-population of exposed human individuals is increased by the quantities λS , while it is decreased by the quantity μE_h , $\tau \sigma E$ and $(1 - \tau)\sigma E$, where σ is progression rate from exposed to the infectious compartment, τ is probability of exposed individuals becomes symptomatic infected and $(1 - \tau)$ is probability of exposed individuals becomes asymptomatic infected. Hence, the rate of change of sub-population of exposed human individuals is given by

$$\frac{dE}{dt} = \lambda S - \tau \sigma E - (1 - \tau)\sigma E - \mu E. \quad (2.3)$$

The sub-population of infected human is increased by the quantity $\tau\sigma E$ and decreased by the quantities δI , $\gamma_1 I$, μI and $\mu_2 I$, where δ_h is treatment rate for infected humans, γ_1 , recovery rate of infected humans, and μ_2 deaths through the infection. Hence, the rate of change of the infected human compartment is given by

$$\frac{dI}{dt} = \tau\sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I \quad (2.4)$$

The sub-population of asymptomatic infected human is increased by the quantity $(1 - \tau)\sigma E$ and decreased by the quantities $\gamma_2 A$ and μA , where γ_1 , is the recovery rate of asymptomatic infected humans. Hence, the rate of change of the asymptomatic infected human compartment is given by

$$\frac{dA}{dt} = (1 - \tau)\sigma E - (\gamma_2 + \mu)A. \quad (2.5)$$

The sub-population of treated individual humans is increased by the quantity δI and decreased by the quantities μT and $\gamma_3 T$, where γ_3 denotes the human recovery rate through treatment. Hence, the rate of change of the treatment compartment is given by

$$\frac{dT}{dt} = \delta I - (\gamma_3 + \mu)T. \quad (2.6)$$

The sub-population of recovered humans is increased by the quantities $\gamma_1 I$, $\gamma_2 A$, $\gamma_3 T$ and reduced by the quantities μR and ϕR . Hence, the rate of change of the recovery compartment is given by

$$\frac{dR}{dt} = \gamma_1 I + \gamma_2 A + \gamma_3 T - (\mu + \phi)R. \quad (2.7)$$

Taking into account the above considerations, the schematic flow diagram for the Typhoid model is shown in Figure

Table 1: Description of state variables

Symbol	Description
S	Susceptible humans
V	Vaccinated humans
E	Exposed humans
I	Infected humans
A	Asymptomatic humans
T	Treated humans
R	Recovered humans

Table 2: Description of parameters in the deterministic case

Symbol	Description
Λ	Recruitment rate
β_1	Transmission rate of Symptomatic infectious
β_2	Transmission rate of Asymptomatic infectious
τ	probability that exposed individuals become symptomatic infected
ω	Modification parameter
σ	Rate of progression from exposed to infected
γ_1	Recovery rate of symptomatic individuals
γ_2	Recovery rate of asymptomatic individuals
γ_3	Recovery rate of individuals through treatment
δ	Treatment rate of symptomatic individuals
ϕ	rate of being susceptible again
μ	Natural death rate
μ_2	death rate through infection
α	Vaccine wane rate
ν	Vaccination rate

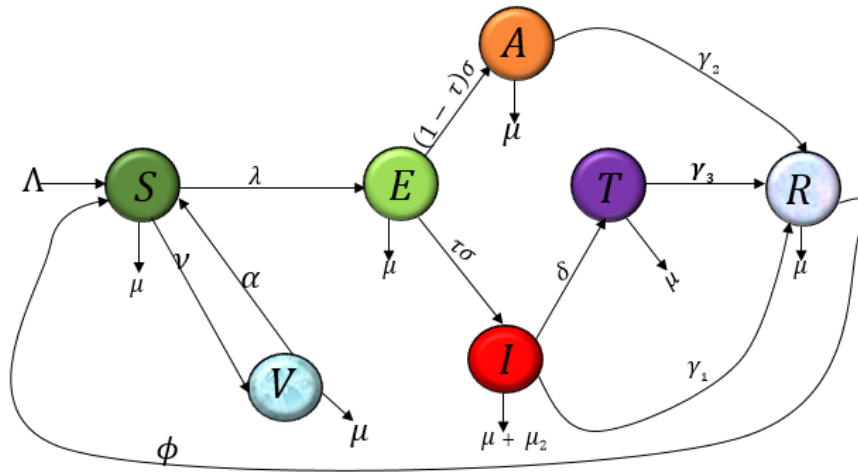


Figure 1: Schematic Diagram for Typhoid Fever

The deterministic model is governed by the following system of ordinary differential equations:

$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda - (\lambda + \mu + \nu)S + \alpha V + \phi R, \\
 \frac{dV}{dt} &= \nu S - (\alpha + \mu)V, \\
 \frac{dE}{dt} &= \lambda S - \tau\sigma E - (1 - \tau)\sigma E - \mu E, \\
 \frac{dI}{dt} &= \tau\sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I, \\
 \frac{dA}{dt} &= (1 - \tau)\sigma E - (\gamma_2 + \mu)A, \\
 \frac{dT}{dt} &= \delta I - (\gamma_3 + \mu)T, \\
 \frac{dR}{dt} &= \gamma_1 I + \gamma_2 A + \gamma_3 T - (\mu + \phi)R.
 \end{aligned} \tag{2.8}$$

The force of infection is defined as

$$\lambda = \frac{\beta_1 I + \omega \beta_2 A}{N},$$

where β_1 and β_2 are the transmission rates of symptomatic and asymptomatic individuals, respectively, and ω is a modification factor.

2.2 Formulation of Deterministic-Jump diffusion Typhoid Model

The deterministic-stochastic SVEIATR model incorporates both Gaussian (Brownian motion) and non-Gaussian (jump process) noise to capture random fluctuations and sudden outbreaks. The model consists of seven compartments, Susceptible individuals, Vaccinated individuals, Exposed individuals, Infected individuals, Asymptomatic infected individuals, Treated individuals and Recovered individuals (SVEIATR):

$$\begin{aligned}
 dS &= (\Lambda - (\lambda + \mu + \nu)S + \alpha V + \phi R) dt, \\
 dV &= (\nu S - (\alpha + \mu)V) dt, \\
 dE &= (\lambda S - (\sigma + \mu)E) dt, \\
 dI &= (\tau\sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I) dt + \eta_I IdW_t + J_I IdN_t, \\
 dA &= ((1 - \tau)\sigma E - (\gamma_2 + \mu)A) dt, \\
 dT &= (\delta I - (\gamma_3 + \mu)T) dt + \eta_T TdW_t + J_T TdN_t, \\
 dR &= (\gamma_1 I + \gamma_2 A + \gamma_3 T - (\mu + \phi)R) dt + \eta_R RdW_t + J_R RdN_t.
 \end{aligned} \tag{2.9}$$

2.2.1 Description of state variables and parameters in the jump-diffusion case

Stochastic perturbations are introduced only in the infected, treated, and recovered compartments to capture random fluctuations in disease progression, treatment response, and immunity dynamics. These compartments are

Symbol	Description
η_I, η_R, η_T	Volatility terms for Brownian motion in the infected, treated and recovered population.
J_I, J_R, J_T	Jump amplitudes for non-Gaussian noise in the infected, treated and recovered population.

Table 3: Description of state variables and para in the jump-diffusion case.

particularly sensitive to environmental variability and sudden outbreak events. Thus, we introduce stochastic effects through Gaussian white noise and jump processes—representing continuous environmental variability and sudden outbreak shocks, respectively. While the susceptible, vaccinated, and exposed populations evolve deterministically due to their relatively stable population sizes.

2.3 Dynamic analysis and solutions of the deterministic typhoid model

A solution of the following deterministic typhoid model is given in this section.

$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda - (\lambda + \mu + \nu)S + \alpha V + \phi R, \\
 \frac{dV}{dt} &= \nu S - (\alpha + \mu)V, \\
 \frac{dE}{dt} &= \lambda S - \tau \sigma E - (1 - \tau)\sigma E - \mu E, \\
 \frac{dI}{dt} &= \tau \sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I, \\
 \frac{dA}{dt} &= (1 - \tau)\sigma E - (\gamma_2 + \mu)A, \\
 \frac{dT}{dt} &= \delta I - (\gamma_3 + \mu)T, \\
 \frac{dR}{dt} &= \gamma_1 I + \gamma_2 A + \gamma_3 T - (\mu + \phi)R.
 \end{aligned} \tag{2.10}$$

2.3.1 Feasibility Solution of the model

$$\begin{aligned}
 \frac{dN}{dt} &= \Lambda - \mu(S + V + E + I + A + T + R) - \delta I - A \\
 &= \Lambda - \mu N - \mu_2 I
 \end{aligned}$$

If $\mu_2 = 0$

$$\therefore, \quad \frac{dN}{dt} = \Lambda - \mu N \tag{2.11}$$

On solving (2.11) we have

$$N(t) \leq \frac{\Lambda}{\mu} \tag{2.12}$$

2.3.2 Positivity of the solution

From the first equation in (2.8)

$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda - (\lambda + \mu + \nu)S + \alpha V + \phi R \\
 \frac{dS}{dt} &\geq -(\lambda + \mu + \nu)S
 \end{aligned} \tag{2.13}$$

After solving (2.13) we have

$$S(t) \geq S(0)e^{-(\lambda + \mu + \nu)t} \geq 0 \quad \text{since } (\lambda + \mu + \nu) > 0 \tag{2.14}$$

Similarly to other variables, the state variables are positive at $t \rightarrow \infty$

2.3.3 Typhoid - free Equilibrium

From system (2.8) at disease-free equilibrium, setting $I^+ = 0, E^+ = I^+ = A^+ = T^+ = R^+ = 0$ it is reduced to

$$\begin{aligned}
 \Lambda - (\mu + \nu)S^+ + \alpha V &= 0 \\
 \nu S^+ - (\alpha + \mu)V &= 0
 \end{aligned} \tag{2.15}$$

In solving (2.15) simultaneously
From the second equation, we have

$$V^+ = \frac{\nu S^+}{(\mu + \nu)} \quad (2.16)$$

Putting (2.16) into first equation of (2.15) we have

$$S^+ = \frac{\Lambda(\alpha + \mu)}{\mu(\alpha + \mu + \nu)} \quad (2.17)$$

Replace S^+ in (2.16) with (2.17) to obtain

$$V^+ = \frac{\Lambda\nu}{\mu(\alpha + \mu + \nu)} \quad (2.18)$$

Hence, the Typhoid-Free equilibrium is $\mathbb{E}^+ = (\frac{\Lambda(\alpha + \mu)}{\mu(\alpha + \mu + \nu)}, \frac{\Lambda\nu}{\mu(\alpha + \mu + \nu)}, 0, 0, 0, 0, 0)$

2.3.4 Typhoid endemic equilibrium

Here, the condition for the existence and uniqueness of the endemic equilibrium of model system equations (2.8) is determined. Endemic equilibrium state is the state where the disease cannot be totally eradicated but persist in the population. Then, all the state variables in 2.8 must not be zero at this equilibrium state

In order to obtain the endemic equilibrium state, each equation (2.8) is equated to zero and solve it simultaneously

$$\begin{aligned} \Lambda - (\lambda + \mu + \nu)S^* + \alpha V^* + \phi R^* &= 0, \\ \nu S^* - (\alpha + \mu)V^* &= 0, \\ \lambda S^* - \tau\sigma E^* - (1 - \tau)\sigma E^* - \mu E^* &= 0, \\ \tau\sigma E^* - (\gamma_1 + \delta + \mu_2 + \mu)I^* &= 0, \\ (1 - \tau)\sigma E^* - (\gamma_2 + \mu)A^* &= 0, \\ \delta I^* - (\gamma_3 + \mu)T^* &= 0, \\ \gamma_1 I^* + \gamma_2 A^* + \gamma_3 R^* - (\mu + \phi)R^* &= 0. \end{aligned} \quad (2.19)$$

to obtain

$$\mathbb{E}^* = \begin{cases} S^* = \frac{\Lambda a_5 a_3 a_7}{[a_3 a_7 (a_5 a_6 + \mu\nu) - \lambda\phi\tau\sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta\tau a_3)]} \\ V^* = \frac{\Lambda\nu a_3 a_7}{[a_3 a_7 (a_5 a_6 + \mu\nu) - \lambda\phi\tau\sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta\tau a_3)]} \\ E^* = \frac{\Lambda\lambda a_5 a_3}{[a_3 a_7 (a_5 a_6 + \mu\nu) - \lambda\phi\tau\sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta\tau a_3)]} \\ I^* = \frac{\Lambda\lambda\tau\sigma a_5}{[a_3 a_7 (a_5 a_6 + \mu\nu) - \lambda\phi\tau\sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta\tau a_3)]} \\ A^* = \frac{\Lambda\lambda\tau\sigma a_5 (1 - \tau) a_3}{\tau a_2 [a_3 a_7 (a_5 a_6 + \mu\nu) - \lambda\phi\tau\sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta\tau a_3)]} \\ T^* = \frac{a_3 [a_3 a_7 (a_5 a_6 + \mu\nu) - \lambda\phi\tau\sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta\tau a_3)]}{\Lambda\lambda a_5 \sigma [\gamma_1 \tau a_1 a_2 + \gamma_2 a_1 (1 - \tau) a_2 + \gamma_3 \delta\tau a_3]} \\ R^* = \frac{a_1 a_2 a_4 [a_3 a_7 (a_5 a_6 + \mu\nu) - \lambda\phi\tau\sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta\tau a_3)]}{a_1 a_2 a_4 [a_3 a_7 (a_5 a_6 + \mu\nu) - \lambda\phi\tau\sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta\tau a_3)]} \end{cases} \quad (2.20)$$

where,

$$\begin{aligned} a_1 &= \gamma_3 + \mu, a_2 = \gamma_2 + \mu, a_3 = \gamma_1 + \delta + \mu_2 + \mu, \\ a_4 &= \mu + \phi, a_5 = \alpha + \mu, \\ a_6 &= \lambda + \mu, a_7 = \sigma + \mu \end{aligned}$$

Therefore, to ensure the existence of a positive endemic-equilibrium, it is required that all $S^*, V^*, E^*, I^*, A^*, T^*, R^* > 0$, the endemic-equilibrium $\mathbb{E}^*$ is positive and $I^* > 0$. This is the condition for the existence and uniqueness of the state of the endemic-equilibrium for the model (2.8).

2.3.5 Basic Reproduction Number via the Next Generation Matrix

To compute the basic reproduction number, we apply the next generation matrix method.

The infected compartments are

$$x = (E, I, A)^T.$$

The disease-free equilibrium (DFE) is given by

$$\mathbb{E}^+ = (S^+, V^+, 0, 0, 0, 0, 0),$$

where

$$S^+ = \frac{\Lambda(\alpha + \mu)}{\mu(\alpha + \mu + \nu)}, \quad N^+ = \frac{\Lambda}{\mu}.$$

Hence,

$$\frac{S^+}{N^+} = \frac{\alpha + \mu}{\alpha + \mu + \nu}.$$

Step 1: New Infection Matrix F The new infection terms are

$$F_1 = \frac{S^+}{N^+}(\beta_1 I + \omega \beta_2 A), \quad F_2 = 0, \quad F_3 = 0.$$

The Jacobian matrix F evaluated at the DFE is

$$F = \begin{pmatrix} 0 & \beta_1^* & \beta_2^* \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

where

$$\beta_1^* = \frac{\beta_1(\alpha + \mu)}{\alpha + \mu + \nu}, \quad \beta_2^* = \frac{\omega \beta_2(\alpha + \mu)}{\alpha + \mu + \nu}.$$

Step 2: Transition Matrix V Let

$$k_1 = \sigma + \mu, \quad k_2 = \gamma_1 + \delta + \mu_2 + \mu, \quad k_3 = +\gamma_2 + \mu.$$

Then

$$V = \begin{pmatrix} k_1 & 0 & 0 \\ -\tau\sigma & k_2 & 0 \\ -(1-\tau)\sigma & 0 & k_3 \end{pmatrix}.$$

Step 3: Inverse of V Since V is lower triangular, its inverse is

$$V^{-1} = \begin{pmatrix} \frac{1}{k_1} & 0 & 0 \\ \frac{\tau\sigma}{k_1 k_2} & \frac{1}{k_2} & 0 \\ \frac{(1-\tau)\sigma}{k_1 k_3} & 0 & \frac{1}{k_3} \end{pmatrix}.$$

Step 4: Next Generation Matrix The next generation matrix is

$$K = FV^{-1}.$$

Multiplying gives

$$K = \begin{pmatrix} \frac{\beta_1^* \tau \sigma}{k_1 k_2} + \frac{\beta_2^* (1-\tau) \sigma}{k_1 k_3} & \frac{\beta_1^*}{k_2} & \frac{\beta_2^*}{k_3} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

Step 5: Eigenvalues of K Since K is upper triangular, its eigenvalues are the diagonal entries:

$$\lambda_1 = \frac{\beta_1^* \tau \sigma}{k_1 k_2} + \frac{\beta_2^* (1-\tau) \sigma}{k_1 k_3}, \quad \lambda_2 = 0, \quad \lambda_3 = 0.$$

Step 6: Basic Reproduction Number The basic reproduction number is defined as the spectral radius of K :

$$R_0 = \rho(K) = \max\{\lambda_1, \lambda_2, \lambda_3\}.$$

Therefore,

$$R_0 = \frac{\alpha + \mu}{\alpha + \mu + \nu} \left[\frac{\beta_1 \tau \sigma}{(\sigma + \mu)(\gamma_1 + \delta + \mu_2 + \mu)} + \frac{\omega \beta_2 (1-\tau) \sigma}{(\sigma + \mu)(\gamma_2 + \mu)} \right],$$

$$R_0 = \frac{\sigma(\alpha + \mu) [\beta_1 \tau (\gamma_2 + \mu) + \omega \beta_2 (1-\tau) (\gamma_1 + \delta + \mu_2 + \mu)]}{(\sigma + \mu)(\alpha + \mu + \nu)(\gamma_1 + \delta + \mu_2 + \mu)(+\gamma_2 + \mu)}. \quad (2.21)$$

Threshold Property The basic reproduction number R_0 determines the persistence or extinction of the disease in the population.

- If $R_0 < 1$, Disease dies out. each infectious individual produces less than one secondary infection on average. Therefore, the disease-free equilibrium $\mathbb{E}^+$ is locally asymptotically stable, and the disease dies out over time.
- If $R_0 > 1$, Disease persists. each infectious individual produces more than one secondary infection on average. Consequently, the disease-free equilibrium $\mathbb{E}^+$ is unstable, and the disease persists in the population.
- If $R_0 = 1$, Threshold case. the system is at a threshold point, and the disease dynamics depends on higher-order nonlinear terms.

Thus, the basic reproduction number R_0 serves as a threshold parameter that governs the transmission dynamics of the disease. The disease-free equilibrium $\mathbb{E}^+$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

2.3.6 Local Stability of the Deterministic of the Model

To determine the stability of the system (2.8) at the disease-free equilibrium point $\mathbb{E}^+$, the behavior of the model population near the equilibrium point is examined to determine the conditions that must be met for the disease-free equilibrium state to be stable and also for the disease to be completely eradicated from the population.

Theorem 2.1 Let

$$R_0 = \frac{\sigma(\alpha + \mu) \left[\beta_1 \tau (\gamma_2 + \mu) + \omega \beta_2 (1 - \tau) (\gamma_1 + \delta + \mu_2 + \mu) \right]}{(\sigma + \mu)(\alpha + \mu + \nu)(\gamma_1 + \delta + \mu_2 + \mu)(\gamma_2 + \mu)}$$

be the basic reproduction number of system (2.8). Then, the disease-free equilibrium point E_0 is locally asymptotically stable whenever $R_0 < 1$ and unstable whenever $R_0 > 1$.

Proof 2.1 The proof of the theorem can be established by constructing a Jacobian matrix for the model system (2.8) evaluated at the DFE point $\mathbb{E}^+$.

At DFE, $I^+ = 0$.

$$J_{\mathbb{E}^+} = \begin{pmatrix} -P_1 & \alpha & 0 & -G & -\omega H & 0 & \phi \\ \nu & -P_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -P_3 & G & \omega H & 0 & 0 \\ 0 & 0 & \tau \sigma & -P_4 & 0 & 0 & 0 \\ 0 & 0 & (1 - \tau) \sigma & 0 & -P_5 & 0 & 0 \\ 0 & 0 & 0 & \delta & 0 & -P_6 & 0 \\ 0 & 0 & 0 & \gamma_1 & \gamma_2 & \gamma_3 & -P_7 \end{pmatrix} \quad (2.22)$$

In this section, we determine the conditions under which the disease-free equilibrium (DFE) $\mathbb{E}^+$ of the system (2.8) is locally asymptotically stable. Epidemiological, local stability of the DFE implies that the disease will die out for sufficiently small initial levels of infection.

The Jacobian matrix evaluated at $\mathbb{E}^+$, denoted by $J_{\mathbb{E}^+}$ (see Eq. (2.22)), governs the local dynamics near the DFE. The Jacobian matrix evaluated at the disease-free equilibrium $\mathbb{E}^+$ has characteristic equation

$$(\lambda + P_6)(\lambda + P_7)(\lambda^2 + W_1\lambda + W_2)(\lambda^3 + W_3\lambda^2 + W_4\lambda + W_5) = 0, \quad (2.23)$$

where

$$\begin{aligned} P_1 &= \mu + \nu, & P_2 &= \alpha + \mu, \\ P_3 &= \sigma + \mu, & P_4 &= \gamma_1 + \delta + \mu_2 + \mu, \\ P_5 &= \gamma_2 + \mu, & P_6 &= \gamma_3 + \mu, \\ P_7 &= \mu + \phi, \end{aligned}$$

and

$$\begin{aligned} W_1 &= P_1 + P_2, \\ W_2 &= P_1 P_2 - \nu \alpha, \\ W_3 &= P_3 + P_4 + P_5, \\ W_4 &= P_3 P_4 + P_3 P_5 + P_4 P_5 - \sigma(\tau G + \omega H(1 - \tau)), \\ W_5 &= P_3 P_4 P_5 - \sigma(\tau G P_5 + \omega H(1 - \tau) P_4), \end{aligned}$$

with

$$G = \frac{\beta_1 S^+}{N}, \quad H = \frac{\beta_2 S^+}{N^+},$$

and

$$S^+ = \frac{\Lambda(\alpha + \mu)}{\mu(\alpha + \mu + \nu)}, \quad N^+ = \frac{\Lambda}{\mu}.$$

Applying the Routh–Hurwitz criterion to the quadratic factor

$$\lambda^2 + W_1\lambda + W_2,$$

gives the conditions

$$W_1 > 0, \quad W_2 > 0.$$

Since all model parameters are positive, it follows that

$$W_1 = P_1 + P_2 > 0.$$

Furthermore,

$$W_2 = (\mu + \nu)(\alpha + \mu) - \nu\alpha = \mu(\mu + \nu + \alpha) > 0.$$

Next, applying the Routh–Hurwitz criterion to the cubic factor

$$\lambda^3 + W_3\lambda^2 + W_4\lambda + W_5,$$

yields the conditions

$$W_3 > 0, \quad W_4 > 0, \quad W_5 > 0, \quad W_3W_4 - W_5 > 0.$$

Since

$$P_3 = \sigma + \mu > 0, \quad P_4 = \gamma_1 + \delta + \mu_2 + \mu > 0, \quad P_5 = \gamma_2 + \mu > 0,$$

we have

$$W_3 = P_3 + P_4 + P_5 > 0.$$

Moreover,

$$G = \frac{\beta_1(\alpha + \mu)}{\alpha + \mu + \nu}, \quad H = \frac{\beta_2(\alpha + \mu)}{\alpha + \mu + \nu},$$

and hence

$$W_5 = P_3P_4P_5 - \frac{\sigma(\alpha + \mu)}{\alpha + \mu + \nu} \left[\beta_1\tau P_5 + \omega\beta_2(1 - \tau)P_4 \right].$$

Using the definition of the basic reproduction number R_0 , it follows that

$$\frac{\sigma(\alpha + \mu)}{\alpha + \mu + \nu} \left[\beta_1\tau P_5 + \omega\beta_2(1 - \tau)P_4 \right] = P_3P_4P_5R_0.$$

Therefore,

$$W_5 = P_3P_4P_5(1 - R_0). \quad (2.24)$$

Since

$$P_3 > 0, \quad P_4 > 0, \quad P_5 > 0,$$

equation (2.24) implies that

$$W_5 > 0 \iff R_0 < 1.$$

Consequently, when $R_0 < 1$, all the Routh–Hurwitz conditions are satisfied and every eigenvalue of (2.23) has a negative real part. Hence, the disease-free equilibrium point E_0 is locally asymptotically stable.

On the other hand, if $R_0 > 1$, then from (2.24),

$$W_5 < 0,$$

which violates the Routh–Hurwitz criterion. Therefore, at least one eigenvalue has a positive real part, implying that the disease-free equilibrium point E_0 is unstable. Biologically, this means that the disease will die out. This completes the proof.

2.4 Dynamic analysis of the nonlinear jump diffusion typhoid-endemic model

Consider the system of equations given in (2.9), the influx of randomness driven by W_t and N_t as given in remark 6.1.1 has a direct effect on the compartments I , R , and T . Hence, the nonlinear jump-diffusion typhoid endemic model.

$$\begin{aligned} dI &= (\tau\sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I) dt + \eta_I IdW_t + J_I IdN_t, \\ dR &= (\gamma_1 I + \gamma_2 A + \gamma_3 R - (\mu R + \phi)R) dt + \eta_R RdW_t + J_R RdN_t, \\ dT &= (\delta I - (\gamma_3 + \mu)T) dt + \eta_T TdW_t + J_T TdN_t. \end{aligned} \quad (2.25)$$

Thus, case 1 of (2.25) can be written as:

$$dI = \alpha(t, I)dt + \beta(t, I)dW_t + \zeta(t, I)dN_t, \quad (2.26)$$

where, E^* is as defined in (2.20), and

$$\begin{aligned} \alpha(t, I) &= (\tau\sigma E^* - (\gamma_1 + \delta + \mu_2 + \mu)I) \\ \beta(t, I) &= \eta_I I \\ \zeta(t, I) &= J_I I \end{aligned} \quad (2.27)$$

It is clearly seen that (2.26) is a nonlinear stochastic differential equation with a jump process. The main concern in this work is to determine the conditions for reducing (2.26) to a linear form and, secondly, to determine a solution of the reduced linear jump-diffusion endemic model.

2.4.1 Conditions for the Reducible nonlinear jump-diffusion typhoid-endemic model

Next, the sufficient condition for the reducibility of the nonlinear stochastic typhoid-endemic model is established using the method in Kloeden & Platen (2010) in [48] for an SDE without jumps.

The nonlinear SDE with jumps in (2.26) can be reduced to a linear stochastic typhoid model with jumps in f_t :

$$df_t = (\alpha_1(t)f_t + \alpha_2(t))dt + (\beta_1(t)f_t + \beta_2(t))dW_t + (\zeta_1(t)f_t + \zeta_2(t))dN_t, \quad (2.28)$$

if, $f_t = g(t, I_t)$ such that $\frac{\partial g(t, I)}{\partial I} \neq 0$ and the solution of (2.26) has the form

$$I_t = V(t, f_t) \quad (2.29)$$

where, $f = g(t, V(t, f))$ and $I = g(t, V(t, I))$, given that f satisfies (2.28).

In what follows, we shall derive a sufficient condition for the reducibility of the stochastic model (2.26)

Applying Lemma 6.1.1 to $g(t, I_t)$, we get the following.

$$\begin{aligned} dg(t, I) &= \left(\frac{\partial g(t, I_{t-})}{\partial t} + \alpha \frac{\partial g(t, I_{t-})}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g(t, I_{t-})}{\partial I^2} \right) dt + \\ &\quad \beta \frac{\partial g(t, I_{t-})}{\partial I} dW_t + \left(g(I_{t-} + \gamma) - g(I_{t-}) - \gamma \frac{\partial g(t, I_{t-})}{\partial I} \right) dN_t \end{aligned} \quad (2.30)$$

where,

$g(t, I_{t-})$ is the population size just before the arrival of a jump due to infection. γ = jump size.

Now, eq.(2.28) can be linked with eq. (2.30), if

$$\frac{\partial g}{\partial t} + \alpha \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g}{\partial I^2} = \alpha_1 g(t, I_t) + \alpha_2 \quad (2.31)$$

$$\beta \frac{\partial g(t, I_t)}{\partial I} = \beta_1 g(t, I_t) + \beta_2 \quad (2.32)$$

$$g(I_{t-} + \gamma) - g(I_{t-}) - \gamma \frac{\partial g}{\partial I} = \zeta_1 g(I_{t-}) + \zeta_2 \quad (2.33)$$

Consider $\alpha_1(t) = \beta_1(t) = \zeta_1(t) = 0$, then, (2.31) - (2.33) becomes:

$$\frac{\partial g}{\partial t} + \alpha \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g}{\partial I^2} = \alpha_2(t) \quad (2.34)$$

$$\beta \frac{\partial g(t, I_t)}{\partial I} = \beta_2(t) \quad (2.35)$$

$$g(I_{t-} + \gamma) - g(I_{t-}) - c \frac{\partial g}{\partial I} = \zeta_2(t) \quad (2.36)$$

Also, for $\alpha_2(t) = \varsigma(t)$, $\beta_2(t) = \kappa(t)$ and $\zeta_2(t) = \nu(t)$, then the above (2.34) - (2.36) becomes:

$$\frac{\partial g}{\partial t} + \alpha \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g}{\partial I^2} = \varsigma(t) \quad (2.37)$$

$$\beta \frac{\partial g(t, I_t)}{\partial I} = \kappa(t) \quad (2.38)$$

$$g(I_{t-} + \gamma) - g(I_{t-}) - c \frac{\partial g}{\partial I} = \nu(t) \quad (2.39)$$

From eq. (2.37) above, we have that:

$$\frac{\partial g}{\partial t} = - \left(\alpha(t, I) \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g}{\partial I^2} \right) + \varsigma(t) \quad (2.40)$$

Differentiating eq. (2.40) wrt I , gives:

$$\frac{\partial^2 g}{\partial t \partial I} = - \frac{\partial}{\partial I} \left(\alpha(t, I) \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g}{\partial I^2} \right) \quad (2.41)$$

Similarly, from eq. (2.38) and eq. (2.39)

$$\begin{aligned} \frac{\partial}{\partial I} \left(\beta \frac{\partial g(t, I_t)}{\partial I} \right) &= 0 \\ \frac{\partial g}{\partial I} (I_{t-} + \gamma) - \frac{g(I_{t-})}{\partial I} - c \frac{\partial^2 g}{\partial I^2} &= 0 \end{aligned}$$

Again, taking the derivative of eq. (2.38) wrt. t gives:

$$\frac{\partial}{\partial t} \left(\beta \frac{\partial g(t, I_t)}{\partial I} \right) = \frac{\partial}{\partial t} \left(\kappa(t) \right) \implies \beta(t, I) \frac{\partial^2 g}{\partial t \partial I} + \frac{\partial g}{\partial I} \frac{\partial \beta(t, I)}{\partial t} = \kappa'(t) \quad (2.42)$$

Note that from eq. (2.38) and eq. (2.39), $\frac{\partial g}{\partial I} = \frac{\kappa(t)}{\beta(t, I)}$ and $\frac{\partial g}{\partial I} = \frac{1}{c} \left(g(I_{t-} + \gamma) - g(I_{t-}) - \nu(t) \right)$, respectively.

$$\implies \frac{\partial g}{\partial I} = \frac{\kappa(t)}{\beta(t, I)} = \frac{1}{c} \left(g(I_{t-} + \gamma) - g(I_{t-}) - \nu(t) \right)$$

Substituting into eq. (2.42) for $\frac{\partial g(t, I_t)}{\partial I}$, $\frac{\partial^2 g}{\partial I^2}$ and also for $\frac{\partial g(t, I_t)}{\partial I} = \frac{\kappa(t)}{\beta(t, I)}$, then, eq. (2.42) can be expressed as:

$$\begin{aligned} \kappa'(t) &= -\beta(t, I) \frac{\partial}{\partial I} \left(\alpha(t, I) \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g}{\partial I^2} \right) + \frac{\partial g}{\partial I} \frac{\kappa(t)}{\beta(t, I)} \\ \kappa'(t) &= -\beta(t, I) \frac{\partial}{\partial I} \left(\alpha(t, I) \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial}{\partial I} \left(\frac{\partial g}{\partial I} \right) \right) + \frac{\partial g}{\partial I} \frac{\kappa(t)}{\beta(t, I)} \\ \kappa'(t) &= -\beta(t, I) \frac{\partial}{\partial I} \left(\alpha(t, I) \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial}{\partial I} \left(\frac{\kappa(t)}{\beta(t, I)} \right) \right) + \frac{\partial g}{\partial I} \frac{\kappa(t)}{\beta(t, I)} \end{aligned} \quad (2.43)$$

Upon solving, we obtain:

$$\kappa'(t) = \kappa(t) \Gamma(t, I) \quad (2.44)$$

In eq. (2.44), since κ' is independent of I , then,

$$\frac{\partial \Gamma}{\partial I}(t, I) = 0$$

where,

$$\Gamma(t, I) = \frac{1}{\beta(t, I)} \frac{\partial g}{\partial t} - \beta(t, I) \frac{\partial}{\partial I} \left(\frac{\alpha(t, I)}{\beta(t, I)} - \frac{1}{2} \frac{\partial g}{\partial I}(t, I) \right) \quad (2.45)$$

Eq. (2.44) is a sufficient condition for the reducibility of the non-linear stochastic model (2.26) to the explicitly integrable model

$$df_t = \alpha(t)dt + \beta_1(t)dW_t + \zeta_1(t)dN_t, \quad (2.46)$$

2.4.2 Solution of the linear stochastic typhoid-endemic Model

The reducibility of the nonlinear stochastic model eq. (2.25) has been established in the previous subsection. In this subsection, the procedure for reducing eq. (2.25) to its linear form and finding the solution in its linear form is given explicitly.

Recall from eq. (2.25) that

$$dI = (\tau\sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I)dt + \eta_I IdW_t + J_I IdN_t, \quad (2.47)$$

Let $\varphi_t = \ln I_t$, by applying the lemma 6.1.1, to the logarithmic transformation $g(I) = \ln I_t$, we have:

$$dg(I_t) = g'(I_{t-})d(I_t) + \frac{1}{2}g''(I_{t-})\eta_t^2 I_{t-}^2 dt + \left[g(I_{t-} + \gamma) - g(I_{t-}) - \gamma g'(I_{t-}) \right] dN_t \quad (2.48)$$

Considering (2.47) via (2.47), given that the probability of exposed individuals becoming symptomatic infected tends to zero. Then, the continuous and jump parts are obtained, respectively as:

$$\left(-(\gamma_1 + \delta + \mu_2 + \mu) - \frac{1}{2}\eta_t^2 \right) dt + \eta_t dW_t \quad (2.49)$$

and

$$\ln(1 + \gamma)dN_t \quad (2.50)$$

Combining (2.49) and (2.50) gives:

$$d\varphi_t = \left(-(\gamma_1 + \delta + \mu_2 + \mu) - \frac{1}{2}\eta_t^2 \right) dt + \eta_t dW_t + \ln(1 + \gamma)dN_t \quad (2.51)$$

Hence, eq. (2.51) is said to be a linear additive SDE with jumps in φ and its solution can be obtained by direct integration from 0 to t .

Thus,

$$\varphi_t = \varphi_0 + \left(-(\gamma_1 + \delta + \mu_2 + \mu) - \frac{1}{2}\eta_t^2 \right) t + \eta_t W_t + \ln(1 + \gamma)N_t \quad (2.52)$$

For $\varphi_t = \ln I_t \implies \varphi_0 = \ln I_0$, then,

$$\ln I_t = \ln I_0 + \left(-(\gamma_1 + \delta + \mu_2 + \mu) - \frac{1}{2}\eta_t^2 \right) t + \eta_t W_t + \ln(1 + \gamma)N_t \quad (2.53)$$

Leading to the solution of the linear stochastic typhoid-endemic model:

$$I_t = I_0 \exp \left(\left(-(\gamma_1 + \delta + \mu_2 + \mu) - \frac{1}{2}\eta_t^2 \right) t + \eta_t W_t \right) (1 + \gamma)^{N_t} \quad (2.54)$$

Remark 2.4.1 The above procedures can be used to obtain the solutions of T and R in (2.25)

2.4.3 Basic reproduction number of the typhoid fever jump-diffusion model (R_{jp})

With reference to the non linear jump-diffusion typhoid fever model in (2.25), the major determinant of the degree of spread of the disease is I_t . Hence, the reproduction number of the typhoid fever jump-diffusion R_{jp} can be derived from:

$$dI = (\tau\sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I)dt + \eta_I IdW_t + J_I IdN_t, \quad (2.55)$$

At DFE, for $S = S^*, E = I = A = 0$, $\hat{\alpha} = \frac{\tau\sigma\beta S^*}{\sigma + \mu} - (\gamma_1 + \delta + \mu_2 + \mu)$, where, $\hat{\alpha}$ is the deterministic growth rate. Then, (2.55) becomes:

$$dI = \hat{\alpha} Idt + \eta_I IdW_t + J_I IdN_t. \quad (2.56)$$

Applying Itô's lemma to (2.56) gives:

$$d(\ln I) = \left(\hat{\alpha} - \frac{1}{2}\eta_I^2 \right) dt + \eta_I dW_t + \ln(1 + J_I)dN_t. \quad (2.57)$$

Now, for $\mathbb{E}(dW_t) = 0$, $\mathbb{E}(dN_t) = \lambda_j$, define the jump-diffusion growth rate threshold for typhoid fever $\hat{G}$ such that:

$$\begin{aligned} \hat{G} &= \mathbb{E} \left(d(\ln I) \right) = \mathbb{E} \left(\left(\hat{\alpha} - \frac{1}{2}\eta_I^2 \right) dt + \eta_I dW_t + \ln(1 + J_I)dN_t \right) \\ &\implies \hat{G} = \hat{\alpha} - \frac{1}{2}\eta_I^2 + \lambda_j \ln(1 + J_I). \end{aligned} \quad (2.58)$$

where, $\mathbb{E}(x)$ is the expected value of x . Also, for $\hat{\alpha} = (\gamma_1 + \delta + \mu_2 + \mu)(R_0 - 1)$ in (2.58), the jump-diffusion reproduction number (R_{jp}) for typhoid fever is obtained as:

$$R_{jp} = R_0 - \frac{\eta_I^2}{2(\gamma_1 + \delta + \mu_2 + \mu)} + \frac{\lambda_J}{(\gamma_1 + \delta + \mu_2 + \mu)} \ln(1 + J_I) \quad (2.59)$$

2.5 Threshold Dynamics of the Jump-Diffusion Typhoid Fever Model

In this section, we establish the extinction and persistence conditions for the stochastic jump-diffusion typhoid fever model. The analysis is based on the infected class, which plays a central role in disease transmission.

Recall the stochastic infected equation:

$$dI = \hat{\alpha}I dt + \eta_I I dW_t + J_I I dN_t, \quad (2.60)$$

where

$$\hat{\alpha} = \frac{\tau\sigma\beta_1 S^*}{\sigma + \mu} - (\gamma_1 + \delta + \mu_2 + \mu). \quad (2.61)$$

Furthermore,

$$\hat{\alpha} = (\gamma_1 + \delta + \mu_2 + \mu)(R_0 - 1). \quad (2.62)$$

2.5.1 Extinction of the Typhoid Fever Disease

Theorem 2.2 Suppose that

$$R_{jp} < 1, \quad (2.63)$$

where

$$R_{jp} = R_0 - \frac{\eta_I^2}{2(\gamma_1 + \delta + \mu_2 + \mu)} + \frac{\lambda_J}{(\gamma_1 + \delta + \mu_2 + \mu)} \ln(1 + J_I). \quad (2.64)$$

Then the infected population satisfies

$$\lim_{t \rightarrow \infty} I(t) = 0 \quad \text{a.s.} \quad (2.65)$$

Consequently, the disease becomes extinct almost surely.

Proof 2.2 Define the Lyapunov function

$$V(I) = \ln I. \quad (2.66)$$

Applying Itô's formula to (2.66) yields

$$d(\ln I) = \left(\hat{\alpha} - \frac{\eta_I^2}{2} \right) dt + \eta_I dW_t + \ln(1 + J_I) dN_t. \quad (2.67)$$

Integrating from 0 to t gives

$$\ln I(t) = \ln I(0) + \left(\hat{\alpha} - \frac{\eta_I^2}{2} \right) t + \eta_I W_t + \ln(1 + J_I) N_t. \quad (2.68)$$

Dividing by t gives

$$\frac{\ln I(t)}{t} = \frac{\ln I(0)}{t} + \left(\hat{\alpha} - \frac{\eta_I^2}{2} \right) + \eta_I \frac{W_t}{t} + \ln(1 + J_I) \frac{N_t}{t}. \quad (2.69)$$

Using the strong law of large numbers for Brownian motion and Poisson processes,

$$\lim_{t \rightarrow \infty} \frac{W_t}{t} = 0 \quad \text{a.s.}, \quad (2.70)$$

and

$$\lim_{t \rightarrow \infty} \frac{N_t}{t} = \lambda_J \quad \text{a.s.} \quad (2.71)$$

Therefore,

$$\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} = \hat{\alpha} - \frac{\eta_I^2}{2} + \lambda_J \ln(1 + J_I). \quad (2.72)$$

Substituting

$$\hat{\alpha} = (\gamma_1 + \delta + \mu_2 + \mu)(R_0 - 1), \quad (2.73)$$

gives

$$\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} = (\gamma_1 + \delta + \mu_2 + \mu)(R_{jp} - 1). \quad (2.74)$$

Since $R_{jp} < 1$, it follows that

$$\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} < 0. \quad (2.75)$$

Hence,

$$\lim_{t \rightarrow \infty} I(t) = 0 \quad \text{a.s.} \quad (2.76)$$

Therefore, the typhoid disease becomes extinct almost surely.

2.5.2 Persistence of the Typhoid Fever Disease

Theorem 2.3 Assume that

$$R_{jp} > 1. \quad (2.77)$$

Then the infected population persists in the mean, that is,

$$\liminf_{t \rightarrow \infty} \frac{1}{t} \int_0^t I(s) ds > 0 \quad \text{a.s.} \quad (2.78)$$

Consequently, the typhoid fever disease persists in the population.

Proof 2.3 If $R_{jp} > 1$, then

$$\hat{\alpha} - \frac{\eta_I^2}{2} + \lambda_J \ln(1 + J_I) > 0. \quad (2.79)$$

Hence,

$$\liminf_{t \rightarrow \infty} \frac{\ln I(t)}{t} > 0. \quad (2.80)$$

Therefore, there exists a positive constant $c > 0$ such that

$$I(t) \geq c \quad (2.81)$$

for sufficiently large t with probability one.

Consequently,

$$\liminf_{t \rightarrow \infty} \frac{1}{t} \int_0^t I(s) ds \geq c > 0. \quad (2.82)$$

Thus, the infected population remains bounded away from zero on average and the typhoid fever disease persists in the population.

Corollary 2.1 The jump-diffusion reproduction number

$$R_{jp} = R_0 - \frac{\eta_I^2}{2(\gamma_1 + \delta + \mu_2 + \mu)} + \frac{\lambda_J}{(\gamma_1 + \delta + \mu_2 + \mu)} \ln(1 + J_I) \quad (2.83)$$

serves as the threshold parameter governing the long-term dynamics of typhoid fever. Specifically,

$$R_{jp} < 1 \implies \text{Typhoid Disease Extinction}, \quad (2.84)$$

while

$$R_{jp} > 1 \implies \text{Typhoid Disease Persistence}. \quad (2.85)$$

3 Sensitivity Analysis and Numerical Simulation of the Model

Sensitivity analysis is used to check the effect of each parameter in order to reduce the transmission of typhoid fever from humans to humans. The comparative contributions of each parameter applied in the model are responsible for the transmission and prevalence of typhoid fever which is being discussed here. The Table 4 displayed each parameter used in this section chosen from the realistic ranges for illustrative purposes.

Table 4: Parameter values of Typhoid Fever Model

Symbol	Value	Reference
Λ	100	Assumed
β_1	0.6 [0 - 1]	[28, 33, 62]
β_2	0.4 [0 - 1]	Assumed
τ	0.85	[14]
ω	0.85	Assumed
σ	0.4	[28, 33, 36]
γ_1	0.08	Assumed
γ_2	0.1	Assumed
γ_3	0.12	Assumed
δ	0.01 [0 - 1]	Assumed
ϕ	0.02	[1, 2, 14, 24]
μ	0.01	[1, 33, 36, 39, 43]
μ_2	0.02	[2, 24]
α	0.04	Assumed
ν	0.1 [0 - 1]	[1, 43, 62]
η_I	0.2	Assumed
η_R	0.2	Assumed
η_T	0.2	Assumed
J_I	0.3	Assumed
J_R	0.3	Assumed
J_T	0.3	Assumed

3.1 Sensitivity Indices of R_0

Definition 3.1 Normalized forward sensitivity index of a variable W that differentially depends on a parameter m , is defined as: $Z_m^W = \frac{\partial W}{\partial m} \times \frac{m}{W}$ where, $Z_m^{R_0}$ is the sensitivity index of R_0 with respect to parameter, m .

Given the explicit form of $R_0^{b,h}$; analytical expression for its sensitivity to each parameter is obtained by applying the normalized forward sensitivity index in [9], [10], [13], [65] given as:

$$Z_m^{R_0} = \frac{\partial R_0}{\partial m} \times \frac{m}{R_0} \quad (3.1)$$

Sensitivity indices of R_0 corresponding to these parameters: $\beta_1, \beta_2, \tau, \omega, \sigma, \gamma_1, \gamma_2, \delta, \mu, \mu_2$, and α were derived and computed as follows.

$$\left. \begin{aligned} Z_{\beta_1}^{R_0} &= \frac{\partial R_0}{\partial \beta_1} \times \frac{\beta_1}{R_0} = +0.90160 \\ Z_{\beta_2}^{R_0} &= \frac{\partial R_0}{\partial \beta_2} \times \frac{\beta_2}{R_0} = +0.09836 \\ Z_{\nu}^{R_0} &= \frac{\partial R_0}{\partial \nu} \times \frac{\nu}{R_0} = -0.6666667 \end{aligned} \right\} \quad (3.2)$$

The remaining indices are generated following the same method and presented in Table 5.

Table 5: Sensitivity indices of R_0

Parameter	Baseline	Sensitivity index
β_1	0.6	0.9016393438
β_2	0.4	0.09836065569
τ	0.85	0.3442622950
ω	0.85	0.09836065569
σ	0.4	0.02439024369
γ_1	0.08	-0.6010928956
γ_2	0.1	-0.08941877837
δ	0.01	-0.07513661195
μ	0.01	0.02486459955
μ_2	0.02	-0.1502732239
α	0.04	0.5333333329
ν	0.1	-0.6666666664

After differentiating R_0 partially with respect to each of the parameter involved, the sensitivity indices are obtained and presented in the table above. The most sensitive parameters are: $\nu, \gamma_1, \mu_2, \gamma_2$ and δ , while the least is the most positive value. Using the above parameter values in Table 4, computed on maple to obtained $R_0 = 1.532889875$, when vaccination rate ($\nu = 0.0$) and reduced drastically when ($\nu = 1.0$) to obtained $R_0 = 0.2189842677$ consequently, when the Contact rate is reduced from 0.6 to 0.05, then R_0 changes from 1.532889875 to 0.3811283567. This shows that the Typhoid fever is controllable.

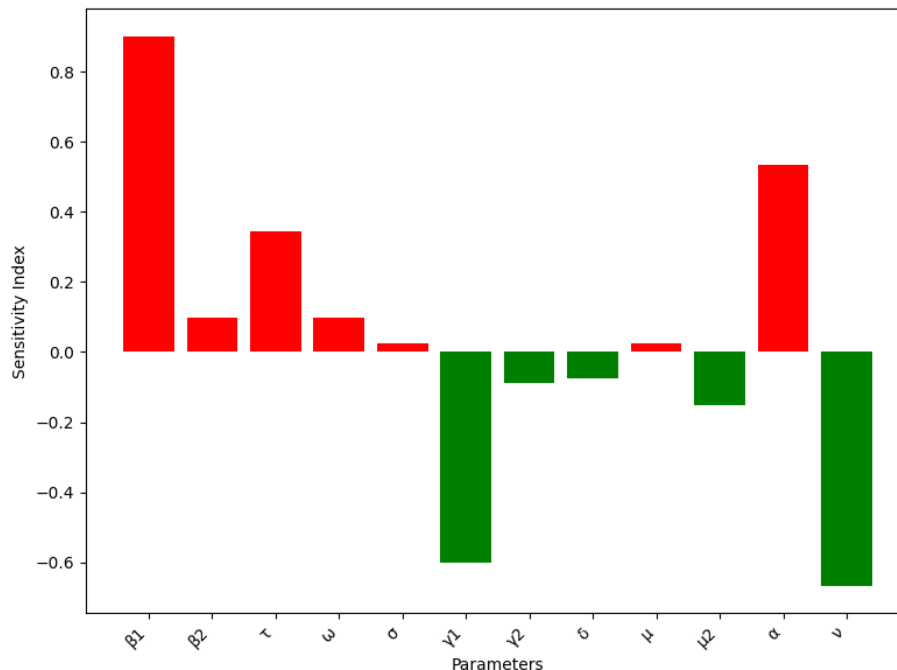


Figure 2: Sensitivity Analysis of Typhoid Fever

From Table 5, the indices with positive signs show that the value of R_0 increases, when the corresponding parameters increase and indices with negative signs show that the value of R_0 reduces with increase in the corresponding parameters. This analysis is obtained to establish which parameters govern the results of our analysis. Hence, it is clear from Table 5 that R_0 will be minimised with increases in the values of the relevant parameters, since the sensitivity indices of these parameters are negative.

3.2 PRCC Sensitivity Analysis

In the global sensitivity analysis, two different methods can be carry out. These methods are PRCC (Partial Rank Correlation Coefficient) and Sobol method. In this work, paper, the PRCC (Partial Rank Correlation Coefficient) method is utilized. The PRCC graph illustrates the strength and direction of the relationship between each model parameter and the basic reproduction number (R_0), while controlling for the effects of all other parameters.

General Interpretation Rules

- PRCC (> 0): Increasing the parameter increases (R_0) and enhances Typhoid transmission.
- PRCC (< 0): Increasing the parameter reduces (R_0) and suppresses Typhoid transmission.
- Large $|PRCC|$ values: The parameter has a strong influence on (R_0).
- PRCC close to 0: The parameter has a weak or negligible effect.

Table 6: PRCC Sensitivity indices of R_0

Parameter	PRCC Sensitivity index
β_1	0.92676536
β_2	0.41036103
τ	0.70018609
ω	0.41545726
σ	0.25123389
γ_1	-0.7832032
γ_2	-0.03779341
δ	-0.03779341
μ	0.83339351
μ_2	-0.82330497
α	0.26235564
ν	-0.15144619

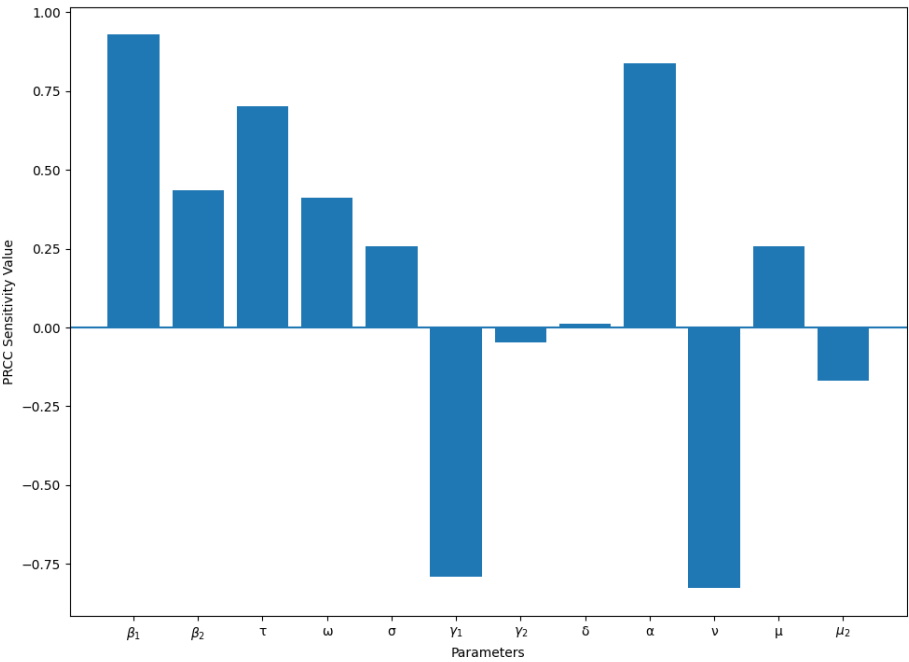


Figure 3: PRCC Sensitivity Analysis

From the Table 6 and diagram above in Figure (3), the parameter interpretation of the graph is as follows:

- β_1 and α exhibits the largest positive PRCC values, indicating that they are the most influential parameters promoting Typhoid transmission. Small increases in β_1 and α lead to substantial increases in (R_0).
- β_2 and ω : These parameters show moderate positive PRCC values, implying that they contribute positively to typhoid transmission, but less strongly than β_1 and α . Reducing these parameters would moderately lower (R_0).
- τ exhibits large positive PRCC value, indicating that it is one of the most influential parameter promoting Typhoid transmission. Small increases in τ lead to substantial increases in (R_0).
- σ exhibits the largest positive PRCC value, indicating that it is the most influential parameter promoting TB transmission. Small increases in δ lead to substantial increases in (R_0).

- v. γ_1 and ν : has a strong negative PRCC, showing that they plays a key role in suppressing transmission. They represents an effective control parameter.
- vi. γ_2 : The PRCC for γ_2 is negative but relatively small, suggesting a weak protective effect on Typhoid transmission.
- vii. δ : The PRCC for δ is positive but relatively small, suggesting a weak protective effect on TB transmission.
- viii. μ_2 : The PRCC value of Ψ is negative, indicating an inverse relationship with (R_0) . Increasing (μ_2) reduces Typhoid spread.

3.3 Effect of Transmission Rate and Control Parameter on R_0

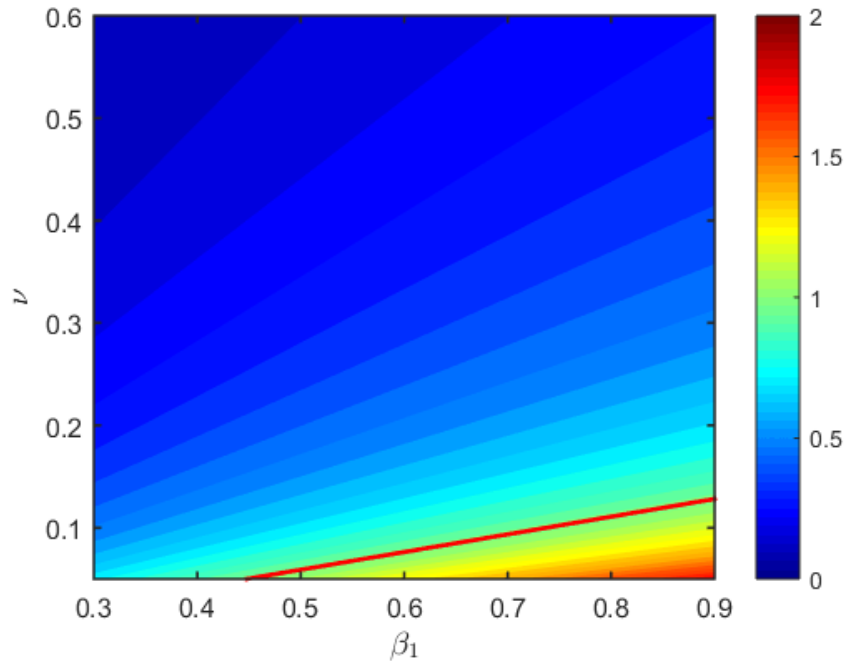


Figure 4: Heatmap on R_0 vs ν and β_1

In Figure 4 the heatmap illustrates the variation of the basic reproduction number R_0 with respect to the transmission rate β_1 and the vaccination control rate ν . It is observed that R_0 increases steadily as β_1 increases, indicating that higher transmission intensity leads to greater disease spread. In contrast, R_0 decreases with increasing ν , reflecting the mitigating effect of this parameter on transmission. The contour patterns further show that at low values of ν , R_0 remains relatively high across the range of β_1 , whereas higher values of ν significantly suppress R_0 . This demonstrates the effectiveness of the vaccination control rate mechanism associated with ν in reducing the reproduction number.

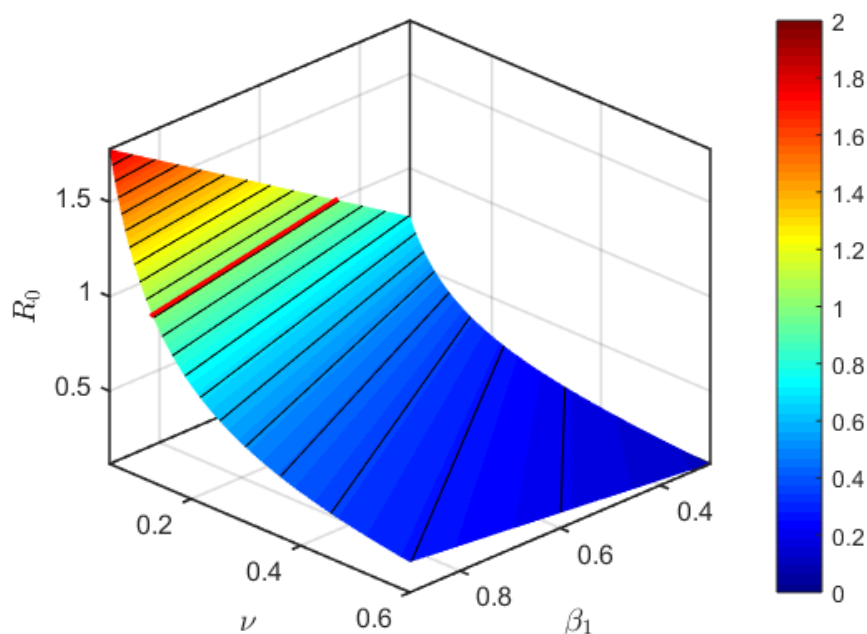


Figure 5: Surface Plot R_0 vs ν and β_1

In Figure 5 the surface plot provides a three-dimensional representation of the relationship between R_0 , β_1 , and ν . The surface rises along the β_1 -axis, confirming that R_0 is positively influenced by the transmission rate. Conversely, the surface declines sharply as ν increases, indicating a strong negative relationship between ν and R_0 . The curvature of the surface suggests a nonlinear interaction, where the impact of increasing ν becomes more pronounced at higher values of β_1 . This reinforces the role of ν as an effective vaccination control rate parameter capable of substantially reducing disease transmission even in high-risk scenarios.

3.4 Effect of the Brownian volatility (η_I) and the jump-diffusion intensity parameters (λ_J) on the stochastic reproduction ratio (R_{jp})

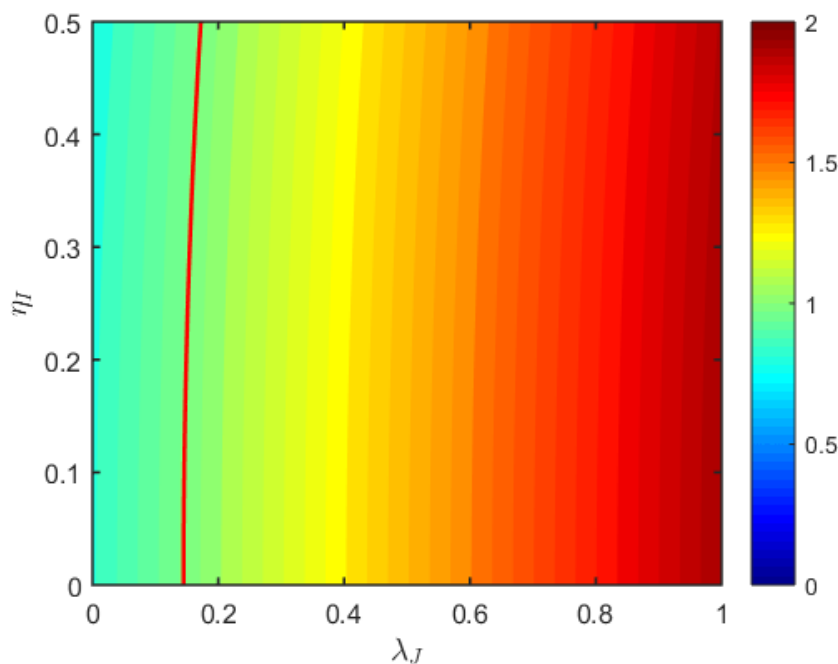


Figure 6: Heatmap on R_{jp} vs η_I and λ_J

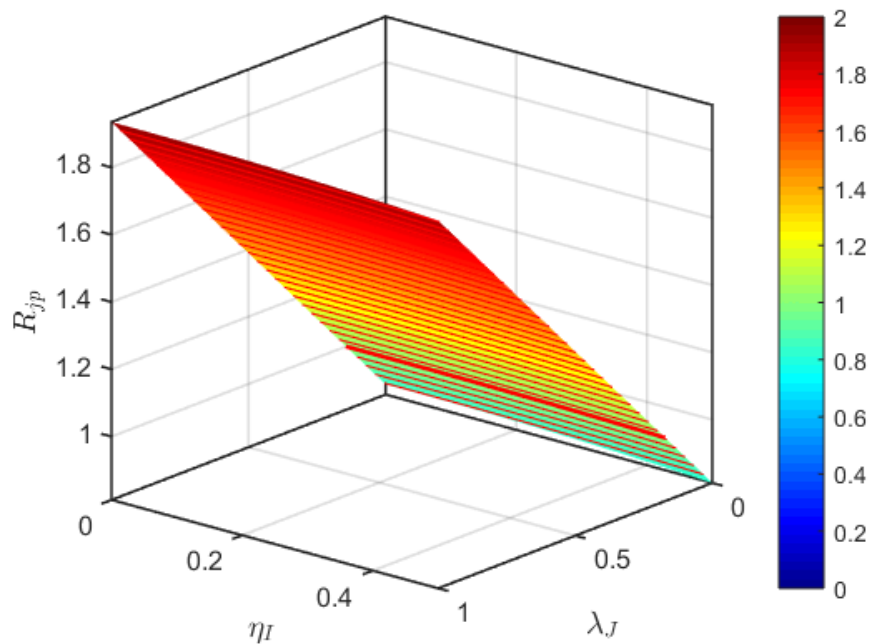


Figure 7: Surface Plot R_{jp} vs η_I and λ_J

Figures 6 and 7 depict the combined effects of the Brownian volatility parameter (η_I) and the jump-diffusion intensity parameter (λ_J) on the stochastic reproduction ratio (R_{jp}). The results indicate that R_{jp} decreases as η_I increases, demonstrating that continuous random fluctuations in the infected population tend to suppress disease transmission. In contrast, R_{jp} increases with increasing λ_J , suggesting that jump-induced transmission shocks enhance disease persistence and outbreak potential. Furthermore, the surface plot reveals that R_{jp} is more sensitive to variations in λ_J than to changes in η_I , highlighting the dominant influence of jump-diffusion effects on the stochastic epidemic dynamics.

At the parameter values $\lambda_J = 0.1$ and $\eta_I = 0.4$, the stochastic reproduction ratio is found to be $R_{jp} = 1.08$. Since $R_{jp} > 1$, the disease-free equilibrium is not stochastically stable, implying that the disease can persist within the population. This result suggests that the positive contribution of jump-diffusion effects outweighs the mitigating influence of Brownian fluctuations, thereby sustaining disease transmission despite the presence of environmental noise.

Table 7: Case 1: Varying η_I against J_I and λ_J on R_0 and R_{jp}

η_I	J_I	λ_J	R_0	R_{jp}
0.0	0.0	0.0	1.532889875	1.532889875
0.2	0.3	0.1	1.532889875	1.584860095
0.4	0.3	0.1	1.532889875	1.084860095
0.6	0.3	0.1	1.532889875	0.2515267621
0.8	0.3	0.1	1.532889875	-0.9151399049
1.0	0.3	0.1	1.532889875	-2.415139905

Table 7 illustrates the effects of varying the diffusion intensity parameter (η_I) in the infected compartment on the deterministic reproduction number (R_0) and the stochastic reproduction number (R_{jp}), while keeping the jump magnitude ($J_I = 0.3$) and jump intensity ($\lambda_J = 0.1$) fixed. As expected, the deterministic reproduction number remains constant at ($R_0 = 1.5329$), indicating that stochastic perturbations do not affect the threshold dynamics of the deterministic model.

In contrast, the stochastic reproduction number exhibits substantial sensitivity to changes in (η_I). When ($\eta_I = 0$), the absence of stochastic fluctuations yields ($R_{jp} = R_0$), confirming consistency between the deterministic and stochastic formulations. A small increase in diffusion intensity ($\eta_I = 0.2$) causes (R_{jp}) to increase slightly above (R_0), suggesting that weak stochastic fluctuations may temporarily enhance transmission. However, as (η_I) increases further, (R_{jp}) decreases monotonically, falling below the epidemic threshold ($R_{jp} < 1$) at ($\eta_I = 0.6$). This indicates that sufficiently strong stochastic fluctuations suppress disease transmission and promote disease extinction, even though the deterministic model predicts persistence ($R_0 > 1$).

For larger values of (η_I) ($0.8 \leq \eta_I \leq 1.0$), (R_{jp}) becomes negative, reflecting a dominant stochastic stabilisation effect. Epidemiologically, this suggests that strong environmental variability, behavioural heterogeneity, or intervention-induced fluctuations can significantly reduce the effective transmission potential of typhoid fever. These

findings highlight the importance of incorporating diffusion effects into epidemic models, as deterministic analyses alone may overestimate disease persistence under highly variable transmission environments.

Table 8: Case 2: Varying J_I against η_I and λ_J on R_0 and R_{jp}

J_I	η_I	λ_J	R_0	R_{jp}
-0.5	0.2	0.1	1.532889875	0.7886005575
-0.3	0.2	0.1	1.532889875	1.068994088
-0.1	0.2	0.1	1.532889875	1.278422778
0.1	0.2	0.1	1.532889875	1.445648358
0.3	0.2	0.1	1.532889875	1.584860095
0.5	0.2	0.1	1.532889875	1.704110798

Table 8 presents the effects of varying the jump magnitude parameter (J_I) in the infected compartment on the deterministic reproduction number (R_0) and the stochastic reproduction number (R_{jp}), while keeping the diffusion intensity ($\eta_I = 0.2$) and jump frequency ($\lambda_J = 0.1$) fixed. The deterministic reproduction number remains unchanged at ($R_0 = 1.5329$) for all values of (J_I), indicating that the deterministic transmission dynamics are independent of jump perturbations.

In contrast, the stochastic reproduction number (R_{jp}) exhibits a strong dependence on the magnitude and direction of jump events. For negative values of (J_I), (R_{jp}) decreases substantially, with ($R_{jp} = 0.7886$) when ($J_I = -0.5$). Since ($R_{jp} < 1$), the disease is expected to decline and eventually become extinct under sufficiently large negative jumps. Epidemiologically, negative jump amplitudes may represent sudden reductions in transmission arising from effective intervention campaigns, rapid treatment uptake, improved sanitation, or successful outbreak containment measures.

As (J_I) increases from negative to positive values, (R_{jp}) increases monotonically. The epidemic threshold is crossed between ($J_I = -0.3$) and ($J_I = -0.1$), beyond which ($R_{jp} > 1$), indicating conditions favourable for disease persistence. For positive jump amplitudes, (R_{jp}) exceeds the deterministic threshold and continues to increase, reaching ($R_{jp} = 1.7041$) when ($J_I = 0.5$). This behaviour suggests that positive jumps amplify disease transmission by generating abrupt increases in the number of infectious individuals. Such events may arise from sudden contamination of water sources, population displacement, breakdowns in sanitation infrastructure, or delays in healthcare delivery.

Overall, the results demonstrate that the jump magnitude is a critical determinant of the stochastic epidemic threshold. While negative jumps promote disease elimination, positive jumps enhance transmission and increase the likelihood of endemic persistence. These findings highlight the importance of accounting for rare but significant outbreak events when assessing typhoid fever dynamics and designing effective disease-control strategies. Table 9

Table 9: Case 3: Varying λ_J against η_I and J_I on R_0 and R_{jp}

λ_J	η_I	J_I	R_0	R_{jp}
0.0	0.0	0.0	1.532889875	1.532889875
0.2	0.2	0.3	1.532889875	1.803496982
0.4	0.2	0.3	1.532889875	2.240770756
0.6	0.2	0.3	1.532889875	2.678044530
0.8	0.2	0.3	1.532889875	3.115318305
1.0	0.2	0.3	1.532889875	3.552592079

examines the effect of varying the jump intensity parameter (λ_J) on the deterministic reproduction number (R_0) and the stochastic reproduction number (R_{jp}), while keeping the diffusion intensity and jump magnitude fixed at ($\eta_I = 0.2$) and ($J_I = 0.3$), respectively. The deterministic reproduction number remains constant at ($R_0 = 1.5329$) for all values of (λ_J), confirming that (R_0) is independent of stochastic jump effects and reflects only the deterministic transmission structure of the model.

By contrast, (R_{jp}) increases monotonically as (λ_J) increases. In the absence of stochastic perturbations ($\lambda_J = 0, \eta_I = 0, J_I = 0$), the stochastic and deterministic thresholds coincide, with ($R_{jp} = R_0 = 1.5329$). When (λ_J) is increased from (0.2) to (1.0), (R_{jp}) rises from (1.8035) to (3.5526), indicating that more frequent positive jump events substantially increase the effective transmission potential of the disease. Since ($R_{jp} \geq 1$) for all positive values of (λ_J) considered, the stochastic model predicts persistence of typhoid fever under recurrent positive outbreak shocks.

Epidemiologically, this result implies that frequent jump events in the infected compartment, such as sudden water contamination, sanitation breakdown, population displacement, delayed diagnosis, or interruption of treatment services, can amplify disease transmission even when the deterministic reproduction number remains unchanged. The increasing values of (R_{jp}) show that the frequency of outbreak shocks is a critical driver of stochastic disease persistence. Therefore, reducing the occurrence of such jump events through improved sanitation, rapid outbreak detection, stable healthcare delivery, and timely treatment interventions is essential for lowering the stochastic epidemic threshold and controlling typhoid fever transmission.

4 Numerical Simulation of the Model

To numerically simulate the stochastic typhoid fever model, a time discretisation approach based on the Euler–Maruyama scheme combined with a Poisson jump process was employed. Let $(t_n = n\Delta t)$, where (Δt) denotes the simulation time step.

The Brownian increment is approximated by

$$\Delta W_n = W(t_{n+1}) - W(t_n) \approx \sqrt{\Delta t} Z_n,$$

where, $(Z_n \sim N(0, 1))$ is a standard normal random variable. In the context of the typhoid fever model, (ΔW_n) represents continuous random fluctuations in disease transmission and progression arising from environmental variability, behavioural heterogeneity, seasonal effects, reporting errors, and uncertainties in healthcare delivery. These perturbations are assumed to occur continuously over time and affect the infected, treated, and recovered populations through the diffusion coefficients (η_I) , (η_T) , and (η_R) , respectively.

The jump increment is given by

$$\Delta N_n = N(t_{n+1}) - N(t_n),$$

where (ΔN_n) follows a Poisson distribution with parameter $(\lambda_J \Delta t)$. Here, (λ_J) denotes the average frequency of jump events per unit time. Epidemiologically, (ΔN_n) captures sudden and discrete changes in disease dynamics that cannot be represented by continuous fluctuations alone. Such events may include contamination of water supplies, breakdowns in sanitation infrastructure, population displacement, interruptions in treatment programmes, healthcare system shocks, or localised outbreak clusters. The jump amplitudes (J_I) , (J_T) , and (J_R) quantify the magnitude of these abrupt changes in the infected, treated, and recovered compartments, respectively.

Consequently, the stochastic terms $(\eta_k X_k \Delta W_n)$ model continuous environmental and demographic variability, whereas the jump terms $(J_k X_k \Delta N_n)$ account for rare but potentially significant outbreak events. The combined jump–diffusion framework therefore enables the model to capture both gradual stochastic variability and abrupt epidemiological shocks that characterise real-world typhoid fever transmission dynamics.

The force of infection is given by

$$\lambda = \frac{\beta_1 I + \omega \beta_2 A}{N}, \quad (4.1)$$

where

$$N = S + V + E + I + A + T + R$$

represents the total human population.

Applying the Euler–Maruyama discretization to system (2.9) yields

$$S_{n+1} = S_n + [\Lambda - (\lambda_n + \mu + \nu)S_n + \alpha V_n + \phi R_n] \Delta t, \quad (4.2)$$

$$V_{n+1} = V_n + [\nu S_n - (\alpha + \mu)V_n] \Delta t, \quad (4.3)$$

$$E_{n+1} = E_n + [\lambda_n S_n - (\sigma + \mu)E_n] \Delta t, \quad (4.4)$$

$$I_{n+1} = I_n + [\tau \sigma E_n - (\gamma_1 + \delta + \mu_2 + \mu)I_n] \Delta t + \eta_I I_n \Delta W_n + J_I I_n \Delta N_n, \quad (4.5)$$

$$A_{n+1} = A_n + [(1 - \tau)\sigma E_n - (\gamma_2 + \mu)A_n] \Delta t, \quad (4.6)$$

$$T_{n+1} = T_n + [\delta I_n - (\gamma_3 + \mu)T_n] \Delta t + \eta_T T_n \Delta W_n + J_T T_n \Delta N_n, \quad (4.7)$$

$$R_{n+1} = R_n + [\gamma_1 I_n + \gamma_2 A_n + \gamma_3 T_n - (\mu + \phi)R_n] \Delta t + \eta_R R_n \Delta W_n + J_R R_n \Delta N_n. \quad (4.8)$$

The above discretized system was computationally implemented to generate stochastic trajectories of the model variables over time. The simulations illustrate the influence of stochastic perturbations and jump processes on the transmission dynamics of typhoid fever.

All numerical simulations were performed using Python and the resulting trajectories were plotted to examine the temporal evolution of the epidemiological compartments.

To explore the dynamical behavior of the typhoid fever model, we conducted numerical simulations of both the deterministic and stochastic systems. This allowed us to compare the impact of random fluctuations and jumps on disease transmission with the baseline deterministic dynamics.

4.1 Deterministic Simulation

The deterministic model corresponds to the system of ordinary differential equations (ODEs) described in Eq. (2.9) without stochastic terms (i.e., setting $\eta_i = J_i = 0$ for all compartments). The deterministic system was solved numerically using the `odeint` function from `SciPy`, which employs the LSODA algorithm. LSODA automatically switches between non-stiff (Adams) and stiff (Backward Differentiation Formula, BDF) methods, providing high accuracy and stability for multi-compartment epidemic systems.

Let $y(t) = [S(t), V(t), E(t), I(t), A(t), T(t), R(t)]^\top$ denote the state vector. Then, the deterministic solution is obtained by solving:

$$\frac{dy}{dt} = f(y, t),$$

with initial conditions corresponding to the population distribution at $t = 0$. The solver produces continuous trajectories for all compartments over the simulation time horizon.

4.2 Stochastic Simulation

The stochastic system incorporates environmental and demographic noise through Brownian motion and non-Gaussian jumps. The stochastic differential equations (SDEs) for the model include terms of the form:

$$\eta_i X_i dW_t + J_i X_i dN_t,$$

where dW_t represents standard Brownian increments and dN_t is a Poisson jump process with intensity λ_J .

Due to the presence of stochastic terms, we implemented the Euler-Maruyama method, which is the standard numerical scheme for SDEs. The system is discretized as:

$$X_{n+1} = X_n + f(X_n)\Delta t + \eta_i X_n \Delta W_n + J_i X_n \Delta N_n,$$

where $\Delta W_n \sim \mathcal{N}(0, \Delta t)$ and $\Delta N_n \sim \text{Poisson}(\lambda_J \Delta t)$.

This approach is widely used in stochastic epidemic modeling because it efficiently handles both continuous fluctuations and discrete jump events while maintaining accuracy over large simulation horizons.

4.3 Simulation Strategy

The deterministic and stochastic simulations were performed over the same time horizon with identical initial conditions and parameter values. For the stochastic system, multiple realizations were generated to illustrate the range of possible trajectories and quantify variability due to randomness. Comparisons between deterministic and stochastic trajectories provide insight into the effects of noise on typhoid transmission dynamics and the robustness of control strategies.

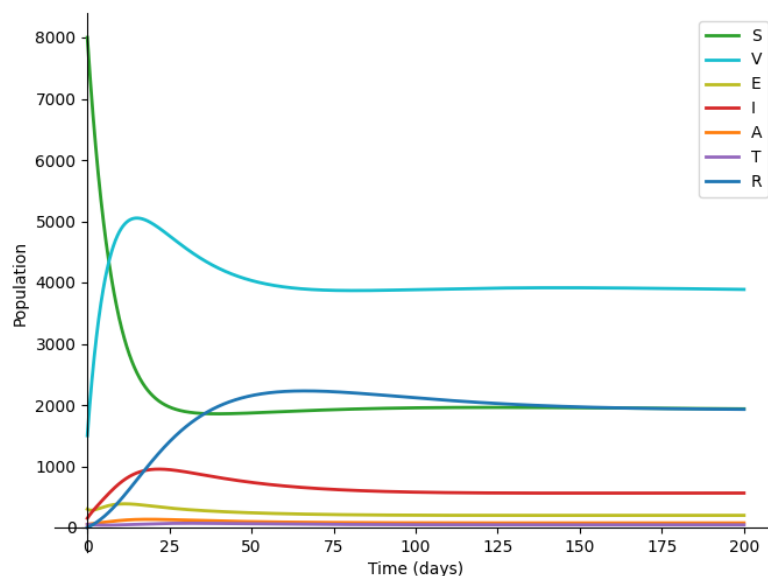


Figure a: Deterministic Case

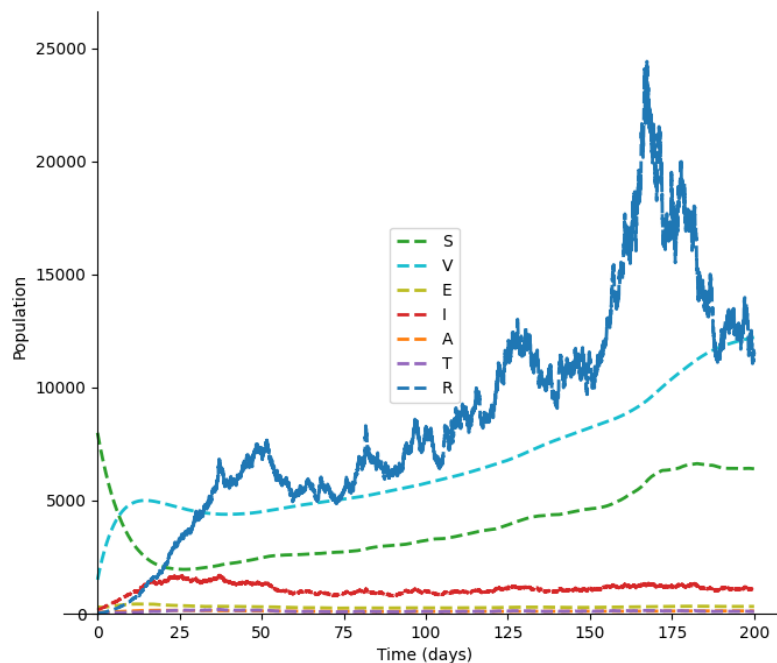


Figure b: Stochastic Case

Figure 8: Graph of deterministic and stochastic Typhoid models, respectively

Figure 8 presents the time evolution of the model compartments for both the deterministic and stochastic formulations. The deterministic simulation 8a shows smooth trajectories converging to an endemic equilibrium, where susceptible individuals decline while vaccinated and recovered populations increase over time. The infected population exhibits a single peak before stabilizing. In contrast, the stochastic simulation 8b displays irregular fluctuations and multiple peaks, reflecting random variations in disease transmission and highlighting the impact of environmental and demographic noise on the system dynamics.

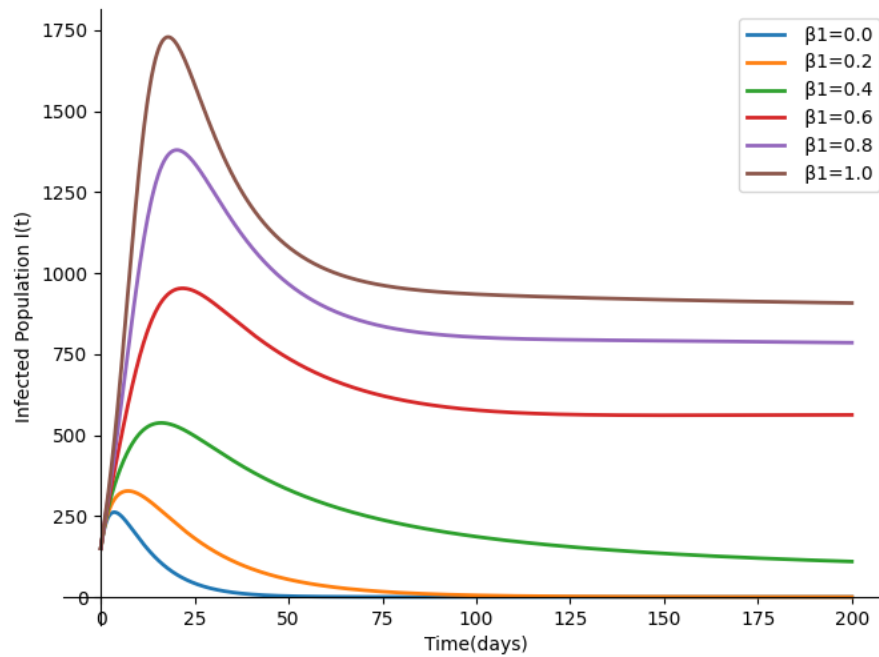


Figure a: Deterministic Case

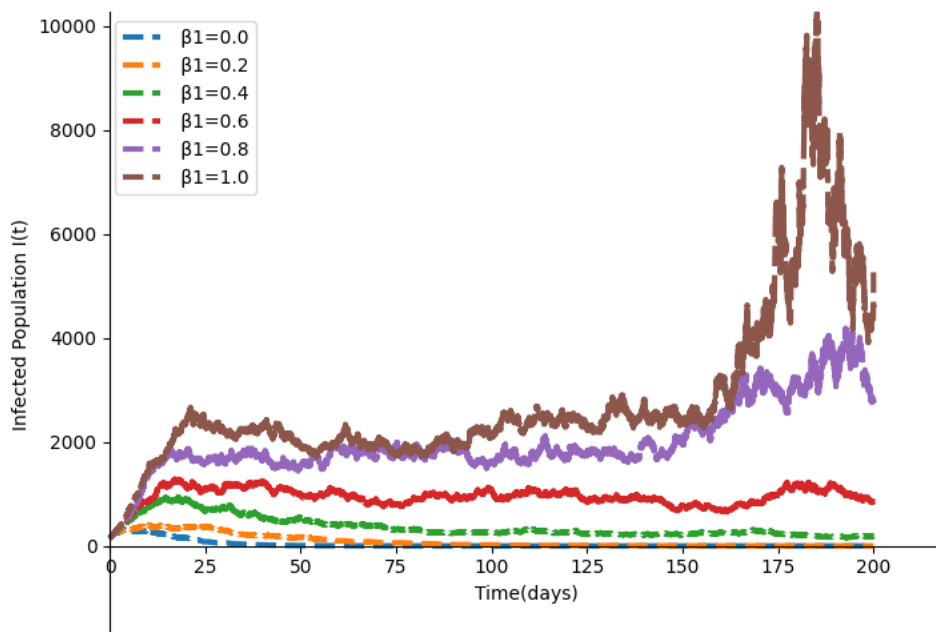


Figure b: Stochastic Case

Figure 9: Effects of symptomatic contact rate on infected population $I(t)$

Figure 9 presents the effect of the contact rate β_1 on the infected population. The deterministic simulation in Figure 9a shows that increasing the contact rate β_1 significantly increases the maximum and steady-state level of the infected population, indicating faster disease transmission and greater endemic burden. In contrast, the stochastic simulation in Figure 9b exhibits pronounced fluctuations and multiple peaks, with higher values of β_1 leading to increased variability and more severe outbreak patterns. This highlights the critical role of contact rate in driving infection dynamics under both deterministic and stochastic conditions.

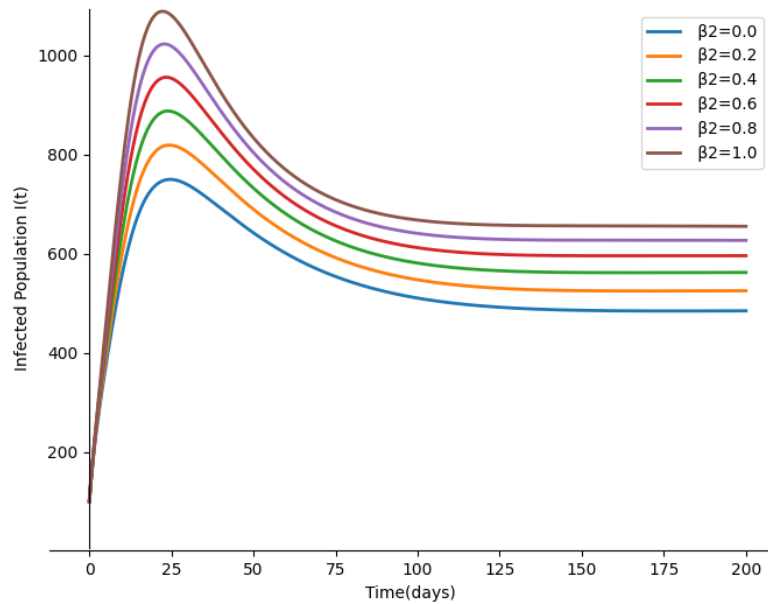


Figure a: Deterministic Case

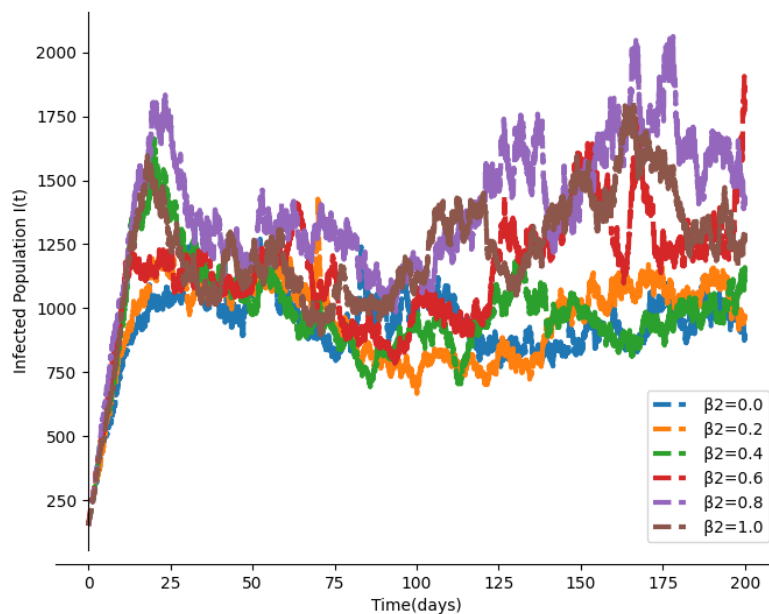


Figure b: Stochastic Case

Figure 10: Effects of asymptomatic contact rate on infected population $I(t)$

Both graphs illustrate the variation of the infected population $I(t)$ over time for different values of the asymptomatic contact rate β_2 . As β_2 increases, the maximum number of infections increases significantly, while the timing of the peak remains relatively unchanged. After peak, infection levels decline but stabilize at higher values for larger β_2 , indicating that an increase in asymptomatic contact rate intensifies the outbreak and maintains higher infection levels in the population.

Stochastic simulation showing the effect of the asymptomatic contact rate (β_2) on the infected population $I(t)$. As β_2 increases, the level of infection increases and exhibits stronger fluctuations with multiple peaks. The irregular patterns reflect random environmental effects and highlight the significant role of asymptomatic individuals in sustaining disease transmission.

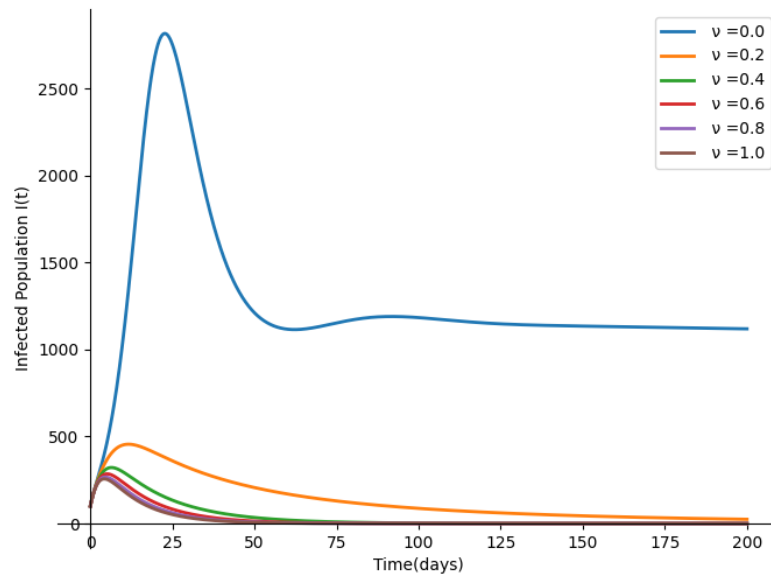


Figure a: Deterministic Case

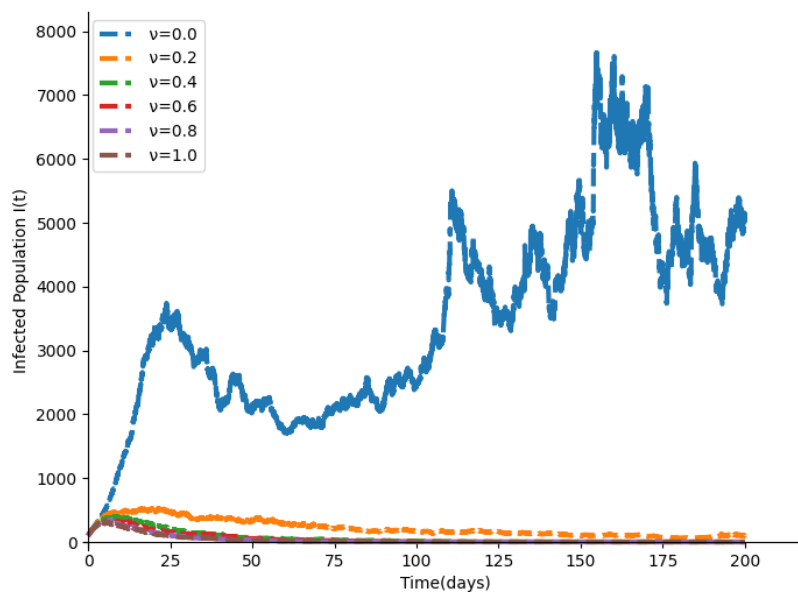


Figure b: Stochastic Case

Figure 11: Effects of vaccination rate on infected population $I(t)$

From Fig. 11a, which represents the deterministic case, it is observed that as the vaccination rate increases, the number of infectious individuals decreases significantly. Higher vaccination rates reduce the pool of susceptible individuals, thus limiting disease transmission. As a result, both the peak and the long-term level of infection are substantially reduced, indicating that vaccination is an effective control strategy to mitigate the spread of typhoid fever.

In contrast, Fig. 11b illustrates the stochastic case, where the trajectories exhibit noticeable fluctuations due to random environmental and demographic effects. Despite these irregular variations, the overall trend remains consistent with the deterministic results. Specifically, increasing the vaccination rate leads to a reduction in the magnitude of the infected population. However, the presence of stochastic perturbations introduces variability, resulting in multiple peaks and oscillatory behavior in the infection curves.

Furthermore, at lower vaccination rates, the infected population remains high and exhibits stronger fluctuations, suggesting a higher risk of recurrent outbreaks. As the vaccination rate increases, both the intensity and variability

of the infection decrease, demonstrating that vaccination not only reduces the average number of infected individuals but also stabilizes the system against random disturbances.

Overall, the results highlight the critical role of vaccination in controlling typhoid fever dynamics. The comparison between deterministic and stochastic simulations further emphasizes that while deterministic models provide a baseline understanding of disease control, stochastic models offer a more realistic representation by capturing the effects of randomness and uncertainty in transmission dynamics.

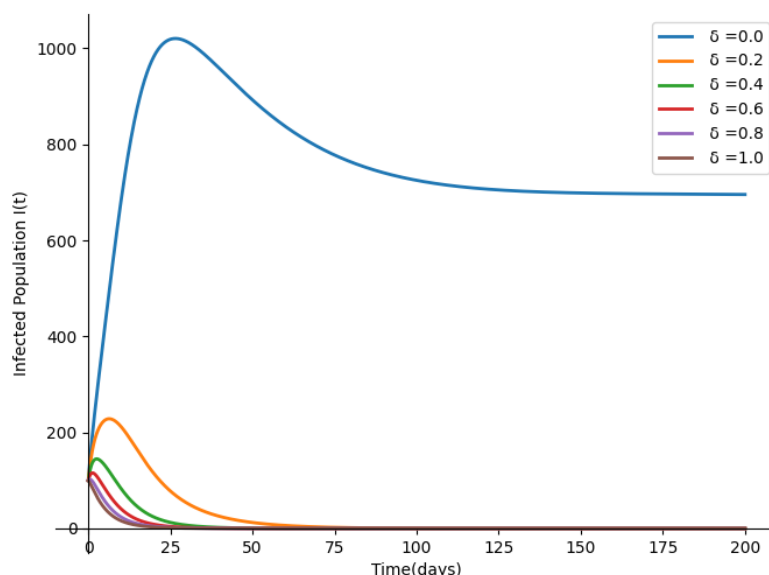


Figure a: Deterministic Case

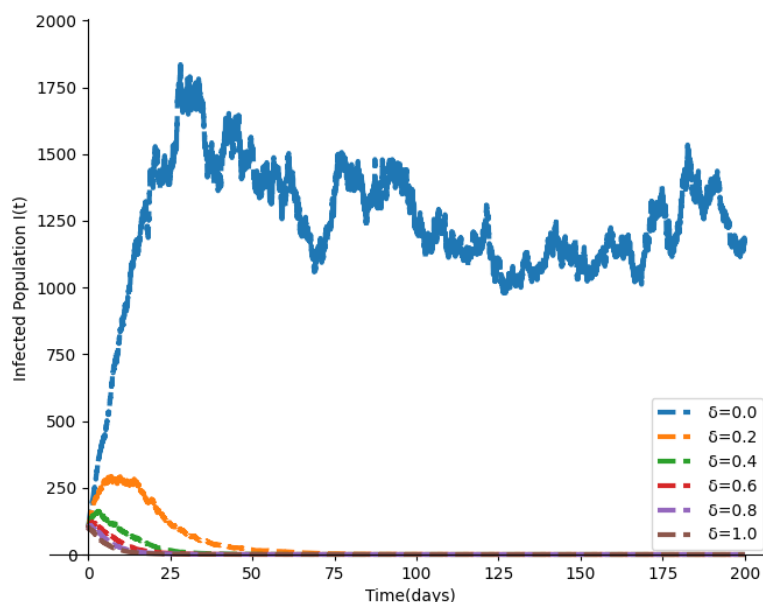


Figure b: Stochastic Case

Figure 12: Effects of treatment rate on infected population $I(t)$

Here, the results of the numerical simulation showing the impact of the treatment rate (δ) on the number of infectious individuals $I(t)$ are presented. The results displayed in Fig. 12 are obtained by varying the vaccination rate from $\delta = 0.0$ to $\delta = 1.0$, while keeping all other model parameters constant.

5 Discussion and Conclusion

This study developed and analyzed a seven-compartment deterministic-stochastic (jump-diffusion) model for typhoid fever transmission by integrating Brownian diffusion process (ΔW_n) and Poisson jump process (ΔN_n) into the infected, treated, and recovered compartments. The motivation for this hybrid framework stems from the observation that typhoid fever transmission in endemic regions is rarely governed by smooth deterministic dynamics alone ([15, 35]). Environmental contamination, disruptions in healthcare delivery, population movement, and sudden failures in sanitation infrastructure frequently generate abrupt changes in the occurrence of typhoid disease which cannot be adequately represented by classical deterministic models. The proposed jump-diffusion framework therefore provides a more realistic representation of typhoid epidemiology by simultaneously capturing continuous random fluctuations and discrete outbreak shocks via ΔW_n and ΔN_n respectively.

The deterministic analysis established the positivity, boundedness, and epidemiological feasibility of the model, guaranteeing that all state variables remain biologically meaningful throughout the simulation period. The existence of both typhoid-free and endemic equilibria demonstrates that the disease may either be eradicated or persist depending on the prevailing epidemiological conditions. Furthermore, the derived basic reproduction number R_0 serves as a threshold parameter governing disease persistence. The local stability analysis confirmed that the disease-free equilibrium is locally asymptotically stable whenever $R_0 < 1$ and unstable whenever $R_0 > 1$, a result consistent with classical epidemic theory and previous deterministic typhoid models.

The estimated value of $R_0 = 1.533$ indicates that typhoid fever remains endemic under the baseline parameter assumptions. Biologically, this implies that each infectious individual generates more than one secondary infection, thereby sustaining disease transmission within the population. This result agrees with previous findings reported in deterministic typhoid studies ([8]) where inadequate sanitation, delayed treatment, and insufficient vaccination coverage were identified as major contributors to disease persistence. The result further suggests that current intervention efforts may be insufficient to achieve elimination in regions with similar epidemiological characteristics.

The sensitivity analysis revealed important epidemiological insights. The symptomatic transmission rate β_1 exhibited the largest positive sensitivity index, indicating that disease spread is primarily driven by symptomatic infectious individuals. This finding emphasizes the importance of rapid diagnosis, isolation, and treatment of symptomatic cases. Although asymptomatic transmission (β_2) contributed less to disease spread, its positive sensitivity index demonstrates that asymptomatic individuals remain epidemiologically significant reservoirs of infection. Such individuals may unknowingly contaminate food and water sources, thereby sustaining hidden chains of transmission. The vaccination parameter ν emerged as one of the strongest negative determinants of R_0 , indicating that increased vaccination coverage substantially suppresses disease spread. Numerical results showed that increasing vaccination rates reduced R_0 from approximately 1.53 to below unity, demonstrating the potential of vaccination programs to drive disease elimination. This finding supports recommendations by the World Health Organization ([20]) advocating expanded typhoid conjugate vaccine deployment in endemic regions.

The recovery parameter γ_1 also displayed strong negative sensitivity, implying that improved treatment outcomes significantly reduce disease burden. This result highlights the critical role of healthcare accessibility, early diagnosis, and effective antimicrobial therapy. Conversely, the relatively low treatment rate (δ) used in the baseline scenario suggests delayed healthcare-seeking behavior and limited treatment availability. Such conditions are common in many low-income communities where healthcare infrastructure remains inadequate. The global PRCC analysis further confirmed the dominant influence of β_1 , τ , and μ in promoting transmission, while recovery and vaccination parameters were found to suppress disease spread. Unlike local sensitivity measures, the PRCC analysis evaluates parameter influence over a wider range of uncertainty, thereby strengthening the robustness of these findings. The agreement between local and global sensitivity analyses increases confidence in the identified control priorities.

One of the major contributions of this study lies in the introduction of stochastic jump-diffusion effects into typhoid fever modeling. The stochastic reproduction number R_{jp} derived in this work extends the classical threshold concept by explicitly incorporating environmental variability and outbreak shocks. Interestingly, the diffusion term was found to reduce disease transmission through the negative contribution of the volatility parameter. This phenomenon suggests that random environmental fluctuations may occasionally interrupt transmission chains and suppress disease persistence. In contrast, the jump component was found to amplify transmission. The jump amplitude (J_I , J_R , and J_T , and jump frequency (λ_I , λ_R , and λ_T ,) parameters represent sudden outbreak events such as contamination of public water supplies, flooding, breakdowns in sanitation systems, population displacement, or disruptions in healthcare services. Numerical results demonstrated that increasing jump intensity significantly increased R_{jp} , often beyond the deterministic threshold. This finding is epidemiologically important because it suggests that rare but severe contamination events may have greater influence on disease persistence than gradual transmission mechanisms.

The comparison between deterministic and stochastic simulations revealed substantial differences in disease behavior. While the deterministic model predicted smooth convergence toward endemic equilibrium, the jump-diffusion model generated multiple outbreak peaks, abrupt fluctuations, and irregular epidemic trajectories. Such patterns closely resemble real-world typhoid outbreaks observed in endemic regions ([50]) where disease incidence often fluctuates unpredictably due to environmental and socioeconomic factors. Particularly noteworthy is the behavior of the recovered compartment, which exhibited the largest stochastic fluctuations. This result suggests that recovery dynamics are highly sensitive to sudden improvements or disruptions in healthcare delivery. The infected and treated compartments also demonstrated moderate jump behavior, reflecting sudden outbreaks and emergency treatment interventions. In contrast, susceptible and vaccinated populations evolved relatively smoothly because they are largely governed by demographic processes and planned public health programs rather than abrupt epidemic shocks.

The numerical experiments further demonstrated that strong diffusion effects can eradicate positive jump effects and drive R_{jp} below unity, even when the deterministic model predicts persistence. This observation has significant practical implications. It indicates that strengthening surveillance systems, increasing healthcare responsiveness, and maintaining adaptive intervention strategies can effectively dampen the impact of sudden outbreak events. However, when jump amplitudes (J_I, J_R , and J_T) and frequencies (λ_I, λ_R , and λ_T) become sufficiently large, disease transmission can remain persistent despite substantial control efforts. From a public health perspective, the results suggest that typhoid control strategies should not rely solely on constant interventions. Instead, adaptive and responsive control mechanisms are required. Public health authorities should prioritize rapid outbreak detection, emergency response planning, water quality monitoring, sanitation infrastructure improvement, and vaccination campaigns.

Special attention should also be directed toward asymptomatic carriers, whose role in disease transmission is often underestimated. A notable strength of this work is that it bridges the gap between deterministic epidemic models and real-world outbreak behavior. While previous typhoid studies have focused primarily on deterministic, optimal control, or fractional-order frameworks, the present study explicitly incorporates both continuous randomness and sudden outbreak shocks. This allows the model to capture epidemic phenomena that are commonly observed but poorly represented in traditional frameworks. Nevertheless, some limitations remain. The model assumes homogeneous mixing of the population and does not account for spatial heterogeneity, age structure, antimicrobial resistance, climatic variability, or socioeconomic disparities. In addition, parameter values were partly obtained from literature and assumptions rather than large-scale epidemiological datasets.

Future studies should therefore focus on parameter estimation using real surveillance data, incorporation of spatial transmission networks, antimicrobial-resistant typhoid strains, climate-driven environmental forcing, and integration with machine learning and artificial intelligence techniques for real-time outbreak forecasting. Overall, the study demonstrates that jump-diffusion modeling provides a powerful framework for understanding and predicting typhoid fever transmission under realistic environmental and epidemiological conditions. The findings highlight the necessity of adaptive public health interventions capable of responding to both gradual transmission processes and sudden outbreak shocks. Future research should extend this framework to include spatial heterogeneity, antimicrobial resistance, climate variability, optimal control, and hybrid artificial intelligence systems for real-time epidemic forecasting and decision support.

Declarations

Ethical approval Not Applicable

Consent to Participate

Not Applicable

Consent to Publication

No consent Needed

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Data Availability

All data used in this work are presented in the tables given in the work

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Conflict of Interest

All the authors have declared that there is no conflict of interest.

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Appendix

6.1 Preliminaries

The following preliminaries are given to support models that are driven by jump-diffusion processes, thereby presenting the basic concept in stochastic calculus, which forms the basis for the models used in this paper.

Definition 6.1 *Filtered Probability Space ([44])*

Let $(\Omega, \mathcal{F}, P, F)$ be a quadruple that comprises all possible results of Ω in a random experiment, where $\mathcal{F}$ is the σ -algebra, containing subsets of Ω , P , a probability measure, such that $P(\Omega) = 1$ and $F = \{\mathcal{F}_t : t \in [0, \infty]\}$, a collection of information of events from $\mathcal{F}$ to time $t \geq 0$, then $(\Omega, \mathcal{F}, P, F)$ is a filtered probability space.

Remark 6.1.1 *In this model, it is assumed that the outbreak of the typhoid disease on the infected, treated and recovered classes follow a stochastic process $(X_t(\omega), \omega \in \Omega)$ defined on $(\Omega, \mathcal{F}, P, F)$, with some jumps which satisfies the following conditions:*

- $X_t(\omega)$ is measurable.
- $X_t(\omega)$ is adapted to filtration and this implies that F is adapted.
- $X_t(\omega)$ is integrable $\int_{\Omega} |X_t(\omega)| dP(\omega) = \int_{\mathbb{R}} |x| dP(x) < \infty$
- $X_t(\omega)$ is stochastically continuous such that $\lim_{s \rightarrow t} P|X_t(\omega) - X_s(\omega)| > \epsilon = 0, \epsilon > 0$
- $X_t(\omega)$ is a càdlàg ie, $X_{t-}(\omega) = X_{t+}(\omega)$ such that $X_{t-}(\omega) = \lim_{s \rightarrow t^-}, \quad X_{t+}(\omega) = \lim_{s \rightarrow t^+}$
- $X_t(\omega)$ is a jump process with jump size J , such that the occurrence of jumps in the infected, treated and removed classes are Poisson processes N_t^I, N_t^T and N_t^R respectively.

Definition 6.2 *Jump-diffusion Process ([38, 31] [4])*

A stochastic process $X_t(\omega)$ is said to be a jump-diffusion process if its dynamics is defined as:

$$dX_t = \tilde{\mu}(t, X_t)dt + \tilde{\sigma}(t, X_t)dW_t + \int_{\mathbb{R}^d} \tilde{\nu}(t, X_t, \gamma)\tilde{N}(dt, d\gamma) \quad (6.1)$$

where, $\tilde{\mu}(t, X_t) : [0, T] \times \mathbb{R}^n$ is the drift, $\tilde{\sigma}(t, X_t) : [0, T] \times \mathbb{R}^n \rightarrow \mathbb{R}^{n \times m}$, is the diffusion coefficient, W_t is a standard Brownian motion defined on $(\Omega, \mathcal{F}, P, F)$ and $\tilde{\nu}(t, X_t, \gamma) : [0, T] \times \mathbb{R}^n \times \mathbb{R}^m \rightarrow \mathbb{R}^n$ is a function that determines the jump size, $\tilde{N}(dt, d\gamma) = N(dt, d\gamma) - \beta(d\gamma)dt$ is the compensated Poisson random measure given that $N(dt, d\gamma)$ accounts for the number of jump size $\gamma \in \mathbb{R}^d$ and $\beta(d\gamma)$ is the Levy measure (distribution of jump size γ)

Remark 6.1.2 The last term in the SDE (1) above can be written as:

$$\sum_{i=1}^{N_t} \tilde{\nu}(t, X_t^-, \gamma) dN_t \quad (6.2)$$

Lemma 6.1.1 (Itô's Formula for jump-diffusion process ([44]))

Let $Y = f(t, X_t)$, such that $f \in C^{1,2}([0, T] \times \mathbb{R}^n)$, then,

$$\begin{aligned} df(t, X_t) &= f_t(t, X_t)dt + \nabla f(t, X_t)^\top \tilde{\mu}(t, X_t)dt + \frac{1}{2} \text{Tr}[\sigma(t, X_t^-) H_f(t, X_t^-) \tilde{\sigma}(t, X_t)]dt \\ &\quad + \nabla f(t, X_t)^\top \tilde{\sigma}(t, X_t) dW_t \\ &\quad + \int_R^d [f(t, X_t + \nu(t, X_t, \gamma)) - f(t, X_t) - \nabla f(t, X_t)^\top \nu(t, X_t, \gamma)] \beta(d\gamma)dt \end{aligned} \quad (6.3)$$

where,

$\nabla f(t, X_t)$ is the gradient of f with respect to x

$H_f(t, X_t^-)$ is the Hessian matrix of f_{xx}

$\text{Tr}[\cdot]$ denote the trace of a matrix.