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A deterministic-stochastic modeling of typhoid fever: Integrating jump-diffusion for epidemic control and decision support

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Abstract

Environmental and behavioral changes that lead to random contact variations and sudden discrete events such as outbreaks, spikes, policy changes, and shocks of the healthcare system in typhoid fever are so common that existing deterministic-epidemiological models can no longer explain the dynamics of these realities. This study has therefore filled the existing gap, by developing a deterministic-stochastic modeling framework using the jump-diffusion process in a seven compartmental model in which the infected, treated, and recovery classes are driven by fluctuations and jumps in the typhoid fever spread dynamics. The analysis of the deterministic part of the model showed positive and well posedness conditions. In addition, typhoid-free and endemic equilibria, reproduction numbers of the deterministic and jump diffusion models were obtained. The conditions for the reducibility of the nonlinear jump-diffusion typhoid-endemic models and solutions of the reduced linear stochastic differential equations via the Itô's formula were obtained. In the absence of these realities, the results show a retention of typhoid fever due to the assumed high level of population growth, a higher level of transmission from symptomatic individuals compared to individuals without symptoms; and a very slow rate of treatment depicting a high

level of sensitivity by the recovery rate. The estimated rates in the jump-diffusion scenario show moderate variability in contact rates, treatment outcomes, and recovery patterns, as well as a **30%**-sudden spike in the outbreak of typhoid fever. A comparison of deterministic and jump-diffusion dynamics shows that the recovered population has the highest number of fluctuations and jump sizes, while the infected and treated dynamics portrayed sudden mini-outbreaks and treatment interventions, respectively. In contrast, the susceptible, exposed, asymptomatic and vaccinated population curves show minimal randomness with few jumps. The results on the stochastic reproduction number R_{jp} is driven by the interaction between diffusion and jump processes. The diffusion parameter in R_{jp} was found to reduce the disease transmission even when the deterministic model predicts persistence. The jump parameters in R_{jp} introduced sudden changes that suppressed or amplified transmission. The study therefore concludes that the jump-diffusion is a better framework for the random realities in the typhoid fever dynamics and recommends non-constant health care responses for sudden mini-outbreaks, investment in hospitals, medications, and early detection of asymptomatic cases.

Keywords: Deterministic - Stochastic Modeling, Stability Analysis, Typhoid, Sensitivity Analysis, Numerical Simulation

1 Introduction

Communicable diseases ([8, 9, 14, 16, 18, 53]) continue to be significant threats to human populations with immense effects on health and social economic well-being. Coronavirus ([9]), typhoid fever ([26]), tuberculosis ([49]), and HIV/AIDS ([40]), are some of the infections that have adversely affected human lives. Typhoid fever, as one of such diseases, has been a serious global public health issue until now. A significant threat, especially in low- and middle-income countries with poor sanitation and access to clean water, is typhoid fever caused by the bacterium *Salmonella Typhi* ? [17, 19, 22, 51]. The disease has high morbidity and mortality rates ([32, 42, 43]), caused by food and water contamination, which are the main modes of transmission of the disease. Even with improved medical treatment and vaccination, there are periodic outbreaks caused by environmental, socioeconomic, and infrastructural influences ([13, 52]).

Mathematical modeling is an effective instrument for investigating the nature of transmission of typhoid fever ([21, 27, 34]) and evaluating successful control measures. Deterministic epidemiological models provide a systematic approach to the analysis of disease development based on the susceptible, exposed, infected, and recovered compartments. The models are based on continuous populations with predictable disease dynamics and thus are applicable to search for long-term trends and policy evaluation. However, real-life disease spread is determined by natural randomness, sudden outbreak (which may be caused by sudden financial constraints resulting in human consumption of contaminated food and water), and unanticipated external

variables such as fluctuations caused by uncertainties in human behavior, environmental circumstances, and health measures by the population.

Stochastic modeling has become relevant to solving these uncertainties in the study of infectious diseases. Stochastic models are based on randomness and probabilistic factors, and epidemic variations can be more realistically represented. Specifically, jump-diffusion models that are a hybrid of the Gaussian noise (Brownian motion) and non-Gaussian jumps offer a solid framework to describe sudden rises in the rate of infections caused by environmental contamination, population movement, or vaccine lapses. By using a combination of deterministic and stochastic elements, a better understanding of the dynamics of typhoid fever would be gained and its intervention approach more effective [12, 25, 54].

Numerous studies [12, 19, 24, 25, 37, 44, 47, 48, 54]) have been conducted in the context of deterministic modeling, including epidemiological diseases and how they affect peoples' lives. These include Mathematical models, which have been widely used to describe the problem of communicable infection's spread. Despite these numerous efforts to obtain measures to curtail the spread of typhoid fever, there have been noticeable threats to human lives. In view of this, Lawal *et al* (2024) in [26] and Tijani *et al* (2023) in [46] developed deterministic compartmental optimal control models that incorporated hygiene and environmental factors such as environmental sanitation, personal hygiene, and treatment controls. The findings based on the analysis of their work proved that a combination of the above mentioned is most effective. The focus on increasing control measures in deterministic typhoid fever models would further enhance better results, spawning the work of Asamoh *et al* (2025), in [11]. The findings on the five control measures in [11], namely vaccination, booster vaccination, hygiene, treatment, and bacteria sterilization, showed that a combined strategy is better than a single intervention. Deterministic fractional order typhoid models have been applied to the dynamics of the spread of the disease in the works of [39], [30], [55] and [56]. Validations of the work in [39], [30], [55] and [56] have been achieved using various numerical methods such as in [57], [59] and [15].

It can be clearly seen that the deterministic models of typhoid fever are well established in the extant literature. However, there are stochastic models of the spread of typhoid fever, but they are limited. The existing stochastic models which have been used to model the spread of diseases can be categorised into three. The first category is agent-based stochastic-simulation models ([36]), which capture random infection events, variability in transmission dynamics, and randomness in disease persistence. Stochastic differential models as in [58]- wherein stochastic modeling of Typhoid fever via transition probabilities and Markov processes were used, in the second category. The third category are hybrid Artificial Intelligence stochastic computational models in [60], [61], which combines existing models with AI tools for predictive purposes. The reoccurrence of typhoid fever in the Sub-Saharan Africa region and other parts of the world ([29], [35], [45]) as a one-time epidemic is rare, instead it has been found to be endemic in the population with random outbreaks and spikes in different locations

due to environmental and hygiene factors that lead to sudden water contamination events. Although, to the best of the current literature, stochastic-jump diffusion work is common in the COVID-19 models ([62]), malaria([5]), and financial mathematics inspired models ([23], [31], [3]), there are little or no results for the dynamics of transmission of typhoid fever. We are therefore propelled to build better modeling tools for a better outlook of the real life dynamics of disease spread. In this study, we develop a hybrid deterministic-jump diffusion (stochastic) epidemiological model for typhoid fever that incorporates small random fluctuations of events of infections (driven by the Brownian term) and sudden outbreak of the disease caused by environmental contamination events(driven by the jumps), e.g, water contamination, etc, in the infected, treated, and recovered classes of the population. The deterministic component captures the average behavior of disease transmission, while the stochastic jump-diffusion framework accounts for sudden outbreak events and variations. By simulating the model with standard control measures such as vaccination, treatment, and recovery, the effectiveness of these interventions was evaluated under both deterministic and stochastic conditions. This approach provided insight into disease persistence, eradication feasibility, and potential public health responses without relying on optimal control strategies.

This research has contributed to the growing body of literature on infectious disease modeling by bridging deterministic and stochastic approaches to better inform public health interventions. The findings have provided insight into how sudden changes in infection rates and availability of treatment impact disease control, ultimately supporting more adaptive and responsive public health policies for the management of typhoid fever. This research aims to provide answers to the following questions: How did deterministic and stochastic factors influence the transmission dynamics of typhoid fever? What was the impact of vaccination, treatment, and recovery on controlling typhoid fever in both deterministic and jump-diffusion settings? How did sudden outbreaks and variations in medical interventions affect long-term disease control? How did the stability analysis using the Jacobian matrix inform the feasibility of eradicating typhoid fever? To provide answers, the study have formulated a seven compartmental deterministic-jump diffusion model by incorporating the Gaussian and non-Gaussian (jump process) noise to model random fluctuations and sudden outbreak of the typhoid fever into the infected, treated, and recovered compartments. In the absence of variability due to random fluctuations in the outbreak, the feasibility and positivity were calculated to account for the well-posedness of the formulated model. In addition, the free and endemic-typhoid equilibrium was estimated to determine the conditions of existence and uniqueness of the endemic equilibrium. The basic reproduction number and the local stability of the model were determined to determine the rate of persistence and extinction of the disease. Also, in the presence of variability due to random fluctuations and jumps in infected, treated, and recovered classes, the conditions to reduce the nonlinear jump-diffusion typhoid endemic model were determined. This was done to prove that nonlinear SDEs are reducible to linear forms. An explicit solution of the linearized stochastic model (jump-diffusion) was obtained using Itô's formula. Numerical simulations were performed in two scenarios.

The remaining part of the work is structured as follows: The next section contains the materials and methods used in this work, the basic preliminaries to solving stochastic differential equations with jumps are spelled out in this section. The formulated model and its solutions are given in the deterministic and stochastic perspectives. The sensitivity analysis of the parameters in the model is explicitly given in section three of the paper. The numerical simulation, discussion, and conclusion are presented in sections four and five.

2 Materials and Methods

2.1 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 1 Filtered Probability Space ([38])

Let $(\Omega, \mathcal{F}, \mathbb{P}, \mathbb{F})$ 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 , $\mathbb{P}$, a probability measure, such that $\mathbb{P}(\Omega) = 1$ and $\mathbb{F} = \{\mathcal{F}_t : t \in [0, \infty]\}$, a collection of information of events from $\mathcal{F}$ to time $t \geq 0$, then $(\Omega, \mathcal{F}, \mathbb{P}, \mathbb{F})$ is a filtered probability space.

Remark 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 $(\Omega, \mathcal{F}, \mathbb{P}, \mathbb{F})$, with some jumps which satisfies the following conditions:

- $X_t(\omega)$ is measurable.
- $X_t(\omega)$ is adapted to filtration and this implies that $\mathbb{F}$ is adapted.
- $X_t(\omega)$ is integrable $\int_{\Omega} |X_t(\omega)| d\mathbb{P}(\omega) = \int_{\mathbb{R}} |x| d\mathbb{P}(x) < \infty$
- $X_t(\omega)$ is stochastically continuous such that $\lim_{s \rightarrow t} \mathbb{P}|X_t(\omega) - X_s(\omega)| > \epsilon = 0, \epsilon > 0$
- $X_t(\omega)$ is a càdlàg ie, $X_{t-}(\omega) = X_{t+}(\omega)$ such that $X_{t-}(\omega) = \lim_{s \rightarrow t, s < t}, X_{t+}(\omega) = \lim_{s \rightarrow t, s > t}$
- $X_t(\omega)$ 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 2 Jump-diffusion Process ([23, 31] [4])

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

$$dX_t = \tilde{\mu}(t, X_t)dt + \tilde{\sigma}(t, X_t)dW_t + \int_{\mathbb{R}^d} \tilde{\nu}(t, X_t, \gamma)\tilde{N}(dt, d\gamma) \quad (1)$$

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 $(\Omega, \mathcal{F}, \mathbb{P}, \mathbb{F})$ 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 2 The last term in the SDE (1) above can be written as:

$$\sum_{i=1}^{N_t} \tilde{\nu}(t, X_t^-, \gamma) dN_t \quad (2)$$

Theorem 1 (Itô's Formula for jump-diffusion process ([38]))

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

$$\begin{aligned} df(t, X_t) = & f_t(t, X_t)dt + \nabla f(t, X_t)^\top \tilde{\mu}(t, X_t)dt + \frac{1}{2} \text{Tr}[\sigma(t, X_t^-) H_f(t, X_t^-) \tilde{\sigma}(t, X_t)]dt \\ & + \nabla f(t, X_t)^\top \tilde{\sigma}(t, X_t) dW_t + \int_{\mathbb{R}} [f(t, X_t + \nu(t, X_t, \gamma)) \\ & - f(t, X_t) - \nabla f(t, X_t)^\top \nu(t, X_t, \gamma)] \beta(d\gamma) dt \end{aligned} \quad (3)$$

$$- f(t, X_t) - \nabla f(t, X_t)^\top \nu(t, X_t, \gamma)] \beta(d\gamma) dt \quad (4)$$

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.

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

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - (\lambda + \mu + \nu)S + \alpha V + \phi R, \\ \frac{dV}{dt} &= \nu S - (\alpha + \mu)V, \\ \frac{dE}{dt} &= \lambda S - \tau \sigma E - (1 - \tau) \sigma E - \mu E, \\ \frac{dI}{dt} &= \tau \sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I, \\ \frac{dA}{dt} &= (1 - \tau) \sigma E - (\gamma_2 + \mu)A, \\ \frac{dT}{dt} &= \delta I - (\gamma_3 + \mu)T, \\ \frac{dR}{dt} &= \gamma_1 I + \gamma_2 A + \gamma_3 T - (\mu + \phi)R. \end{aligned} \quad (5)$$

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.

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

2.3 Formulation of Deterministic-Jump diffusion Typhoid Model

The deterministic-stochastic 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):

$$\begin{aligned}
dS &= (\Lambda - (\lambda + \mu + \nu)S + \alpha V + \phi R) dt, \\
dV &= (\nu S - (\alpha + \mu)V) dt, \\
dE &= (\lambda S - (\sigma + \mu)E) dt, \\
dI &= (\tau \sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I) dt + \eta_I I dW_t + J_I I dN_t, \\
dA &= ((1 - \tau) \sigma E - (\gamma_2 + \mu)A) dt, \\
dT &= (\delta I - (\gamma_3 + \mu)T) dt + \eta_T T dW_t + J_T T dN_t, \\
dR &= (\gamma_1 I + \gamma_2 A + \gamma_3 T - (\mu + \phi)R) dt + \eta_R R dW_t + J_R R dN_t.
\end{aligned} \tag{6}$$

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

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.

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 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.

2.3.1 Schematic Diagram

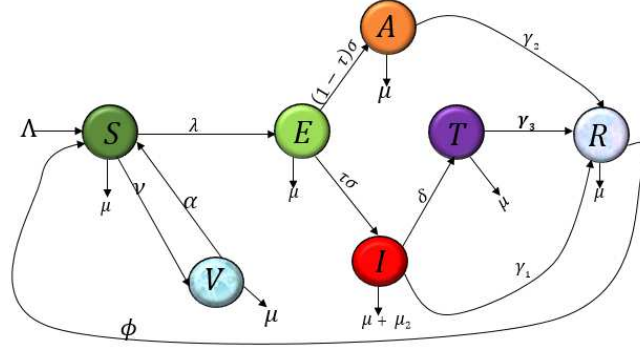


Fig. 1: Schematic Diagram for Typhoid Fever

2.4 Dynamic analysis and solutions of the deterministic typhoid model

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

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - (\lambda + \mu + \nu)S + \alpha V + \phi R, \\
\frac{dV}{dt} &= \nu S - (\alpha + \mu)V, \\
\frac{dE}{dt} &= \lambda S - \tau \sigma E - (1 - \tau)\sigma E - \mu E, \\
\frac{dI}{dt} &= \tau \sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I, \\
\frac{dA}{dt} &= (1 - \tau)\sigma E - (\gamma_2 + \mu)A, \\
\frac{dT}{dt} &= \delta I - (\gamma_3 + \mu)T, \\
\frac{dR}{dt} &= \gamma_1 I + \gamma_2 A + \gamma_3 T - (\mu + \phi)R.
\end{aligned} \tag{7}$$

2.4.1 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 $\mu_2 = 0$

$$\therefore, \quad \frac{dN}{dt} = \Lambda - \mu N \tag{8}$$

On solving (8) we have

$$N(t) \leq \frac{\Lambda}{\mu} \tag{9}$$

2.4.2 Positivity of the solution

From the first equation in (7)

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - (\lambda + \mu + \nu)S + \alpha V + \phi R \\
\frac{dS}{dt} &\geq -(\lambda + \mu + \nu)S
\end{aligned} \tag{10}$$

After solving (10) we have

$$S(t) \geq S(0)e^{-(\lambda + \mu + \nu)t} \geq 0 \quad \text{since } (\lambda + \mu + \nu) > 0 \tag{11}$$

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

2.4.3 Typhoid - free Equilibrium

From system (7) at disease-free equilibrium, setting $I^+ = 0$, $E^+ = I^+ = A^+ = T^+ = R^+ = 0$ it is reduced to

$$\begin{aligned}\Lambda - (\mu + \nu)S^+ + \alpha V &= 0 \\ \nu S^+ - (\alpha + \mu)V &= 0\end{aligned}\tag{12}$$

In solving (12) simultaneously, it follows from the second equation, that:

$$V^+ = \frac{\nu S^+}{(\mu + \nu)}\tag{13}$$

Putting (13) into first equation of (12) we have

$$S^+ = \frac{\Lambda(\alpha + \mu)}{\mu(\alpha + \mu + \nu)}\tag{14}$$

Replace S^+ in (13) with (14) to obtain

$$V^+ = \frac{\Lambda\nu}{\mu(\alpha + \mu + \nu)}\tag{15}$$

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

2.4.4 Typhoid endemic equilibrium

Here, the condition for the existence and uniqueness of the endemic equilibrium of model system equations (7) 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 7 must not be zero at this equilibrium state i.e $E^* = (S^*, V^*, E^*, I^*, A^*, T^*, R^*) \neq (0, 0, 0, 0, 0, 0, 0)$:

In order to obtain the endemic equilibrium state, each equation (7) is equated to zero and solve it simultaneously

$$\begin{aligned}\Lambda - (\lambda + \mu + \nu)S^* + \alpha V^* + \phi R^* &= 0, \\ \nu S^* - (\alpha + \mu)V^* &= 0, \\ \lambda S^* - \tau\sigma E^* - (1 - \tau)\sigma E^* - \mu E^* &= 0, \\ \tau\sigma E^* - (\gamma_1 + \delta + \mu_2 + \mu)I^* &= 0, \\ (1 - \tau)\sigma E^* - (\gamma_2 + \mu)A^* &= 0, \\ \delta I^* - (\gamma_3 + \mu)T^* &= 0, \\ \gamma_1 I^* + \gamma_2 A^* + \gamma_3 R^* - (\mu + \phi)R^* &= 0.\end{aligned}\tag{16}$$

to obtain

$$\mathbb{E}^* = \begin{cases} S^* = \frac{\wedge a_5 a_3 a_7}{[a_3 a_7 (a_5 a_6 + \mu \nu) - \lambda \phi \tau \sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta \tau a_3)]} \\ V^* = \frac{\wedge \nu a_3 a_7}{[a_3 a_7 (a_5 a_6 + \mu \nu) - \lambda \phi \tau a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta \tau a_3)]} \\ E^* = \frac{\wedge \lambda a_5 a_3}{[a_3 a_7 (a_5 a_6 + \mu \nu) - \lambda \phi \tau \sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta \tau a_3)]} \\ I^* = \frac{\wedge \lambda \tau \sigma a_5}{[a_3 a_7 (a_5 a_6 + \mu \nu) - \lambda \phi \tau \sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta \tau a_3)]} \\ A^* = \frac{\wedge \lambda \tau \sigma a_5 (1 - \tau) a_3}{\tau a_2 [a_3 a_7 (a_5 a_6 + \mu \nu) - \lambda \phi \tau \sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta \tau a_3)]} \\ T^* = \frac{\wedge \delta \lambda}{a_3 [a_3 a_7 (a_5 a_6 + \mu \nu) - \lambda \phi \tau \sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta \tau a_3)]} \\ R^* = \frac{\wedge \lambda a_5 \sigma [\gamma_1 \tau a_1 a_2 + \gamma_2 a_1 (1 - \tau) a_2 + \gamma_3 \delta \tau a_3]}{a_1 a_2 a_4 [a_3 a_7 (a_5 a_6 + \mu \nu) - \lambda \phi \tau \sigma a_5 (\gamma_2 a_1 a_2 + \gamma_3 \delta \tau a_3)]} \end{cases} \quad (17)$$

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 (7).

2.4.5 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

$$\mathcal{E}_0 = (S^*, V^*, 0, 0, 0, 0, 0),$$

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

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

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

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

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,

$$\begin{aligned} R_0 &= \frac{\alpha + \mu}{\alpha + \mu + \nu} \left[\frac{\beta_1 \tau \sigma}{(\sigma + \mu)(\gamma_1 + \delta + \mu_2 + \mu)} + \frac{\omega \beta_2 (1 - \tau) \sigma}{(\sigma + \mu)(\gamma_2 + \mu)} \right], \\ R_0 &= \frac{\sigma(\alpha + \mu) [\beta_1 \tau (\gamma_2 + \mu) + \omega \beta_2 (1 - \tau) (\gamma_1 + \delta + \mu_2 + \mu)]}{(\sigma + \mu)(\alpha + \mu + \nu)(\gamma_1 + \delta + \mu_2 + \mu)(\gamma_2 + \mu)}. \end{aligned} \quad (18)$$

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 $\mathcal{E}_0$ 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 $\mathcal{E}_0$ 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 $\mathcal{E}_0$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

2.4.6 Local Stability of the Deterministic of the Model

To determine the stability of the system (7) at the disease-free equilibrium point $\mathbb{E}^0$, 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 The disease-free equilibrium point $\mathbb{E}^0$ is locally asymptotically stable if $R_0 < 1$ for $tr(J_{\mathbb{E}^0}) < 0$ and $det(J_{\mathbb{E}^0}) > 0$, and unstable if $R_0 > 1$ for $tr(J_{\mathbb{E}^0}) > 0$ and $det(J_{\mathbb{E}^0}) < 0$.

Proof. The proof of the theorem can be established by constructing a Jacobian matrix for the model system (7) evaluated at the DFE point $\mathbb{E}^0$.

At DFE, $I^0 = 0$.

$$J_{\mathbb{E}^0} = \begin{pmatrix} -P_1 & \alpha & 0 & -G & -\omega H & 0 & 0 \\ \nu & -P_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -P_3 & G & \omega H & 0 & 0 \\ 0 & 0 & \tau \sigma & -P_4 & 0 & 0 & 0 \\ 0 & 0 & (1-\tau) \sigma & 0 & -P_5 & 0 & 0 \\ 0 & 0 & 0 & \delta & 0 & -P_6 & 0 \\ 0 & 0 & 0 & \gamma_1 & \gamma_2 & \gamma_3 & -P_7 \end{pmatrix} \quad (19)$$

In this section, we determine the conditions under which the disease-free equilibrium (DFE) $\mathbb{E}^0$ of the system (7) 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 $\mathbb{E}^0$, denoted by $J_{\mathbb{E}^0}$ (see Eq. (19)), governs the local dynamics near the DFE.

The trace of $J_{\mathbb{E}^0}$ is given by

$$\begin{aligned} \text{tr}(J_{\mathbb{E}^0}) &= -P_1 - P_2 - P_3 - P_4 - P_5 - P_6 - P_7 \\ &= -\sum_{i=1}^7 P_i < 0, \end{aligned} \quad (20)$$

since $P_i > 0$ for all $i = 1, \dots, 7$.

Furthermore, the determinant of $J_{\mathbb{E}^0}$ can be expressed as

$$\det(J_{\mathbb{E}^0}) = P_6 P_7 (P_5 \tau \sigma G + \sigma \omega H P_4 (1 - \tau) - P_5 P_4 P_3) (P_2 P_1 - \nu \alpha), \quad (21)$$

$$\det(J_{\mathbb{E}^0}) = P_6 P_7 (P_2 P_1 - \nu \alpha) P_5 P_4 P_3 [R_0 - 1]. \quad (22)$$

Since all $P_i > 0$, it follows that

$$\det(J_{\mathbb{E}^0}) > 0 \quad \text{whenever} \quad R_0 < 1,$$

provided

$$P_1 P_2 < \nu \alpha.$$

Hence, the disease-free equilibrium $\mathbb{E}^0$ is locally asymptotically stable whenever

$$R_0 < 1. \quad (23)$$

Biologically, this means that the infection dies out and the disease cannot invade the population.

Conversely, if $R_0 > 1$, the disease-free equilibrium becomes unstable and the infection persists in the population.

where,

$$P_1 = \mu + \nu, P_2 = \alpha + \mu, P_3 = \sigma + \mu, P_4 = \gamma_1 + \delta + \mu_2 + \mu, P_5 = \gamma_2 + \mu, P_6 = \gamma_3 + \mu, P_7 = \mu + \phi, G = \frac{\beta_1 S^+}{N}, H = \frac{\beta_2 S^+}{N^+}, S^+ = \frac{\Lambda(\alpha + \mu)}{\mu(\alpha + \mu + \nu)}, N^+ = \frac{\Lambda}{\mu}.$$

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

Consider the system of equations given in (2.3), the influx of randomness driven by W_t and N_t as given in remark 2.1.1 has a direct effect on the compartments I , R , and T . Hence, the nonlinear jump-diffusion typhoid endemic model.

$$\begin{aligned} dI &= (\tau\sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I) dt + \eta_I I dW_t + J_I I dN_t, \\ dR &= (\gamma_1 I + \gamma_2 A + \gamma_3 R - (\mu R + \phi)R) dt + \eta_R R dW_t + J_R R dN_t, \\ dT &= (\delta I - (\gamma_3 + \mu)T) dt + \eta_T T dW_t + J_T T dN_t. \end{aligned} \quad (24)$$

Thus, case 1 of (24) can be written as:

$$dI = \alpha(t, I)dt + \beta(t, I)dW_t + \zeta(t, I)dN_t, \quad (25)$$

where, E^* is as defined in (17), and

$$\begin{aligned} \alpha(t, I) &= (\tau\sigma E^* - (\gamma_1 + \delta + \mu_2 + \mu)I) \\ \beta(t, I) &= \eta_I I \\ \zeta(t, I) &= J_I I \end{aligned} \quad (26)$$

It is clearly seen that (25) is a nonlinear stochastic differential equation with a jump process. The main concern in this work is to determine the conditions for reducing (25) to a linear form and, secondly, to determine a solution of the reduced linear jump-diffusion endemic model.

2.5.1 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 [41] for an SDE without jumps.

The nonlinear SDE with jumps in (25) can be reduced to a linear stochastic typhoid model with jumps in f_t :

$$df_t = (\alpha_1(t)f_t + \alpha_2(t))dt + (\beta_1(t)f_t + \beta_2(t))dW_t + (\zeta_1(t)f_t + \zeta_2(t))dN_t, \quad (27)$$

if, $f_t = g(t, I_t)$ such that $\frac{\partial g(t, I)}{\partial I} \neq 0$ and the solution of (25) has the form

$$I_t = V(t, f_t) \quad (28)$$

where, $f = g(t, V(t, f))$ and $I = g(t, V(t, I))$, given that f satisfies (27).

In what follows, we shall derive a sufficient condition for the reducibility of the stochastic model (25)

Applying Lemma 1 to $g(t, I_t)$, we get the following.

$$\begin{aligned} dg(t, I) = & \left(\frac{\partial g(t, I_{t-})}{\partial t} + \alpha \frac{\partial g(t, I_{t-})}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g(t, I_{t-})}{\partial I^2} \right) dt + \\ & \beta \frac{\partial g(t, I_{t-})}{\partial I} dW_t + \left(g(I_{t-} + \gamma) - g(I_{t-}) - \gamma \frac{\partial g(t, I_{t-})}{\partial I} \right) dN_t \end{aligned} \quad (29)$$

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

Now, eq.(27) can be linked with eq. (29), if

$$\frac{\partial g}{\partial t} + \alpha \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g}{\partial I^2} = \alpha_1 g(t, I_t) + \alpha_2 \quad (30)$$

$$\beta \frac{\partial g(t, I_t)}{\partial I} = \beta_1 g(t, I_t) + \beta_2 \quad (31)$$

$$g(I_{t-} + \gamma) - g(I_{t-}) - c \frac{\partial g}{\partial I} = \zeta_1 g(I_{t-}) + \zeta_2 \quad (32)$$

Consider $\alpha_1(t) = \beta_1(t) = \zeta_1(t) = 0$, then, (30) - (32) becomes:

$$\frac{\partial g}{\partial t} + \alpha \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g}{\partial I^2} = \alpha_2(t) \quad (33)$$

$$\beta \frac{\partial g(t, I_t)}{\partial I} = \beta_2(t) \quad (34)$$

$$g(I_{t-} + \gamma) - g(I_{t-}) - c \frac{\partial g}{\partial I} = \zeta_2(t) \quad (35)$$

Also, for $\alpha_2(t) = \varsigma(t)$, $\beta_2(t) = \kappa(t)$ and $\zeta_2(t) = \nu(t)$, then the above (33) - (35) becomes:

$$\frac{\partial g}{\partial t} + \alpha \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g}{\partial I^2} = \varsigma(t) \quad (36)$$

$$\beta \frac{\partial g(t, I_t)}{\partial I} = \kappa(t) \quad (37)$$

$$g(I_{t-} + \gamma) - g(I_{t-}) - c \frac{\partial g}{\partial I} = \nu(t) \quad (38)$$

From eq. (36) above, we have that:

$$\frac{\partial g}{\partial t} = - \left(\alpha(t, I) \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g}{\partial I^2} \right) + \varsigma(t) \quad (39)$$

Differentiating eq. (39) wrt I , gives:

$$\frac{\partial^2 g}{\partial t \partial I} = - \frac{\partial}{\partial I} \left(\alpha(t, I) \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g}{\partial I^2} \right) \quad (40)$$

Similarly, from eq. (37) and eq. (38)

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

Again, taking the derivative of eq. (37) wrt. t gives:

$$\frac{\partial}{\partial t} \left(\beta \frac{\partial g(t, I_t)}{\partial I} \right) = \frac{\partial}{\partial t} \left(\kappa(t) \right) \implies \beta(t, I) \frac{\partial^2 g}{\partial t \partial I} + \frac{\partial g}{\partial I} \frac{\partial \beta(t, I)}{\partial t} = \kappa'(t) \quad (41)$$

Note that from eq. (37) and eq. (38), $\frac{\partial g}{\partial I} = \frac{\kappa(t)}{\beta(t, I)}$ 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. (41) 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. (41) can be expressed as:

$$\begin{aligned} \kappa'(t) &= -\beta(t, I) \frac{\partial}{\partial I} \left(\alpha(t, I) \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial^2 g}{\partial I^2} \right) + \frac{\partial g}{\partial I} \frac{\kappa(t)}{\beta(t, I)} \\ &= -\beta(t, I) \frac{\partial}{\partial I} \left(\alpha(t, I) \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial}{\partial I} \left(\frac{\partial g}{\partial I} \right) \right) + \frac{\partial g}{\partial I} \frac{\kappa(t)}{\beta(t, I)} \\ &= -\beta(t, I) \frac{\partial}{\partial I} \left(\alpha(t, I) \frac{\partial g}{\partial I} + \frac{1}{2} \beta^2 \frac{\partial}{\partial I} \left(\frac{\kappa(t)}{\beta(t, I)} \right) \right) + \frac{\partial g}{\partial I} \frac{\kappa(t)}{\beta(t, I)} \end{aligned} \quad (42)$$

Upon solving, we obtain:

$$\kappa'(t) = \kappa(t) \Gamma(t, I) \quad (43)$$

In eq. (43), since κ' is independent of I , then,

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

where,

$$\Gamma(t, I) = \frac{1}{\beta(t, I)} \frac{\partial g}{\partial t} - \beta(t, I) \frac{\partial}{\partial I} \left(\frac{\alpha(t, I)}{\beta(t, I)} - \frac{1}{2} \frac{\partial g}{\partial I}(t, I) \right) \quad (44)$$

Eq. (43) is a sufficient condition for the reducibility of the non-linear stochastic model (25) to the explicitly integrable model

$$df_t = \alpha(t)dt + \beta_1(t)dW_t + \zeta_1(t)dN_t, \quad (45)$$

2.5.2 Solution of the linear stochastic typhoid-endemic Model

The reducibility of the nonlinear stochastic model eq. (24) has been established in the previous subsection. In this subsection, the procedure for reducing eq. (24) to its linear form and finding the solution in its linear form is given explicitly.

Recall from eq. (24) that

$$dI = (\tau\sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I)dt + \eta_I IdW_t + J_I IdN_t, \quad (46)$$

Let $\varphi_t = \ln I_t$, by applying the lemma 1, to the logarithmic transformation $g(I) = \ln I_t$, we have:

$$dg(I_t) = g'(I_{t-})d(I_t) + \frac{1}{2}g''(I_{t-})\eta_t^2 I_{t-}^2 dt + \left[g(I_{t-} + \gamma) - g(I_{t-}) - \gamma g'(I_{t-}) \right] dN_t \quad (47)$$

Considering (46) via (46), given that the probability of exposed individuals becoming symptomatic infected tends to zero. Then, the continuous and jump parts are obtained, respectively as:

$$\left(-(\gamma_1 + \delta + \mu_2 + \mu) - \frac{1}{2}\eta_t^2 \right) dt + \eta_t dW_t \quad (48)$$

and

$$\ln(1 + \gamma)dN_t \quad (49)$$

Combining (48) and (49) gives:

$$d\varphi_t = \left(-(\gamma_1 + \delta + \mu_2 + \mu) - \frac{1}{2}\eta_t^2 \right) dt + \eta_t dW_t + \ln(1 + \gamma)dN_t \quad (50)$$

Hence, eq. (50) 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,

$$\varphi_t = \varphi_0 + \left(-(\gamma_1 + \delta + \mu_2 + \mu) - \frac{1}{2}\eta_t^2 \right) t + \eta_t W_t + \ln(1 + \gamma)N_t \quad (51)$$

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

$$\ln I_t = \ln I_0 + \left(-(\gamma_1 + \delta + \mu_2 + \mu) - \frac{1}{2}\eta_t^2 \right)t + \eta_t W_t + \ln(1 + \gamma)N_t \quad (52)$$

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

$$I_t = I_0 \exp\left(-((\gamma_1 + \delta + \mu_2 + \mu) - \frac{1}{2}\eta_t^2)t + \eta_t W_t \right) (1 + \gamma)N_t \quad (53)$$

Remark 3 .

The above procedures can be used to obtain the solutions of T and R in (24)

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

With reference to the non linear jump-diffusion typhoid fever model in (24), 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:

$$dI = (\tau\sigma E - (\gamma_1 + \delta + \mu_2 + \mu)I)dt + \eta_I IdW_t + J_I IdN_t, \quad (54)$$

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, (54) becomes:

$$dI = \hat{\alpha} Idt + \eta_I IdW_t + J_I IdN_t. \quad (55)$$

Applying Itô's lemma to (55) gives:

$$d(\ln I) = \left(\hat{\alpha} - \frac{1}{2}\eta_I^2 \right)dt + \eta_I dW_t + \ln(1 + J_I)dN_t. \quad (56)$$

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:

$$\begin{aligned} \hat{G} &= \mathbb{E}\left(d(\ln I)\right) = \mathbb{E}\left(\left(\hat{\alpha} - \frac{1}{2}\eta_I^2\right)dt + \eta_I dW_t + \ln(1 + J_I)dN_t\right) \\ &\implies \hat{G} = \hat{\alpha} - \frac{1}{2}\eta_I^2 + \lambda_j \ln(1 + J_I). \end{aligned} \quad (57)$$

where, $\mathbb{E}(x)$ is the expected value of x . Also, for $\hat{\alpha} = (\gamma_1 + \delta + \mu_2 + \mu)(R_0 - 1)$ in (57), the jump-diffusion reproduction number (R_{jp}) for typhoid fever is obtained as:

$$R_{jp} = R_0 - \frac{\eta_I^2}{2(\gamma_1 + \delta + \mu_2 + \mu)} + \frac{\lambda_j}{(\gamma_1 + \delta + \mu_2 + \mu)} \ln(1 + J_I) \quad (58)$$

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]	[20, 26, 50]
β_2	0.4 [0 - 1]	Assumed
τ	0.85	[11]
ω	0.85	Assumed
σ	0.4	[20, 26, 28]
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, 11, 19]
μ	0.01	[1, 26, 28, 33, 37]
μ_2	0.02	[2, 19]
α	0.04	Assumed
ν	0.1 [0 - 1]	[1, 37, 50]
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 $R_0^{b,h}$

Definition 3 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, $Z_W^{R_0}$ is the sensitivity index of $R_0^{b,h}$ 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 [6], [9], [10], [63] given as:

$$Z_m^{R_0} = \frac{\partial R_0}{\partial m} \times \frac{m}{R_0} \quad (59)$$

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.

$$\left. \begin{aligned} Z_{\beta_1}^{R_0} &= \frac{\partial R_0}{\partial \beta_1} \times \frac{\beta_1}{R_0} = +0.90160 \\ Z_{\beta_2}^{R_0} &= \frac{\partial R_0}{\partial \beta_2} \times \frac{\beta_2}{R_0} = +0.09836 \\ Z_{\nu}^{R_0} &= \frac{\partial R_0}{\partial \nu} \times \frac{\nu}{R_0} = -0.6666667 \end{aligned} \right\} \quad (60)$$

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 $R_0^{b,h}$ changes from 1.532889875 to 0.3811283567. This shows that the Typhoid fever is controllable.

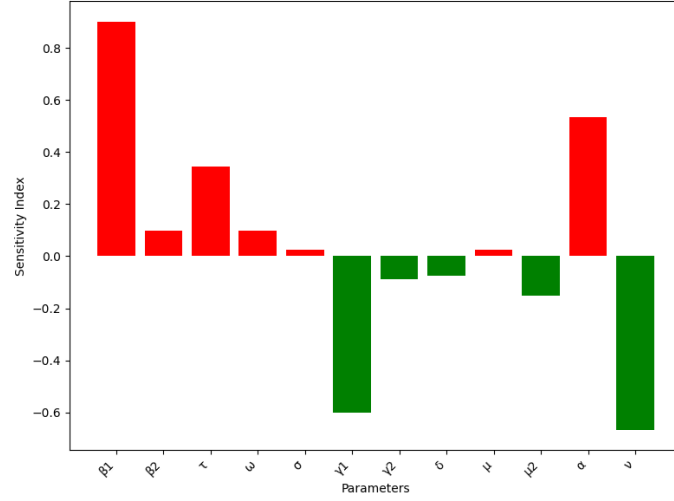


Fig. 2: Sensitivity Analysis of Typhoid Fever

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 Effect of Transmission Rate and Control Parameter on R_0

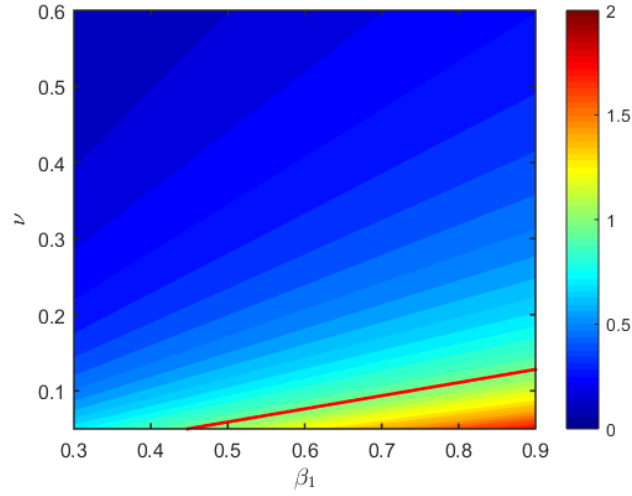


Fig. 3: Heatmap on R_0 vs ν and β_1

In Figure 3 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.

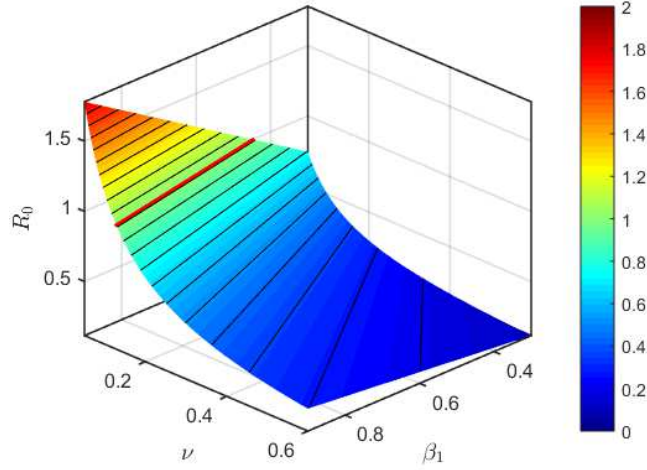


Fig. 4: Surface Plot R_0 vs ν and β_1

In Figure 4 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. This table examines the effect of

Table 6: 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

increasing diffusion intensity η_I while fixing the jump amplitude $J_I = 0.3$ and jump rate $\lambda_J = 0.1$. For $\eta_I = 0$, the stochastic model reduces to the deterministic case, giving $R_{jp} = R_0 = 1.532889875$. For small diffusion ($\eta_I = 0.2$), a slight increase in the reproduction number is observed, indicating that the positive jump effect dominates weak diffusion. However, as η_I increases further, R_{jp} decreases rapidly due to the quadratic growth of the diffusion term. In particular, at $\eta_I = 0.6$, $R_{jp} < 1$, indicating

disease extinction despite $R_0 > 1$. Increasing the intensity of diffusion suppresses disease transmission and can drive the system from persistence ($R_0 > 1$) to extinction ($R_{jp} < 1$). For higher values of η_I , the number of reproductions decreases significantly to less than unity, confirming that strong environmental fluctuations suppress disease transmission. This table shows the impact of varying the jump amplitude J_I while

Table 7: 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

keeping $\eta_I = 0.2$ and $\lambda_J = 0.1$ fixed. For negative values of J_I , the stochastic reproduction number is reduced below the deterministic value, and in the case $J_I = -0.5$, $R_{jp} < 1$, indicating possible disease eradication due to sudden improvements or effective interventions. As J_I increases toward positive values, R_{jp} increases steadily and exceeds R_0 , reflecting the effect of sudden outbreak events. Thus, negative jumps contribute to disease control, whereas positive jumps amplify transmission.

Table 8: 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

Table 8 investigates the influence of the jump rate λ_J with fixed $\eta_I = 0.2$ and $J_I = 0.3$. When $\lambda_J = 0$, the model is driven only by diffusion, leading to $R_{jp} < 1$, which implies the extinction of the disease. As λ_J increases, R_{jp} increases significantly, surpassing the deterministic threshold. This indicates that frequent stochastic shocks enhance disease transmission. For large values of λ_J , the reproduction number becomes substantially greater than unity, suggesting that repeated sudden outbreak the disease can sustain and intensify the epidemic. A key observation is that negative jump amplitudes and low jump rates support disease control, while positive amplitudes and high jump frequencies promote outbreak persistence. Importantly, the model reveals that strong

diffusion can offset the effect of positive jumps, leading to $R_{jp} < 1$, whereas high jump intensity and frequency can dominate diffusion and increase R_{jp} well above R_0 . Overall, the most favorable scenario for disease control occurs when diffusion effects are strong and jump effects are either weak or negative. Conversely, frequent and large positive jumps represent the worst-case scenario, leading to enhanced transmission and sustained outbreaks.

3.3 Numerical Simulation of the Model

To illustrate the dynamical behavior of the stochastic typhoid model (2.3), numerical simulations were carried out using a time discretization scheme. Since the system contains both Brownian motion and jump processes, the Euler–Maruyama method combined with a Poisson jump process was employed.

Let $t_n = n\Delta t$, where Δt denotes the 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. The jump process increment is represented by

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

where ΔN_n follows a Poisson distribution with intensity $\lambda_J \Delta t$.

The force of infection is given by

$$\lambda = \frac{\beta_1 I + \omega \beta_2 A}{N}, \quad (61)$$

where

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

represents the total human population.

Applying the Euler–Maruyama discretization to system (2.3) yields

$$S_{n+1} = S_n + [\Lambda - (\lambda_n + \mu + \nu)S_n + \alpha V_n + \phi R_n] \Delta t, \quad (62)$$

$$V_{n+1} = V_n + [\nu S_n - (\alpha + \mu)V_n] \Delta t, \quad (63)$$

$$E_{n+1} = E_n + [\lambda_n S_n - (\sigma + \mu)E_n] \Delta t, \quad (64)$$

$$I_{n+1} = I_n + [\tau \sigma E_n - (\gamma_1 + \delta + \mu_2 + \mu)I_n] \Delta t + \eta_I I_n \Delta W_n + J_I I_n \Delta N_n, \quad (65)$$

$$A_{n+1} = A_n + [(1 - \tau) \sigma E_n - (\gamma_2 + \mu)A_n] \Delta t, \quad (66)$$

$$T_{n+1} = T_n + [\delta I_n - (\gamma_3 + \mu)T_n] \Delta t + \eta_T T_n \Delta W_n + J_T T_n \Delta N_n, \quad (67)$$

$$R_{n+1} = R_n + [\gamma_1 I_n + \gamma_2 A_n + \gamma_3 T_n - (\mu + \phi)R_n] \Delta t + \eta_R R_n \Delta W_n + J_R R_n \Delta N_n. \quad (68)$$

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.

3.4 Deterministic Simulation

The deterministic model corresponds to the system of ordinary differential equations (ODEs) described in Eq. (2.3) 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.

3.5 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.

3.6 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.

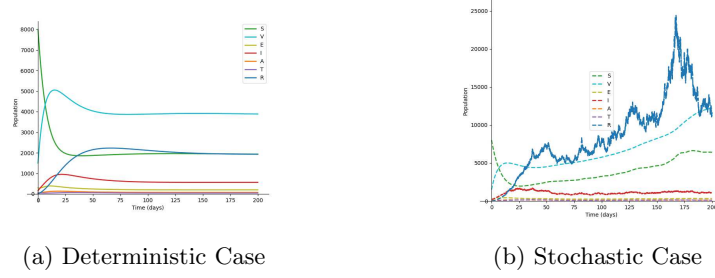


Fig. 5: Graph of deterministic and stochastic Typhoid models, respectively

Figure 5 presents the time evolution of the model compartments for both the deterministic and stochastic formulations. The deterministic simulation 5a 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 5b 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.

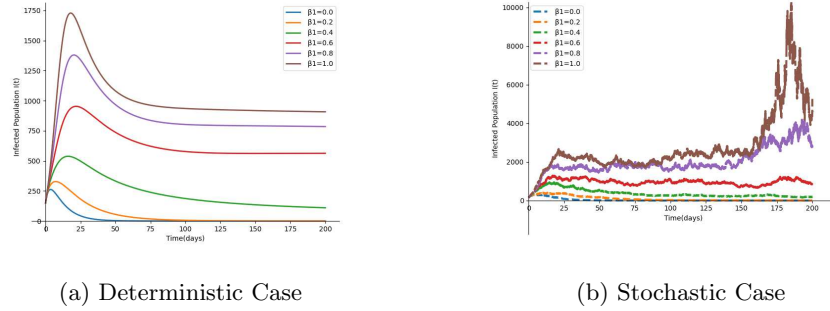


Fig. 6: Effects of symptomatic contact rate on infected population $I(t)$

Figure 6 presents the effect of the contact rate β_1 on the infected population. The deterministic simulation in Figure 6a 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 6b 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.

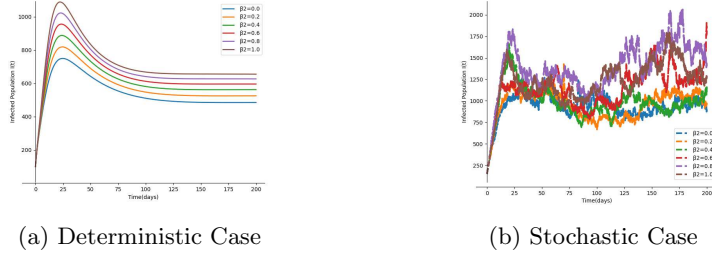


Fig. 7: Effects of asymptomatic contact rate on infected population $I(t)$

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.

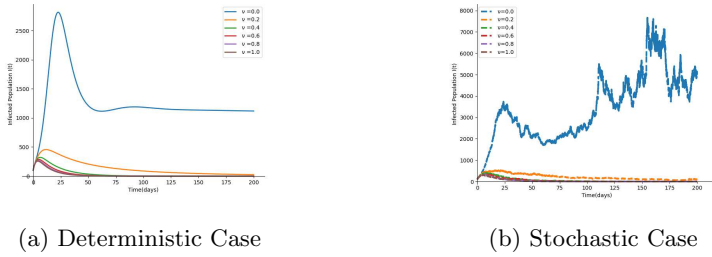


Fig. 8: Effects of vaccination rate on infected population $I(t)$

From Fig. 8a, 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. 8b 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.

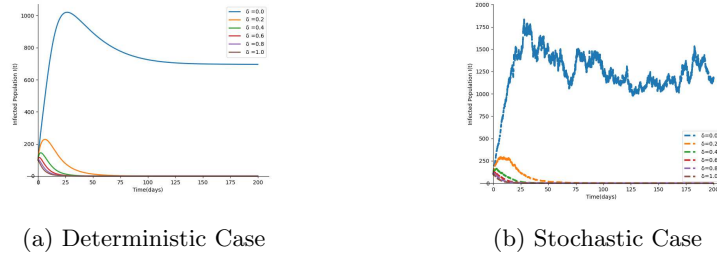


Fig. 9: Effects of treatment rate on infected population $I(t)$

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. 9 are obtained by varying the vaccination rate from $\delta = 0.0$ to $\delta = 1.0$, while keeping all other model parameters constant.

4 Discussion and Conclusion

Typhoid fever dynamics using a hybrid deterministic-stochastic epidemic modeling approach with jump-diffusion to capture continuous random fluctuations(diffusion)

as given in (2.3) for η_I , η_T and η_T which represent environmental and behavioral changes, as well as random contact variations and sudden outbreaks (jumps) for J_I , J_T and J_T which stand for sudden discrete events like outbreak spikes, policy changes (lock-downs, mass testing) and Healthcare system shocks is presented. The significance of the approach used in this work is that the models cater for unpredictable outbreaks and random shocks.

The interpretation of the parameter values of the deterministic model as obtained in Table 2 suggests a growing population either by means of birth or migration which obviously can retain the spread of typhoid fever disease within the population. Moreover, it was seen that 1% of the population dies naturally per unit time. The values of the transmission parameter (β_1, β_2) show a higher level of transmission from people with symptoms of Typhoid fever than from individuals without symptoms. However, the latter case is also found to be relatively high too. The rate at which the exposed individual became infectious was found to be relatively high, this depicts a clinically visible case of the presence of the disease in the population. However, it was discovered that when treatment was applied, the recovery rate was accelerated. In addition, the very slow rate of movement from infected to treated indicates limited health care facilities and other factors that could lead to this, resulting in high mortality per unit of time. The result obtained here also indicated that natural immunity lasts longer than vaccine immunity.

The values of the jump-diffusion parameters obtained in Table 2 above are categorized into two, namely: the diffusion intensities: η_I, η_T, η_T (representing the environmental-random fluctuations) and jump measures: J_I, J_T, J_R , (representing, abrupt or sudden occurrence). The estimated values show moderate variability in contact rates, treatment outcomes, and recovery patterns. In contrast, an interesting and opposite scenario was observed in the case of the jump parameters; wherein, a 30% sudden increase in the outbreak of the disease was found. This must have led to a rapid hospitalization intervention by the government and in-turn to mass recovery. From the findings, it was observed that the rates in the above described were the same. In the absence of random fluctuations and jumps in the system, the sensitivity results obtained show that improving clinical treatment will obviously lead to a reduced infection period, transmission, and the number of recorded deaths. Therefore, investment in hospitals, medications, and early detection of asymptomatic cases is recommended.

The results in Tables 6–8 demonstrate that the stochastic reproduction number R_{jp} is driven by the interaction between diffusion and jump processes. Diffusion, represented by η_I , consistently acts to reduce disease transmission and can drive the system toward extinction even when the deterministic model predicts persistence. In contrast, the jump process, characterized by the jump amplitude J_I and jump rate λ_J , introduces sudden changes that may suppress or amplify transmission.

In comparison of the deterministic dynamic and the jump-diffusion curves as seen in Figs 3-7 (a and b, respectively), the time evolution of the jump-diffusion model

shows that the recovered population has the highest randomness reflecting very large jumps in terms of number and size. This supports the fact that the recovery rate of symptomatic individuals is highly sensitive. The randomness observed in the infected and treated time evolution plots was found to be moderate. This indicates that sudden mini-outbreaks can occur that could be unpredictable, and as such, health care responses should not be a constant. In contrast, the susceptible and vaccinated population curves show minimal randomness and few jumps, indicating that the susceptible and vaccinated population are driven by gradual, planned, and predictable government policies and demography. Similarly, the exposed and asymptomatic curves show very low randomness and no jumps.

In conclusion, this research has contributed to the advancement of epidemiological modeling and improved public health preparedness in the face of sudden and random outbreak of typhoid fever by providing a more comprehensive tool to predict, control, and potentially eradicate typhoid fever.

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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