Journal of Biostatistics and Biometric Applications
Volume 9 | Issue 1
ISSN:2455-765X

Research Article Open Access

Modeling the Dynamics of Rubella Disease Using A Stochastic-Delay Dynamical System

Tariku Merga Tolasa[1], Manalebish Debalike Asfaw[2*], Getinet Alemayehu Wole[3] and Getachew Teshome Tilahun[4]

[1,3,4]Department of Mathematics, Haramaya University, Dire Dawa, Ethiopia

[2]Department of Mathematics, Addis Ababa University, Addis Ababa, 1176, Ethiopia

[*]**Corresponding Author:** Asfaw Manalebish Debalike, Department of Mathematics, Addis Ababa University, Addis Ababa, 1176, Ethiopia, Tel.: +251911531026, E-mail: manalebish.debalike@aau.edu.et

Citation: Tariku Merga Tolasa, Manalebish Debalike Asfaw, Getinet Alemayehu Wole, Getachew Teshome Tilahun (2024) J Biostat Biometric App 9(1): 101

Received Date: January 05, 2024 **Accepted Date:** February 05, 2024 **Published Date:** February 08, 2024

Abstract

Everyone in the world is susceptible to the serious and highly contagious rubella virus disease in humans. It is especially dangerous when pregnant women contract the virus and transmit it to the fetus, causing congenital rubella syndrome. In this study, a stochastic delay mathematical model of rubella dynamics was developed and analyzed to describe how the incubation period and environmental influences, such as temperature changes, affect the disease. To develop this model the entire population was divided into fivecategories: S(t) susceptible, V (t) vaccinated, P(t) protected (received the second dose of vaccine and developed active immunity for the rest of life), I(t) infected, and R(t) recovered. The qualitative behavior of the model is analyzed, and the model analysis results show that the basic reproductive number in the stochastic delay is smaller than the delay due to time delay and stochastic perturbation. Some parameter values were taken from relevant published articles and others were assumed. Numerical simulations were performed to study the effects of basic parameters, and the simulation results were displayed graphically using MATLAB 2019 computer software. Simulation results suggest that reducing contact, exposure, and vertical transmission rates as well as increasing the time delays, recovery rate and rate of first and second-dose vaccination play a role in minimizing the spread of rubella.

Keywords: Rubella, delay, stochastic, stochastic-delay modeling, numerical simulation.

Introduction

Rubella is a serious infectious disease in humans caused by the rubella virus [1]. It is caused by droplets released from the respiratory secretions of a person infected with the rubella virus, by contact with tissues infected with the rubella virus, by contact with the urine of a child infected with rubella, and by drinking fromcups used by a person infected with this virus. A study [2] showed that rubella is a global epidemic spreading in tropical and subtropical regions. It is commonly known as a non-fatal disease. However, it is very dangerous, especially for pregnant women infected with the rubella virus where their infants inherited congenital rubella syndrome (CRS), which puts them at higher risk for fetal development and has been linked to birth defects, still birth, intellectual disability, heart disease, diabetes, encephalitis, and low birth weight (14).

This disease caused health and economic problems worldwide. For instance, complications including 20,000 babies born with CRS, 2,000 cases of encephalitis, 11,250 abortions, and 2,100 infant deaths were reported in the United States between 1964 and 1965 [3]. According to [4], there were about 22,000 children born with CRS in Africa, about 46,000 in Southeast Asia, and about 13,000 in the Western Pacific in 1996. Besides this study, many studies try to explainthat very few countries have included rubella vaccination in their vaccination programs. Their national vaccination programs and the absence of childhood vaccination programs may contribute to the increased incidence of CRS. A study by [5] reported that between 2009 and 2015, there were 2,615 cases of rubella in Ethiopia most of them (52.2%) were female, and the age range of confirmed cases was between one month to forty-two years old. In their study, about threequarters of confirmed rubella cases were over 5 years old, and the number of laboratory-confirmed rubella cases increased linearly from 83 cases in 2009 to 856cases in 2013, but decreased to 222 and 319 cases in 2014 and 2015, respectively. The number of cases recorded is highest in the hot dry season from January to June and lowest in August and September [6]. This indicates that the dynamics of rubella are opportunistic. Some symptoms of this viral disease are mild fever, fatigue, headache, miscarriage, itching, irritability, joint pain, polyarthritis, and loss of appetite [7]. The pink or red rash appears 14 to 17 days after exposure with an average incubation period of 14 days, ranging from 12 to 23 days. It is a vaccine-preventable disease, and the MMR (measles, mumps, and rubella) vaccine is the most recommended vaccine to prevent all three diseases and is considered eradicated [8]. According to the Centers for Disease Control and Prevention, children should get two doses of the MMR vaccine. The first dose of the vaccine provides greater than 95% immunity and should be given to children between 12 and 15 months of age, while the second dose should be given to children between 4 and 6 years of age to confer lifelong immunity. However, there is no specific treatment for rubella infected person [8].

A mathematical model is useful for describing and analyzing dynamic behavior, spread, and control strategies to create a visualization of infection over time [9]. As noted in the study [10], the concept of disease transmission and control is based on mathematical rules used in the research design process to inform the design of infectious diseases. According to the research conducted by [11], developing mathematical models helps to understand epidemiological patterns and predict the consequences of introducing public health measures to control the spread of disease. The ability of a mathematical model to predict disease control strategies highly depends on the assumptions made during the modelingprocess. This is also explained in the study [12] as the idea of a mathematical model is a simplified representation of real-world entities, whether mathematical formulas or computer codes, which are models, developed over time and are systematic in nature. There are various types of mathematical modeling methods. One type of mathematical model is the stochastic model, which takes into account environmental factors such as precipitation, humidity, and temperature, as well as the randomness of infectious agents [13]. A study by [14] found that contacts occurring during the incubation and infectious periods have a significant impact on the level of transmission of all types of infectious diseases. So, we have to include randomness in mathematical models to account for the consequences of environmental variation on the model and help to compare the difference between stochastic and deterministic in mathematical epidemiology. When an intrinsically vulnerable person comes into contact with an infected person, the vulnerability is released and becomes infected but not contagious and the infected person is exposed for several days before being infectious [11]. Therefore, it is prudent to provide a time interval from infection to onset of symptoms using a delay mathematical model. Again by incorporating both the incubation period and environmental factors, we can develop a stochastic delay mathematical model that approximates stochastic differential equations involving auto-correlated and

generalized deterministic, delay, and stochastic differential equations [15]. To study the spread of rubella in the community and predict its control mechanisms, many epidemiological modeling studies have been performed using various mathematical modeling techniques. For instance, the dynamics of rubella re-infection with vaccine-induced transmission have been studied in [2]. Based on the biological behavior of rubella, a deterministic mathematical model was built by dividing the total population into 5 groups: type S(t) susceptible, E(t) exposed, I(t) infected, R(t) recovered and V (t) vaccinated. However, the assumption that the first vaccine

develops lifelong active immunity is inconsistent with a study by [16], in which the MMR vaccine was recommended twice for the development of active immunity (protects against rubella virus). A SEIR mathematical model of rubella outbreaks with optimized ordering using fractional differential equations was also developed in [17]. MATLAB functions are used to obtain optimal values for parameters such as transmission rate, fractional order, and fractal dimension using nonlinear least squares estimation techniques. The results show that this is directly related to the cause of the violation transmission. The SEIRV mathematical model of rubella dynamics was developed and analyzed in [18], with the aim of extending the model to support studies of virus spread in target populations. According to [19], rubella can be detected in the blood even several days after infection and then spreads throughout the body. It is teratogenic and has the ability to cross the placenta infect the fetus, and destroying cells. The author developed the SEIVR mathematical model based on non-singular, non-local fractional derivatives and used three-level Bashforth diagram and Jacobian matrix to research and test the stability of the model's equilibrium point. The existence and uniqueness of the solution are analyzed in detail using the Banach fixed point theorem. This work was supported by [20] by replacing the time derivative with Caputo- Fabrizio partial differential equation model of rubella prevalence. Based on the assessment of national rubella vaccination programs and the CRS, the study in [21] an MSVEIR mathematical model developed. This model predicts rubella virus eradication as part of a vaccination program. Transmission of infection and reduced vaccination effectiveness is a major problem, as vaccinated individuals can be asymptomatic and re-infected. Research by [22] revealed that non-linear rubella epidemic progression interacts in complex ways with both fertility and spatial heterogeneity, leading to increased vaccination rates across demographic and epidemiological needs. This study also explains that rubella infection in children inherited from their mother during early pregnancy can lead to fetal death and multiple CRS due to insufficient MMR coverage. Birth rates, infection rates, and seasonality influence the minimum coverage required for rubella vaccination. The burden of CRS in epidemiological and demographic settings, including the effects of local infection with stochastic decline, was modeled by dividing the total population into five epidemiological groups, namely: maternal immunity M(t), susceptibility S(t), infection I(t), recovery R(t) and vaccination V (t). According to a study by [23], regarding the biological aspects of vaccination, the rubella vaccination strategy aims to eliminate rubella and reduce CRS. As a result, a SEIVR deterministic mathematical model was developed to describe the direct or indirect transmission of rubella.

Many authors have studied the public health complications and transmission of rubella. As a result, many different mathematical models have been developed to predict the mechanism of rubella control. As demonstrated in [6] and [22], the unpredictability of interpersonal contact and environmental factors duringincubation and infection leads to dynamic transmission of the rubella disease is random in nature. As the authors stated, rubella is an opportunistic viral disease influenced by environmental factors such as temperature changes and seasonal factors. Therefore, models must account for randomness using a stochastic approach. As a study conducted by [7], rubella also shows symptoms several days after infection. To the best of our knowledge, there is no study that accounts for randomness and delay simultaneously in their model. Therefore, in this study, we developed a rubella dynamics model using a stochastic delay dynamics system, which takes into account incubation period (time delay), second vaccination, stochastic factors, and vertical transmission from mother to fetus. The remainder of this paper is arranged as follows: Section 2 presents the description and formulation of the model. Section 3 deals with the qualitative analysis of the model. Section 4 deals with the numerical simulations of the model and the simulation of the effects of basic parameters where the results of this simulation are displayed using MATLAB 2019 computer software are discussed. Finally, Section 5 provides a discussion and conclusion for this paper.

Model Description and Formulation

The model divides the total population into six parts, namely: S(t) susceptible, V (t) vaccinated, P(t) permanently immunized, E(t) exposed, I(t) infected, and R(t) recovered). Individuals in the susceptible group had increased recruitment rates π, first vaccinations influx ω, and loss of immunity in convalescents with rates ρ as well as decreased contact rates β and vaccination rates α. Individuals in

the infected group will increase at rate β through contact and vertical transmission from mother to child at rate θ, as well as decrease at rate γ, and die from rubella at rate ε. The recovery class increases with the rate of recovery rate γ of the infected person and decreases with the rate of loss of immunity ρ. The group of vaccinated individuals will increase as the first dose of vaccine is administered to those susceptible at a rate of α. This class is reduced by administering the second dose of vaccine at a rate δ and tapering the first dose of vaccine at a rate ω. The permanent immunized class will be increased by administering the second dose of the vaccine at a rate δ. All classes decreased due to the natural mortality rate μ and all parameters were positive. The main goal of this model is to examine the impact of stochastic perturbations and the incubation period (time delay) on rubella dynamics. Furthermore, the model is developed with the following assumptions:

- Individuals will become infected through direct contact with respiratory droplets from rubella infected person and vertical transmission from mother to fetus [1].
- Rubella infected individuals will recover or die due to rubella disease [2].
- People who have recovered from rubella disease and have been vaccinated for the first time are again susceptible to rubella after several years [2].
- A person who received two doses of MMR vaccine will have active immunity (get permanent immunity against rubella virus) [16].
- All the parameters to be used in this model are positive and due to natural death at a rate of μ, all classes are decreased.
- We also assumed that rubella is an opportunistic viral disease (i.e. it is influenced by environmental factors [6, 22].
- Based on the behavior of rubella, we assume that an individual infected during time $t - \tau$, but not yet contagious (exposed), will become infected after time τ . The probability that an individual survives the incubation period $[t - \tau, t]$ is determined by the bilinear incidence $\beta S(t - \tau)I(t - \tau)e^{-\mu\tau}$, where is the incubation period [16].

Figure 1 shows the Rubella dynamics diagram constructed based on the model description, assumptions, and interrelationships between compartments and parameters.

After introducing Brownian motion Bi(t) and environmental factors σi, we obtained model equation (1).

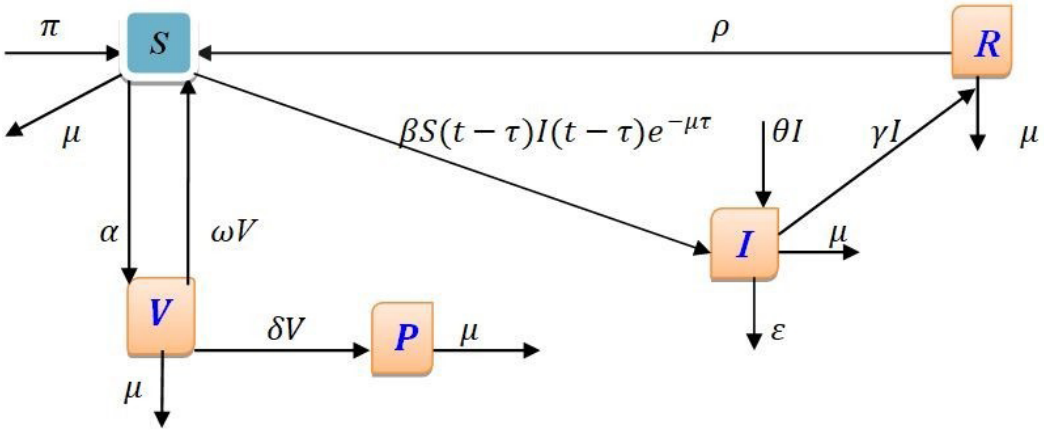

Figure 1: Schematic diagram of rubella disease dynamics.

$$\begin{cases} dS = (\pi + \rho R + \omega V - \beta S(t-\tau)I(t-\tau)e^{-\mu\tau} - (\alpha+\mu)S)\,dt + \sigma_1 S dB_1(t) \\ dV = (\alpha S - (\delta+\omega+\mu)V)\,dt + \sigma_2 V dB_2(t) \\ dP = (\delta V - \mu P)\,dt + \sigma_3 P dB_3(t) \\ dI = (\beta S(t-\tau)I(t-\tau)e^{-\mu\tau} - (\mu+\gamma+\varepsilon-\theta)I)\,dt + \sigma_4 I dB_4(t) \\ dR = (\gamma I - (\mu+\rho)R)\,dt + \sigma_5 R dB_5(t) \end{cases} \tag{1}$$

Qualitative Analysis of the Model

In this section, the qualitative behaviour of the model such as the positivity, boundedness, equilibrium points, basic reproductive number, and sensitivity analysis of the model are analysed.

3.1 Positivity of the Solutions

For biological reasons, since the state variables and corresponding parameters represent population types and proportions, they are assumed to be positive [23].

Theorem 3.1. All solutions of system of equations (1) are non-negative for all values of t > 0, with initial conditions S0 > 0, V0 ≥ 0, P0 ≥ 0, I0 ≥ 0,and R0 ≥ 0.

Proof. To proof Theorem (3.1) we take the first equation of the system ofequation (??),

$$\frac{dS}{dt} = \pi + \rho R(t) + \omega V(t) - \beta S(t-\tau)I(t-\tau)e^{-\mu\tau} - (\alpha+\mu)S(t) \tag{2}$$

considering only the negative part of (2), we get

$$\frac{dS}{dt} \geq -\beta S(t-\tau)I(t-\tau)e^{-\mu\tau} - (\alpha+\mu)S \tag{3}$$

Using a study by [13],S(t − τ) and I(t − τ) represent the individuals who have released the susceptibility and are infected but not infectious. Then, the delay differential equations can then be simplified as limt−→∞ S(t) = limt−→∞ S(t−τ) = S and limt−→∞ I(t) = limt−→∞ I(t − τ) = I . We now apply the method of separation of variables to equation (3), we get

$$\begin{aligned} \frac{dS}{S} &\geq -\left(\beta I e^{-\mu\tau} + \alpha + \mu\right) dt \\ \int \frac{dS}{S} &\geq -\int \left(\beta I e^{-\mu\tau} + \alpha + \mu\right) dt \\ \Rightarrow S(t) &\geq S_0 \exp(-\int \left(\beta I e^{-\mu\tau} + \alpha + \mu\right) dt) > 0 \end{aligned}$$

Next, let's take the second equation of the system of equation (??).

$$\begin{aligned}\frac{dV}{dt} &= \alpha S(t) - (\delta + \omega + \mu) V(t) \\ &\geq -(\delta + \omega + \mu) V(t) \\ \Rightarrow V(t) &\geq V(0) \exp(-(\delta + \omega + \mu) dt)\end{aligned}$$

In the same process, we get the solutions for the other compartment such as P(t) ≥ P0 exp(−μt) > 0, I(t) ≥ I0 exp(−(μ + γ + ε) t) > 0 and R(t) ≥ R0 exp(−(μ + ρ) t + c) > 0. Here since the exponential function is positive, then S(t) > 0 ,V (t) > 0 ,P(t) > 0 ,I(t) > 0 , and R(t) > 0. In other words, the solution of the system of equations is non-negative for non-negative initial conditions.

3.2 Boundedness of the Solution for the Delay Model

This section discusses the region in which solving the system of equations is biologically significant and meaningful [23]. Therefore, in this section, we consider the region where the solutions of the system of equations (1) are bounded.

Theorem 3.2. The solution of the system of equation (1) with initial conditions S0 > 0, V0 ≥ 0, P0 ≥ 0, I0 ≥ 0,R0 ≥ 0 is bounded in the region Ω = (S, V, P, I,R) ∈ R5+ : 0 ≤ N(t) ≤ π μ .

Proof. Let's look at the total population at a given point in time, like this:

$$N(t) = S(t) + V(t) + P(t) + I(t) + R(t). \tag{4}$$

Next let's differentiate both sides of equation (4) concerning time t ;

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dP}{dt} + \frac{dI}{dt} + \frac{dR}{dt},$$

$$\begin{aligned}&= \pi + \rho R + \omega V - \beta S(t-\tau) I(t-\tau) e^{-\mu\tau} - (\alpha + \mu) S + \theta I + \\ &\quad \beta S(t-\tau) I(t-\tau) e^{-\mu\tau} - (\mu + \gamma + \varepsilon - \theta) I + \alpha S - (\delta + \omega + \mu) V + \delta V - \mu P + \gamma I - (\mu + \rho) R \\ &= \pi + \theta I - \varepsilon I - \mu(S + P + V + I + R), \\ &= \pi + \theta I - \varepsilon I - \mu N. \end{aligned} \tag{5}$$

If there are no deaths due to rubella and no rubella-infected newborns, then

$$\begin{aligned}\frac{dN}{dt} &\leq \pi - \mu N \\ N &\leq \frac{\pi - exp(-\mu t + c)}{\mu} \\ &\leq \frac{\pi}{\mu} \frac{-exp(-\mu t + c)}{\mu}\end{aligned}$$

As t −→ ∞ the value of exp(−μt + c) approaches to zero. The non-negative solution of this model is proved in Theorem (3.1) , so N(t) = π/μ for all t ≥ 0.Since all state variables represent a population class, they remain non-negative over the possible region Ω

[11] Therefore, the developed mathematical model is well posed (the behavior of the solution changes continuously according to the initial conditions) and epidemiologically meaningful in the region $\Omega = (S, V, P, I, R) \in R^5_+ : 0 \leq N(t) \leq \pi/\mu$

3.3 Boundedness of the Model for Stochastic Delay Model

Theorem 3.3. The region Ω is almost surely a positively invariant of the system of stochastic equations (1).

Proof. Let's take a large integer K ,so that if (S0, V0, P0, I0,R0) $\in R^5_+$, then every component S(t), V (t), P(t), I(t) and R(t) lies in the interval $[1/k_0, 1]$. For each integer $K \geq k_0$, a stopping time can defined as:

$$\tau_m = \inf\left\{t \in [0, \tau_e] : S(t) \leq \frac{1}{K} \text{ or } I(t) \leq \frac{1}{K} \text{ or } R(t) \leq \frac{1}{K}\right\} \text{ and}$$

$$\tau_\infty = \inf\{t \in [0, \tau_e] : S(t) \leq 0 \text{ or } I(t) \leq 0 \text{ or } R(t) \leq 0\}$$

Next, we want to show $P(\tau = \infty)$, which is $P(\tau < K) = 0$ for $K > 0$ so that it allows us to show $\lim t \to \infty(\sup P(\tau k < 0) = 0)$. Let's consider the Lyapunov function L(t) as follows:

$$V(t) = -\ln S(t) - \ln V(t) - \ln P(t) - \ln I(t) - \ln R(t). \tag{6}$$

Differentiating both sides of (6) gives

$$\begin{aligned} dV(t) &= -\left[\frac{1}{S(t)}dS(t) + \frac{1}{V(t)}dV(t) + \frac{1}{P(t)}dP(t) + \frac{1}{I(t)}dI(t) + \frac{1}{R(t)}dR(t)\right] \\ &= -\frac{1}{S(t)}\left[(\pi + \rho R(t) + \omega V(t) - \beta S(t-\tau)I(t-\tau)e^{-\mu\tau} - (\alpha+\mu)S(t))dt + \sigma_1 S(t)dB_1(t)\right] \end{aligned}$$

$$\begin{aligned} &-\frac{1}{V(t)}\left[(\alpha S(t) - (\delta + \omega + \mu)V(t))dt + \sigma_2 V(t)dB_2(t)\right] \\ &-\frac{1}{P(t)}\left[(\delta V(t) - \mu P(t))\,dt + \sigma_3 P(t)dB_3(t)\right] \\ &-\frac{1}{I(t)}\left[\left(\beta S(t-\tau)I(t-\tau)e^{-\mu\tau} - (\mu + \gamma + \varepsilon - \theta)I(t)\right)dt + \sigma_4 I(t)dB_4(t)\right] \\ &-\frac{1}{R(t)}\left[(\gamma I(t) - (\mu+\rho)R(t))\,dt + \sigma_5 R(t)dB_5(t)\right] \\ &-\frac{1}{(S(t))^2}\left[(\pi + \rho R(t) + \omega V(t) - (\beta I(t-\tau)S(t-\tau)e^{-\mu\tau} + (\alpha+\mu)S(t))dt + \sigma_1 S(t)dB_1(t)\right]^2 \\ &-\frac{1}{(I(t))^2}\left[\left(\beta I(t-\tau)S(t-\tau)e^{-\mu\tau} + \theta I(t) - (\mu+\gamma+\varepsilon)I(t)\right)dt + \sigma_4 I(t)dB_4(t)\right]^2 \end{aligned} \tag{7}$$

Let $a = \pi + \rho R(t) + \omega V(t) - \beta S(t)I(t)e^{-\mu\tau} + (\alpha + \mu)S(t)$, $b = \sigma_1 S(t)$, $c = \beta S(t)I(t)^{e-\mu\tau} + \theta I(t) - (\mu + \gamma + \varepsilon)I(t)$ and $d = \sigma_4 I(t)$.
where $S(t - \tau) = S(t) = S$ and $I(t - \tau) = I(t) = I$ Then equation (7) becomes

$$
\begin{aligned}
dV(t) &= -\frac{1}{S(t)}\left[(\pi+\rho R(t)+\omega V(t)-\beta S(t)I(t)e^{-\mu\tau}-(\alpha+\mu)S(t))dt+\sigma_1 S(t)dB_1(t)\right]\\
&-\frac{1}{V(t)}\left[(\alpha S(t)-(\delta+\omega+\mu)V(t))dt+\sigma_2 V(t)dB_2(t)\right]\\
&-\frac{1}{P(t)}\left[(\delta V(t)-\mu P(t))\,dt+\sigma_3 P(t)dB_3(t)\right]\\
&-\frac{1}{I(t)}\left[\left(\beta S(t)I(t)e^{-\mu\tau}-(\mu+\gamma+\varepsilon-\theta)I(t)\right)dt+\sigma_4 I(t)dB_4(t)\right]\\
&-\frac{1}{R(t)}\left[(\gamma I(t)-(\mu+\rho)R(t))\,dt+\sigma_5 R(t)dB_5(t)\right]\\
&-\frac{1}{S(t)}\left[adt+bdB_1(t)\right]^2\\
&-\frac{1}{I(t)}\left[cdt+ddB_4(t)\right]^2\\
&=\left[-\frac{\pi}{S(t)}-\frac{\rho R(t)}{S(t)}-\frac{\omega V(t)}{S(t)}-\frac{\alpha S(t)}{V(t)}-\frac{\delta V(t)}{P(t)}-\frac{\gamma I(t)}{R(t)}-\beta S(t)e^{-\mu\tau}-\theta-\beta I(t)e^{-\mu\tau}+\alpha\right.\\
&+5\mu+\omega+\delta+\gamma+\varepsilon+\rho-[B_1^2+B_2^2+B_3^2+B_4^2+B_5^2]dt-\sigma_1 dB_1(t)-\sigma_2 dB_2(t)\\
&-\sigma_3 dB_3(t)-\sigma_4 dB_4(t)-\sigma_5 dB_5(t) \qquad (8)
\end{aligned}
$$

Let us represent $LV=\left[-\frac{\pi}{S(t)}-\frac{\rho R(t)}{S(t)}-\frac{\omega V(t)}{S(t)}-\frac{\alpha S(t)}{V(t)}-\frac{\delta V(t)}{P(t)}-\frac{\gamma I(t)}{R(t)}-\beta S(t)e^{-\mu\tau}-\right.$
$\theta-\beta I(t)e^{-\mu\tau}+\alpha+5\mu+\omega+\delta+\gamma+\varepsilon+\rho-[B_1^2+B_2^2+B_3^2+B_4^2+B_5^2]$ and

$D=\alpha+5\mu+\omega+\delta+\gamma+\varepsilon+\rho-B^2{}_1+B^2{}_2+B^2{}_3+B^2{}_4+B^2{}_5]$.Then equation (8) becomes

$$
\begin{aligned}
dV(t) &= LV(t)dt+Ddt-[\sigma_1 dB_1(t)+\sigma_2 dB_2(t)+\sigma_3 dB_3(t)+\sigma_4 dB_4(t)+\sigma_5 dB_5(t)\\
&\le Ddt-[\sigma_1 dB_1(t)+\sigma_2 dB_2(t)+\sigma_3 dB_3(t)+\sigma_4 dB_4(t)+\sigma_5 dB_5(t)] \qquad (9)
\end{aligned}
$$

Let $\tau k \wedge M = \min(\tau n, t)$. Then by integrating both sides (9) with respect to time t runs from 0 to $\tau k \wedge M$ gives

$$
\begin{aligned}
\int_0^{\tau_k\wedge M} dV(X(t)) &= \int_0^{\tau_k\wedge M} Ddt-\int_0^{\tau_k\wedge M}\sigma_1 dB_1(t)-\int_0^{\tau_k\wedge M}\sigma_2 dB_2(t)-\int_0^{\tau_k\wedge M}\sigma_3 dB_3(t)\\
&-\int_0^{\tau_k\wedge M}\sigma_4 dB_4(t)-\int_0^{\tau_k\wedge M}\sigma_5 dB_5(t)\\
V(X(\tau_k\wedge M)) &= V(X(0))+D\int_0^{\tau_k\wedge M}dt-\sigma_1\int_0^{\tau_k\wedge M}dB_1(t)-\sigma_2\int_0^{\tau_k\wedge M}dB_2(t)\\
&-\sigma_3\int_0^{\tau_k\wedge M}dB_3(t)-\sigma_4\int_0^{\tau_k\wedge M}dB_4(t)-\sigma_5\int_0^{\tau_k\wedge M}dB_5(t)\\
\Rightarrow EV(X(\tau_k\wedge M)) &\le V(X(0))+DE\int_0^{\tau_k\wedge M}dt\le V(X(0))+DE \qquad (10)
\end{aligned}
$$

Since V (X($\tau k \wedge M$)) > 0, then

$$EV(X(\tau_k \wedge M)) = E[V(X(\tau_k \wedge M))_{X(\tau_k \leq M)}] + E[V(X(\tau_k \wedge M))_{X(\tau_k > T)}] \geq E[V(X(\tau_k \wedge M))_{X(\tau_k \leq M)}] \quad (11)$$

Now, for τ_k, there are some components of X(τk), say S(τk) such that 0 < S(τk) ≤ 1/k < 1. Therefore, X(τk ∧M) ≥ –ln(1/k) this comes from,

$$X(\tau_k \wedge M) = \ln S(\tau_k) \leq \ln(\frac{1}{k}),$$
$$X(\tau_k \wedge M) \geq \ln(\frac{1}{k}) \text{ which implies } X(\tau_k \wedge M) \geq -\ln(\frac{1}{k}) \quad (12)$$

Thus, from the equations (11) and (12), we get

$$EV(X(\tau_k \wedge M)) \geq E[V(X(\tau_k \wedge M))_{X(\tau_k \leq M)}] \geq E[-\ln(\frac{1}{k})] \quad (13)$$

From the equations (12) and (13) it follows that

$$\begin{aligned} EV(X(\tau_k \wedge M)) &\geq -\ln(\frac{1}{k})P(\tau_k \wedge M), \\ &\geq \ln(k)P(\tau_k < M), \\ P(\tau_k < M) &\leq \frac{EV(X(\tau_k \wedge M))}{\ln(k)} \leq \frac{V(X(0)) + CM}{\ln(k)} \\ &\leq \frac{V(X(0)) + CM}{\ln(k)} \end{aligned} \quad (14)$$

Let $\tau_k \to \infty$ and by taking limit sup to equation (14),for all M > 0, we obtain that $P(\tau_k < M) \leq 0$. Therefore, $\lim_{t\to\infty} \sup(P(\tau_k < M)) = 0$ Then the theorem is proved.

3.4 Equilibrium Points of the Model

In this section, we calculated the points at which the disease disappears and persists for a model consisting of all possible non-negative solutions of the system equations.

3.4.1 The Disease-Free Equilibrium Point of the Model

In this section, the point of disappearance of rubella from the community is calculated using the system of equations (1). To do this, the right side of the equation is set to zero.

$$\Rightarrow \begin{cases} \pi + \rho R(t) + \omega V(t) - \beta S(t-\tau)I(t-\tau)e^{-\mu\tau} - (\alpha+\mu)S(t) = 0 \\ \alpha S(t) - (\delta + \omega + \mu)V(t) = 0 \\ \delta V(t) - \mu P(t) = 0 \\ \beta S(t-\tau)I(t-\tau)e^{-\mu\tau} - (\mu + \gamma + \varepsilon - \theta)I(t) = 0 \\ \gamma I(t) - (\mu + \rho)R(t) = 0 \end{cases} \quad (15)$$

The disease-free equilibrium point is reached when the proportion of infected people is zero (i.e. when there are no infected people in the community). The system of equations (15) is then presented in the following simplified form:

$$\Rightarrow \begin{cases} \pi + \omega V(t) - (\alpha + \mu)S(t) = 0 \\ \alpha S(t) - (\delta + \omega + \mu)V(t) = 0 \\ \delta V(t) - \mu P(t) = 0 \end{cases} \tag{16}$$

From second and third equation of (16), we get $V(t) = \frac{\mu P(t)}{\delta}$ and $S(t) = \frac{(\delta + \mu + \omega)V(t)}{\alpha} = \frac{(\delta + \mu + \omega)\mu P(t)}{\alpha\delta}$. Then, substituting $S(t)$ and $V(t)$ in the first equation of the system (16), we get $P(t) = \frac{\pi\alpha\delta}{((\alpha + \mu)(\delta + \mu) + \omega\mu)\mu}, S(t) = \frac{\pi(\delta + \mu + \omega)}{(\alpha + \mu)(\delta + \mu) + \omega\mu}$ and $V(t) = \frac{\pi\alpha}{(\alpha + \mu)(\delta + \mu) + \omega\mu}$

Therefore, the disease-free equilibrium point of the system of equation (1) is

$$(S(t), V(t), P(t), I(t), R(t)) = \left(\frac{\pi(\delta + \omega + \mu)}{(\alpha + \mu)(\delta + \mu) + \omega\mu}, \frac{\pi\alpha}{(\alpha + \mu)(\delta + \mu) + \omega\mu}, \frac{\pi\alpha\delta}{((\alpha + \mu)(\delta + \mu) + \omega\mu)\mu}, 0, 0\right)$$

3.4.2 The Endemic Equilibrium Point of the Model

To simplify the steady state of these delayed differential equations, we use $\lim_{t\to\infty} S(t) = \lim_{t\to\infty} S(t - \tau) = S$ and $\lim_{t\to\infty} I(t) = \lim_{t\to\infty} I(t - \tau) = I$.Therefore, from the third, second, fourth, and fifth equation of (15), we get,

$$V(t) = \frac{\mu P(t)}{\delta} \tag{17}$$

$$S(t) = \frac{(\delta + \omega + \mu)V(t)}{\alpha} = \frac{(\delta + \omega + \mu)\mu P(t)}{\alpha\delta} \tag{18}$$

$$R(t) = \frac{\gamma I(t)}{\mu + \rho} \tag{19}$$

$$I(t)(\beta S e^{-\mu\tau} + \theta - (\mu + \gamma + \varepsilon)) = 0 \tag{20}$$

Since $I(t) \neq 0$ at the endemic equilibrium point, then equation (20) becomes

$$\beta S e^{-\mu\tau} + \theta - (\mu + \gamma + \varepsilon) = 0$$

$$\Rightarrow S = \frac{(\mu + \gamma + \varepsilon - \theta)}{\beta e^{-\mu\tau}}$$

Using this value we can deduce the following from equations (18), as follows $S = \frac{(\delta+\omega+\mu)\mu P(t)}{\alpha\delta}$ which implies that $\frac{(\mu+\gamma+\varepsilon-\theta)}{\beta e^{-\mu\tau}} = \frac{(\delta+\omega+\mu)\mu P(t)}{\alpha\delta}$. This is true when $P = \frac{\alpha\delta(\mu+\gamma+\varepsilon-\theta)}{(\delta+\omega+\mu)\mu\beta e^{-\mu\tau}}$ Then, putting P in (17) , gives $V = \frac{\alpha(\mu+\gamma+\varepsilon-\theta)}{(\delta+\omega+\mu)\beta e^{-\mu\tau}}$. Next, let's substitute S, P and V in the first equation of (??)

$$\Rightarrow \pi + \rho R(t) + \omega V(t) - \beta S(t-\tau)I(t-\tau)e^{-\mu\tau} - (\alpha+\mu)S(t) = 0$$

$$\pi + \frac{\rho\gamma I}{\mu+\rho} + \frac{\omega\alpha(\mu+\gamma+\varepsilon-\theta)}{(\delta+\omega+\mu)\beta e^{-\mu\tau}} - \frac{\beta e^{-\mu\tau}(\mu+\gamma+\varepsilon-\theta)}{\beta e^{-\mu\tau}} I - \frac{(\alpha+\mu)(\mu+\gamma+\varepsilon-\theta)}{\beta e^{-\mu\tau}} = 0$$

$$I = \frac{(\mu+\rho)[\pi\beta e^{-\mu\tau}(\delta+\omega+\mu) - (\mu+\gamma+\varepsilon-\theta)((\alpha+\mu)(\delta+\mu)+\omega\mu)]}{\beta e^{-\mu\tau}(\delta+\omega+\mu)((\mu+\gamma+\varepsilon-\theta)(\mu+\rho)-\rho\gamma)}$$

Again by inserting the value of I in (19), gives

$$R = \frac{\gamma I}{(\mu+\rho)} = \gamma\left[\frac{[\pi\beta e^{-\mu\tau}(\delta+\omega+\mu) - (\mu+\gamma+\varepsilon-\theta)((\alpha+\mu)(\delta+\mu)+\omega\mu)]}{\beta e^{-\mu\tau}(\delta+\omega+\mu)((\mu+\gamma+\varepsilon-\theta)(\mu+\rho)-\rho\gamma)}\right]$$

Therefore, the endemic equilibrium point of the model is $(S, V, P, I, R) = \left\{\frac{(\mu+\gamma+\varepsilon-\theta)}{\beta e^{-\mu\tau}}, \frac{\alpha(\mu+\gamma+\varepsilon-\theta)}{(\delta+\omega+\mu)\beta e^{-\mu\tau}}, \frac{\alpha\delta(\mu+\gamma+\varepsilon-\theta)}{(\delta+\omega+\mu)\mu\beta e^{-\mu\tau}}, \frac{(\mu+\rho)[\pi\beta e^{-\mu\tau}(\delta+\omega+\mu)-(\mu+\gamma+\varepsilon-\theta)((\alpha+\mu)(\delta+\mu)+\omega\mu)]}{\beta e^{-\mu\tau}(\delta+\omega+\mu)((\mu+\gamma+\varepsilon-\theta)(\mu+\rho)-\rho\gamma)}, \gamma\left[\frac{[\pi\beta e^{-\mu\tau}(\delta+\omega+\mu)-(\mu+\gamma+\varepsilon-\theta)((\alpha+\mu)(\delta+\mu)+\omega\mu)]}{\beta e^{-\mu\tau}(\delta+\omega+\mu)((\mu+\gamma+\varepsilon-\theta)(\mu+\rho)-\rho\gamma)}\right]\right\}$ is the endemic equilibrium point of the model.

3.5 The reproductive Number of the Model

The next-generation matrix method described in many literatures mentioned is used to determine the base reproductive number of the model.

3.5.1 The Reproductive Number of the Model (R^D_0) with Time Delay.

To find the basic reproductive number, let's take the infected class.

$$\frac{dI}{dt} = \theta I(t) + \beta S(t-\tau)I(t-\tau)e^{-\mu\tau} - (\mu+\gamma+\varepsilon)I \qquad (21)$$

Then, using equation (21), we get

$$\begin{cases} f = \theta I(t) + \beta S(t-\tau)I(t-\tau)e^{-\mu\tau} \\ v = (\mu+\gamma+\varepsilon)I(t) \end{cases} \qquad (22)$$

where f denotes the rate of introduction of new infectious agents and v denotes transmission after new infection and individual transfer out of the compartment. So, the Jacobian matrix F of f and V of v with respect to is obtained as follows:

$$F = \left[\frac{\partial f}{\partial I}\right] = \left[\frac{\partial}{\partial I}\left(\frac{\partial(\theta I + \beta S I e^{-\mu\tau})}{\partial I}\right)\right] = \left[\theta + \beta S e^{-\mu\tau}\right] \quad (23)$$

$$V = \left[\frac{\partial v}{\partial I}\right] = \left[\frac{\partial}{\partial I}\left(\frac{\partial(\mu+\gamma+\varepsilon)I(t)}{\partial I}\right)\right] = [\mu+\gamma+\varepsilon] \quad (24)$$

where S(t−τ) = S(t) = S and I(t−τ) = I(t) = I. We obtain, at the disease-free equilibrium point, The Jacobian matrice of F, V, V^{-1} and the product of F and V^{-1} at disease-free equilibrium point are:

$$F_{DFE} = [\theta+\beta S e^{-\mu\tau}] = \left[\theta + \frac{(\delta+\mu+\omega)\pi\beta e^{-\mu\tau}}{(\alpha+\mu)(\delta+\mu)+\omega\mu}\right], V_{DFE} = [\mu+\gamma+\varepsilon], V^{-1} = \frac{1}{(\mu+\gamma+\varepsilon)},$$

$$FV^{-1} = \left[\theta + \frac{(\delta+\mu+\omega)\pi\beta e^{-\mu\tau}}{(\alpha+\mu)(\delta+\mu)+\omega\mu}\right] \times \frac{1}{(\mu+\gamma+\varepsilon)}$$

$$FV^{-1} = \left[\frac{\theta}{(\mu+\gamma+\varepsilon)} + \frac{(\delta+\mu+\omega)\pi\beta e^{-\mu\tau}}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)}\right]$$

Let?s now calculate the characteristic equation of FV^{-1} which is given by $\rho(\lambda) = |FV^{-1} - \lambda|$, where I is the identity matrix.

$$\rho(\lambda) = \left|\frac{\theta}{(\mu+\gamma+\varepsilon)} + \frac{(\delta+\mu+\omega)\pi\beta e^{-\mu\tau}}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)} - \lambda\right|$$
$$= \frac{\theta}{(\mu+\gamma+\varepsilon)} + \frac{(\delta+\mu+\omega)\pi\beta e^{-\mu\tau}}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)} - \lambda \quad (25)$$

The solution of characteristic equation (25) is obtained as follows:

$$\rho(\lambda) = \left|\frac{\theta}{(\mu+\gamma+\varepsilon)} + \frac{(\delta+\mu+\omega)\pi\beta e^{-\mu\tau}}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)} - \lambda\right|$$
$$= \frac{\theta}{(\mu+\gamma+\varepsilon)} + \frac{(\delta+\mu+\omega)\pi\beta e^{-\mu\tau}}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)} - \lambda$$

According to the principle of the next-generation matrix method the largest eigenvalue of the Jacobian matrix (the largest solution of a characteristic equation) is the basic reproductive number of the model. Therefore

$$\mathbf{R}_0^D = \frac{\theta}{(\mu+\gamma+\varepsilon)} + \frac{(\delta+\mu+\omega)\pi\beta e^{-\mu\tau}}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)}$$

3.5.2 The basic reproductive number for the stochastic model ($\mathbf{R}^S_0$)

To derive the basic reproductive for the stochastic model, we used the method that was used in the study by [12] in their paper. So let's take the infected class from the system of equation (1).

$$dI = (\theta I + \beta SI - (\mu+\gamma+\varepsilon)I)\,dt + \sigma_4 I dB_4(t) \quad (26)$$

Using the twice differentiable function of Ito's formula and taking the expansio in the Taylor series, we can drive the basic reproductive number for the stochastic model as follows:

$$df(t,I(t)) = \frac{\partial f}{\partial t}dt + \frac{\partial f}{\partial I}dI(t) + \frac{1}{2}\frac{\partial f^2}{\partial I^2}(dI(t))^2 + \frac{\partial f^2}{\partial t \partial I}dtdI + \frac{1}{2}\frac{\partial f^2}{\partial t^2}(dt)^2 \quad (27)$$

From Ito's formula for twice differentiable function on [0, T], we have f(I) = ln(I),

$$f(t,I(t)) = \ln I(t), \quad \frac{\partial f}{\partial I} = \frac{\partial \ln I(t)}{\partial I} = \frac{1}{I(t)}, \frac{\partial^2 f}{\partial I^2} = \frac{-1}{(I(t)^2} \text{ and}$$
$$\frac{\partial f}{\partial t} = \frac{\partial^{2f}}{\partial t \partial I} = \frac{\partial^2 f}{\partial t^2} = 0$$

Then when it expands in Taylor series

$$\begin{aligned} df(t,I(t)) &= \frac{\partial f}{\partial t}dt + \frac{\partial f}{\partial I}dI(t) + \frac{1}{2}\frac{\partial^2 f}{\partial I^2}(dI(t))^2 + \frac{\partial^2 f}{\partial t \partial I}dtdI + \frac{1}{2}\frac{\partial^2 f}{\partial t^2}(dt)^2 \\ &= 0dt + \frac{1}{I}dI(t) - \frac{1}{2I^2}(dI(t))^2 + 0dtdI + \frac{1}{2}0(dt)^2 \\ &= \frac{1}{I}dI(t) - \frac{1}{2I^2}(dI(t))^2 \end{aligned} \quad (28)$$

Let X = θI + βSI − (μ + γ + ε)I and Y = σ4I. Then

$$df(t,I(t)) = (\theta + \beta S(t) - (\mu + \gamma + \varepsilon))dt + \sigma_4 dB_4(t) - \frac{1}{2(I(t))^2}(X^2 d^2 t + 2XY dt dB_4(t)) + (Y dB_4(t))^2. \quad (29)$$

According to the rules of Brownian motion mentioned in [18], the differentials of the higher order of dt and dB4(t) = dt approach to zero, which means dt.dt = 0 = dt.$dB_4(t)$ and $d^2B4(t) = dB_4(t)dB_4(t) = dt$ (due to variance of a Wiener process).

$$df(t,I(t)) = (\theta + \beta S - (\mu + \gamma + \varepsilon))dt + \sigma_4 dB_4(t) - \frac{1}{2(I(t))^2}(X^2 0 + 2XY 0 + (Y^2 dt).$$

$$= [\theta + \beta S - \frac{\sigma_4^2}{2} - (\mu + \gamma + \varepsilon)]dt + \sigma_4 dB_4(t)] \quad (30)$$

From (30) we have $f = \theta + \beta S - \frac{\sigma_4^2}{2}$ and $v = (\mu + \gamma + \varepsilon)$. Since at disease free equilibrium point, $F = \theta + \frac{\beta\pi(\delta + \mu + \omega)}{(\alpha + \mu)(\delta + \mu) + \omega\mu} - \frac{\sigma_4^2}{2}, V = (\mu + \gamma + \varepsilon)$ and $V^{-1} = \frac{1}{\mu + \gamma + \varepsilon}$, then the product of F and V^{-1} gives

$$FV^{-1} = \left[\frac{\theta}{\mu + \gamma + \varepsilon} + \frac{\beta\pi(\delta + \mu + \omega)}{((\alpha + \mu)(\delta + \mu) + \omega\mu)(\mu + \gamma + \varepsilon)} - \frac{\sigma_4^2}{2(\mu + \gamma + \varepsilon)}\right] \quad (31)$$

Next, we have to compute the characteristic equation of (31) given by

$$\begin{aligned}\rho(\lambda) &= |FV^{-1} - \lambda| \\ \Rightarrow \quad \rho(\lambda) &= \Big|\frac{\theta}{\mu+\gamma+\varepsilon} + \frac{\beta\pi(\delta+\mu+\omega)}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)} - \frac{\sigma_4^2}{2(\mu+\gamma+\varepsilon)} - \lambda\Big| \\ &= \frac{\theta}{\mu+\gamma+\varepsilon} + \frac{\beta\pi(\delta+\mu+\omega)}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)} - \frac{\sigma_4^2}{2(\mu+\gamma+\varepsilon)} - \lambda \quad (32)\end{aligned}$$

Now, let's find the eigenvalues of the characteristic equation of (32) as follows

$$\rho(\lambda) = \Big|\frac{\theta}{\mu+\gamma+\varepsilon} + \frac{\beta\pi(\delta+\mu+\omega)}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)} - \frac{\sigma_4^2}{2(\mu+\gamma+\varepsilon)} - \lambda\Big| = 0$$

$$\lambda = \frac{\theta}{\mu+\gamma+\varepsilon} + \frac{\beta\pi(\delta+\mu+\omega)}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)} - \frac{\sigma_4^2}{2(\mu+\gamma+\varepsilon)}$$

Since the solution of the characteristic equation (i.e. largest eigenvalue) is the basic reproductive number for the developed model, then

$$R_0^S = \frac{\theta}{\mu+\gamma+\varepsilon} + \frac{\beta\pi(\delta+\mu+\omega)}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)} - \frac{\sigma_4^2}{2(\mu+\gamma+\varepsilon)}.$$

Considering only the deterministic part we obtain the basic reproductive number and it is the same as

$$R_0 = \frac{\theta}{\mu+\gamma+\varepsilon} + \frac{\beta\pi(\delta+\mu+\omega)}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)}$$

Similarly, we obtain the basic reproductive number for the stochastic delay model given by

$$\lim_{t\longrightarrow\infty} \sup \frac{1}{t} \ln I(t) \leq (\mu+\gamma+\varepsilon)(R_0^S - 1) < 0 \ \textit{if}\ R_0^S < 1$$

From the developed model, when we compare the basic reproductive number for deterministic, delay, stochastic, and stochastic delay, we obtain the following relationship: R0 < R^D_0 , due to taking into account the incubation period in our model. Including a time delay in our model allows us to minimize the value of the basic reproductive number, which implies that infected individuals remain in the exposed class for certain days and give us as use control mechanisms.$R^D_0 < R^S_0$ due to incorporating the environmental factor on a delay model.

$$R_0^{SD} = R_0^D - \frac{\sigma_4^2}{2(\mu+\gamma+\varepsilon)}.$$

This indicates that the base reproductive number of the stochastic delay model is the difference between the base reproductive number of the delay model and the given values. This means always $R^{SD}_0 < R^D_0$.

3.6 Local Stability of The Disease Free Equilibrium

Theorem 3.4. *For any initial values of S0, V0, P0, I0 and R0 tends to zero almost surely exponentially stable that obey*

$$\lim_{t\longrightarrow\infty} \sup \frac{1}{t} \ln I(t) \leq (\mu+\gamma+\varepsilon)(R_0^S - 1) < 0 \ \textit{if}\ R_0^S < 1$$

Proof. To prove Theorem (3.4) let's take f(t, I(t)) = ln I(t) and use Ito's formula.

$$df(t, I(t)) = d\ln I(t) = (\theta + \beta S(t-\tau)e^{-\mu\tau} - \frac{\sigma_4^2}{2} - (\mu+\gamma+\varepsilon))dt + \sigma_4 dB_4(t) \quad (33)$$

By integrating both sides of the equation (33) with respect to time t when its value runs from 0 to t, we get,

$$\int_0^t d\ln I(t) = \int_0^t (\theta + \beta S e^{-\mu\tau} - \frac{\sigma_4^2}{2} - (\mu+\gamma+\varepsilon))dt + \int_0^t \sigma_4 dB_4(t)$$

$$\ln I(t) = \ln I(0) + (\theta + \beta S e^{-\mu\tau} - \frac{\sigma_4^2}{2} - (\mu+\gamma+\varepsilon))t + \int_0^t \sigma_4 dB_4(t) \quad (34)$$

Where a martingale $G(t) = \int^t_0 \sigma_4 dB_4(t)$ Then at disease free equilibrium point of equation (34) becomes

$$\ln I(t) \leq \ln I(0) + (\theta + \frac{\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)}{(\alpha+\mu)(\delta+\mu)+\omega\mu)} - \frac{\sigma_4^2}{2} - (\mu+\gamma+\varepsilon))t + G(t) \quad (35)$$

Then divide both sides of equation (35) by t and assume t –→ ∞.

$$\frac{\ln I(t)}{t} \leq \frac{\ln I(0)}{t} + (\theta + \frac{\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)}{(\alpha+\mu)(\delta+\mu)+\omega\mu)} - \frac{\sigma_4^2}{2} - (\mu+\gamma+\varepsilon))t + \frac{G(t)}{t} \quad 36)$$

Applying limt–→∞ sup on both sides of equation (36) gives

$$\lim_{t\longrightarrow\infty} \sup\left(\frac{\ln I(t)}{t}\right) \leq \lim_{t\longrightarrow\infty} \sup\left(\frac{\ln I(0)}{t} + (\theta + \frac{\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)}{(\alpha+\mu)(\delta+\mu)+\omega\mu)} - \frac{\sigma_4^2}{2} - (\mu+\gamma+\varepsilon)\right) + \lim_{t\longrightarrow\infty} \sup \frac{G(t)}{t} \quad (37)$$

For a large numbers of martingale, we have $\lim_{t\longrightarrow\infty}\sup\left(\frac{G(t)}{t}\right)=0$ almost surely.

$$\lim_{t\longrightarrow\infty}\sup\frac{\ln I(t)}{t}\leq\lim_{t\longrightarrow\infty}\sup\left(\frac{\ln I(0)}{t}\right)+\theta+\frac{\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)}{(\alpha+\mu)(\delta+\mu)+\omega\mu)}-\frac{\sigma_4^2}{2}-(\mu+\gamma+\varepsilon)<0$$

$$=(\mu+\gamma+\varepsilon)\left(\frac{\theta}{\mu+\gamma+\varepsilon}+\frac{\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)}{(\alpha+\mu)(\delta+\mu)+\omega\mu)(\mu+\gamma+\varepsilon)}-\frac{\sigma_4^2}{2(\mu+\gamma+\varepsilon)}-1\right)$$

$$=(\mu+\gamma+\varepsilon)(R_0^S-1)$$

Since $(\mu+\gamma+\varepsilon)>0$, then (38) is less than one if $R^S_0<1$.

Therefore, the disease-free equilibrium point of the model is locally asymptotically stable, if $R^S_0<1$.

3.7 Global Stability of the Endemic Equilibrium Point

Theorem 3.5. *The endemic equilibrium point of the model is globally asymptotically stable if $R^S_0>1$.*

Proof. To prove this theorem, let's construct the Lyapunov function as follow:

$$V(S^*,V^*,P^*,I^*,R^*)=(S-S^*-S^*\ln S)+(V-V^*-V^*\ln V)+(P-P^*-P^*\ln P)$$
$$+(I-I^*-I^*\ln I)+(R-R^*-R^*\ln R) \quad (38)$$

By differentiating both sides of equation (38) with respect to t, we get the following

$$\frac{dL}{dt}=\left(1-\frac{S^*}{S}\right)\frac{dS}{dt}+\left(1-\frac{V^*}{V}\right)\frac{dV}{dt}+\left(1-\frac{P^*}{P}\right)\frac{dP}{dt}+\left(1-\frac{I^*}{I}\right)\frac{dI}{dt}+\left(1-\frac{R^*}{R}\right)\frac{dR}{dt}$$

$$=\left(1-\frac{S^*}{S}\right)(\pi+\rho R+\omega V-\beta SIe^{-\mu\tau}-(\alpha+\mu)S)+\left(1-\frac{V^*}{V}\right)(\alpha S-(\delta+\omega+\mu)V)$$

$$+\left(1-\frac{P^*}{P}\right)(\delta V-\mu P)+\left(1-\frac{I^*}{I}\right)(\beta SIe^{-\mu\tau}-(\mu+\gamma+\varepsilon-\theta)I)$$

$$+\left(1-\frac{R^*}{R}\right)(\gamma I-(\mu+\rho)R)$$

$$=\pi+S^*(\beta I+\alpha+\mu)+V^*(\delta+\omega+\mu)+P^*\mu+I^*(\mu+\gamma+\varepsilon)+R^*(\mu+\rho)$$

$$-\left[\mu S+\frac{S^*}{S}(\pi+\rho R+\omega V)+\mu V+\frac{V^*\alpha S}{V}+\mu P+\frac{P^*\delta V}{P}+(\mu+\varepsilon-\theta)I\right.$$

$$\left.+I^*(\theta+\beta S)+\mu R+\frac{R^*\gamma I}{R}\right] \quad (39)$$

Let $A=\pi+S^*(\beta I+\alpha+\mu)+V^*(\delta+\omega+\mu)+P^*\mu+\theta I+I^*(\mu+\gamma+\varepsilon)+R^*(\mu+\rho)$ and

$$B=\mu S+\frac{S^*}{S}(\pi+\rho R+\omega V)+\mu V+\frac{V^*\alpha S}{V}+\mu P+\frac{P^*\delta V}{P}+(\mu+\varepsilon)I+I^*(\theta+\beta S)+\mu R+\frac{R^*\gamma I}{R}$$

Then, by substituting A and B in equation (39) we get

$$\frac{dV}{dt} = A - B \qquad (40)$$

Here,dV/dt < 0 , ifA ≤ B and dV/dt = 0 if and only if S = S*, V = V*, P = P*, I = I*,R = R*, in the invariant region. Therefore, by Lasalle?s invariant principle, the endemic equilibrium point of the model is globally asymptotically stable in Ω if A ≤ B.

3.8 Sensitivity Analysis

Sensitivity analysis of basic parameters is done to identify which parameters have impact on the expansion or role in controlling the dynamics of rubella disease. To perform a sensitivity analysis of the model, we used the forward sensitivity index formula given by

$$P_{\chi_i} R_0^S = \frac{\partial R_0^S}{\partial \chi_i} * \frac{\chi_i}{R_0^S}$$

, where χi is the parameter in the basic reproductive number.
For χi = β, then

$$P_\beta R_0^S = \frac{\partial R_0^S}{\partial \beta} * \frac{\beta}{R_0^S} = \frac{\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(2\theta-\sigma_4^2)+2\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)}$$

$$\text{For } \chi_i = \alpha, \text{then } P_\alpha R_0^S = \frac{\partial R_0^S}{\partial \alpha} * \frac{\alpha}{R_0^S}$$

$$= -\frac{\alpha\beta\pi(\delta+\mu+\omega)(\delta+\mu)e^{-\mu\tau}}{((\alpha+\mu)(\delta+\mu)+\omega\mu)[((\alpha+\mu)(\delta+\mu)+\omega\mu)(2\theta-\sigma_4^2)+2\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)]}$$

$$\text{For } \chi_i = \pi, \text{then } P_\pi R_0^S = \frac{\partial R_0^S}{\partial \pi} * \frac{\pi}{R_0^S}$$

$$= \frac{\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(2\theta-\sigma_4^2)+2\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)}$$

$$\text{For } \chi_i = \omega \text{ then } P_\omega R_0^S = \frac{\partial R_0^S}{\partial \omega} * \frac{\omega}{R_0^S}$$

$$= \frac{\beta\pi\alpha\omega(\delta+\mu)e^{-\mu\tau}}{((\alpha+\mu)(\delta+\mu)+\omega\mu)[(\alpha+\mu)(\delta+\mu)+\omega\mu)(2\theta-\sigma_4^2)+2\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)}$$

$$\text{For } \chi_i = \delta, \text{ then } P_\delta R_0^S = \frac{\partial R_0^S}{\partial \delta} * \frac{\delta}{R_0^S}$$

$$= \frac{\beta\pi e^{-\mu\tau}\delta\omega(\alpha+\mu)(\mu-1)}{((\alpha+\mu)(\delta+\mu)+\omega\mu)[((\alpha+\mu)(\delta+\mu)+\omega\mu)(2\theta-\sigma_4^2)+2\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)]}$$

$$\text{For } \chi_i = \theta, \text{ then } P_\theta R_0^S = \frac{\partial R_0^S}{\partial \theta} * \frac{\theta}{R_0^S}$$

$$= \frac{\theta((\alpha+\mu)(\delta+\mu)+\omega\mu)}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(2\theta-\sigma_4^2)+2\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)}$$

$$\text{For } \chi_i = \sigma_4, \text{ then } P_{\sigma_4} R_0^S = \frac{\partial R_0^S}{\partial \theta} * \frac{\theta}{R_0^S}$$

$$= -\frac{\sigma_4((\alpha+\mu)(\delta+\mu)+\omega\mu)}{((\alpha+\mu)(\delta+\mu)+\omega\mu)(2\theta-\sigma_4^2)+2\beta\pi e^{-\mu\tau}(\delta+\mu+\omega)}$$

Sensitivity analysis results show that an increase in the value of a basic parameter with positive sensitivity indices such as β, θ, and ω leads to an increase in the value of the basic reproductive number. This leads to an increase in secondary infections caused by single individual infected with rubella. On the other hand, based on the constructed model increasing the value of a parameter with sensitivity indices such as α, γ, δ, σ4 and τ with negative values plays a role in controlling (reducing) rubella disease in the community. Using the values given in Table (2), we obtain the following values given in Table (2), which represent the sensitivity indices for the basic parameters.

Table 1: Parameters and their sensitivity indices for stochastic delay model

Parameter	Description	Sensitivity indices
θ	vertical transmission rate	1.4223
β	Contact rate	0.0891
ω	Rate of waning out of the first vaccination dose	0.00046518
δ	rate of the second vaccination dose	−0.0453
γ	Recovery rate	−0.3061
α	First vaccination dose	−0.0011
σ_4	Intensity rate	−2.5285

Numerical Simulations

In this section, we attempt to perform and analyze a stochastic delay mathematical model of rubella dynamics using numerical simulations. For this purpose, we used the parameter values obtained from the literature that shown in Table (2). The numerical analysis of the model is graphically illustrated in Figure (2) using the parameter values under the consideration of stochastic factors and time delay. The figure shows the time series solution for model system equation (1). As shown in the figure, the behaviour of the system equation in the model changes over time as the parameter values changes. The figure reveals that the infection and recovery classes are close to zero because of the incorporation of time delay in the system of equation leads to the exposed person to stay in that class for some time which helps to give opportunities for health and all stakeholders to protect against the spread of rubella disease using a preventative mechanism. However, the susceptible, vaccinated, and permanent immunized populations will increase over time in both delay and stochastic delays patterns due to time delay. This suggests that including a time delay in the model slows

the progression of the number of infected people for several days. The figure indicates that the model variables S(t), V (t), P(t), I(t), and R(t) initially fluctuate and eventually approach equilibrium with increasing time delay τ . In delay part it oscillates from the origin to the maximum time delay used in the description which ranges from τ = 0 to τ = 23. Then, after the maximum time delay, the curves of the simultaneous equations become smooth. This suggests that, considering the time delay in our model, the number of people infected with rubella a second time from an infected person will decrease. This suggests that, as we consider the time delay in our model, the number of people infected with rubella is reduced. Furthermore, as when we include time delay and stochastic perturbations, the curves of the system of equation move up and down. Based on the idea mentioned in the study of [24] and the developed model, we have tried to graphically represent it in Figure (2), which shows that the number of infectious can be high or low due to environmental disturbance (stochastic disturbance), as shown in the figure.

Table 2: Parameter, description, value, and source

Parameter	Description	Value	Source
π	The recruitment rate	0.025	Prawoto et al , (2020)
β	Contact rate	0.4	Alqurash (2020)
ε	Death rate due to rubella disease	0.08	Assumed
δ	Rate of second vaccination dose	0.85	[1]
μ	Natural death rate	0.015	Koca (2018)
γ	Recovery rate	0.15	[2]
θ	Rate of infected infants	0.55	Assumed
α	Rate of first vaccinated dose	0.3	Baleanu et al (2020)
ω	waning rate of first vaccination dose	0.6	[2]
ρ	Rate of temporary immunity	0.01	Assumed

4.1 Effect of Contact Rates on Rubella-Infected Individuals

Figure 3 shows the impact of contact rate on the expansion of disease. As shown in the figure, the graph of the delay part oscillates from the initial to the maximum time delay, and after the maximum time delay, the curves of the system of equations are smooth and close to zero. It shows the numerical results obtained by examining the effect of the contact rate on an infected individual by varying its value while keeping other parameters constant. The results show that when we increase the contact rate significantly, it increases the number of infected

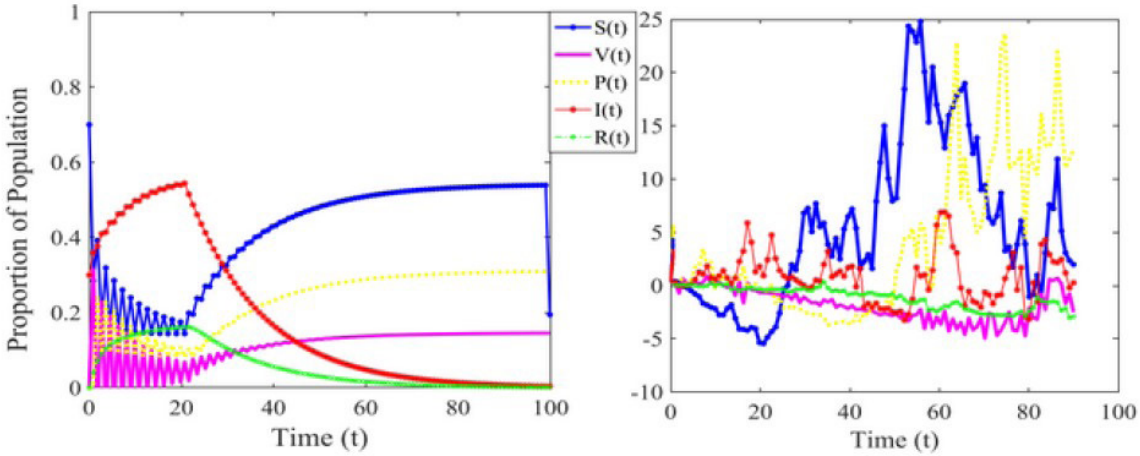

Figure 2: Graph of the delay model (left) and stochastic-delay model (right).

people. This means that when vulnerable people come into contact with rubellacontaminated material or rubella-infected people, the number of rubella-infected people in the community increases

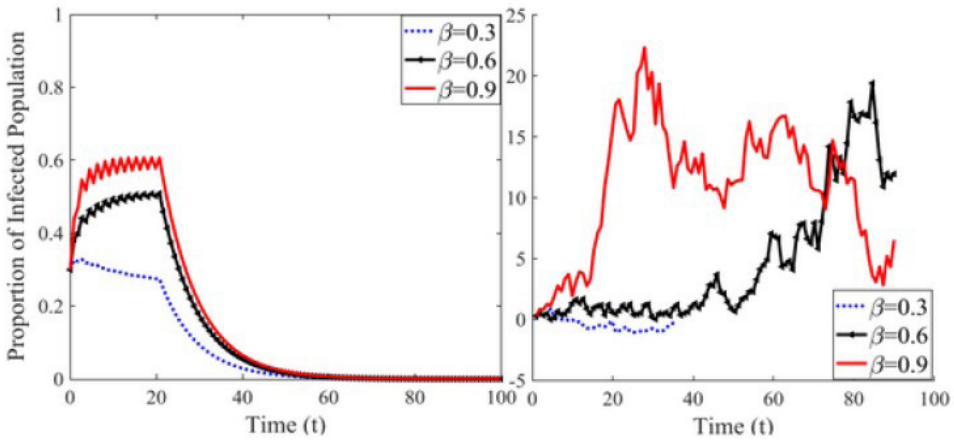

Figure 3: Effects of contact rates on rubella infected.

4.2 Effect of Vertical Transmission Rate on Rubella-Infected Population

Figure 4 shows the results of numerical simulations examining the effect of the vertical transmission rate (θ) on the number of rubella-infected population. The result is obtained by changing the value of θ from 0.45 to 0.55 while keeping the other parameters constant. As shown in the figure, as the proportion of passively infected neonates' increases due to vertical maternal-to-fetal transmission, the number of infected cases spikes.

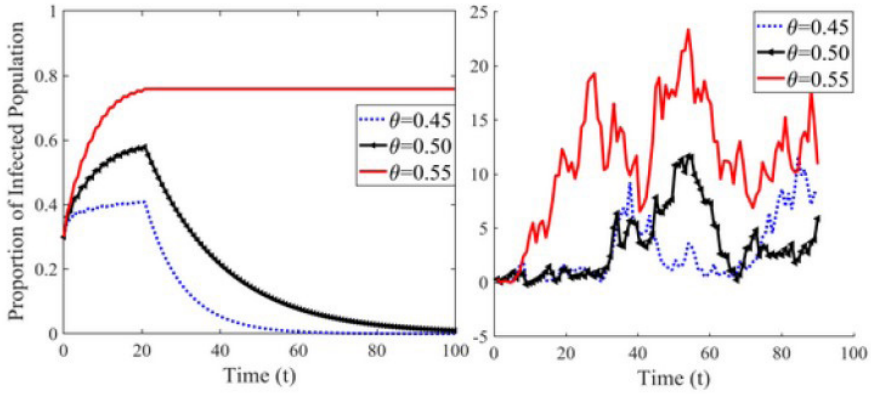

Figure 4: Effects of vertical transmission rates on rubella infected.

4.3 Effect of Recovery and Vaccination Rates on Rubella-Infected People

As shown in Figure (5), as the value of the recovery rate increases, the number of infected people decreases, while the values of the other parameters are assumed to be constant. This suggests that raising this rate will play an important role in eradicating rubella in the community. Moreover, as it is shown in Figure (6), increasing the dose rate of the first and second vaccinations reduces the number of local infections.

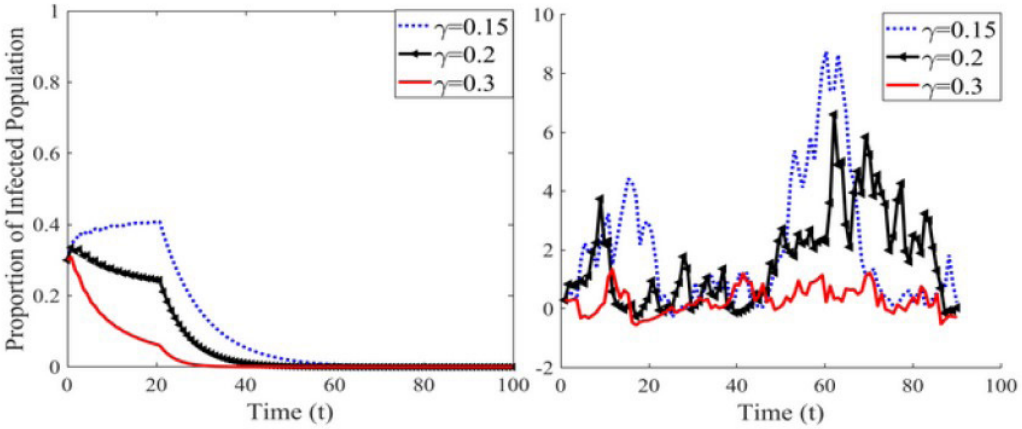

Figure 5: Effect of recovery rate on rubella infected.

Discussion and Conclusion

This study was conducted to investigate the dynamics of rubella disease using a stochastic-delay mathematical modeling approach. Based on the biological behavior of rubella disease dynamics, a SVPIRS stochastic delay mathematical model was developed under the consideration the role of incubation period, stochastic factor, vertical transmission and two dose of MMR vaccine. A basic

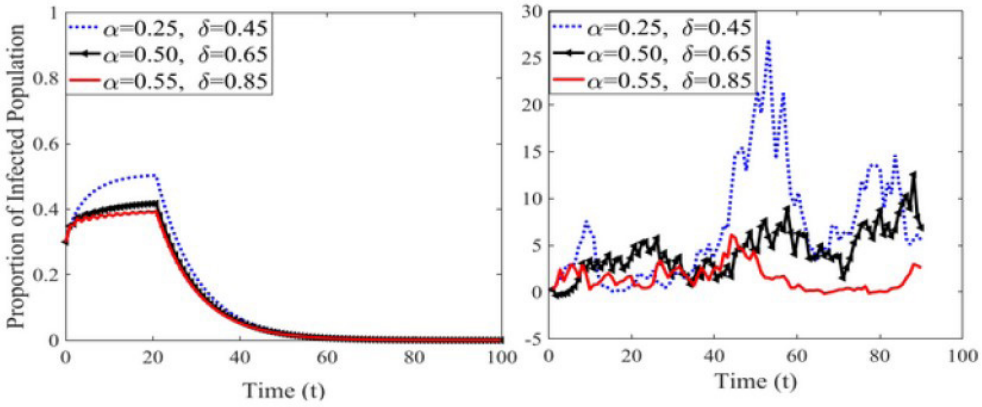

Figure 6: Effects of vaccination rate on rubella infected.

qualitative analysis of the model was performed. The local stability of the diseasefree equilibrium point of the model is determined using the Jacobi method. To determine the presence of rubella disease in the community, basic reproductive number was calculated using a next-generation matrix. To perform numerical simulations, the values of some fundamental parameters are taken from published studies, while others are assumed. Finally, we discuss the results of numerical simulations using MATLAB 2019 computer software to demonstrate the effect of the parameters, and present the results graphically. Results show reducing rates of passively infected newborns, contact rates and increase in recovery rates, incubation periods, and first and second dose vaccination rates play a role in control. From the qualitative analysis of the model, we find that the basic reproductive number of the stochastic model appears to be smaller than that of the delay model of due to environmental variation-induced dynamics of rubella disease. This means that consideration of environmental factors plays a role in minimizing the average number of secondary infections. Because stochastic-delay model includes dynamic behavior, stochastic factors, and time delay, the basic reproductive number obtained from stochastic-delay model is smaller than those for delay and stochastic models. This shows that the mathematical model of stochastic delay is closer to reality than deterministic and delays.

Acknowledgments

We, the authors, would like to express our deepest gratitude to Haramaya University for providing us with internet access and taking the time to write this article. In addition, we would like to thank your journal and the reviewers responsible for reviewing our paper.

Availability of Data and Materials

The data we used for this research is from respective published articles that are cited.

Conflicts of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

Author's Contributions

All authors have equal contributions from model development up to analysis

Funding

No funding received

References

1. T TG, M TT, A WG (2022) Modeling the dynamics of rubella disease with vertical transmission. Heliyon. P PB, Abadi, R A. Dynamic of re-infection Rubella transmission model with vaccination. In AIP Conference Proceedings. 2264.

2. Plotkin SA (2006) Mass vaccination: global aspects-progress and obstacles. 304.

3. YW, MT, GF, KD, FA, BT (2018) Hospital-based surveillance of congenital rubella syndrome cases in the pre-vaccine era in Amhara Regional State, Ethiopia: baseline information for the country. PLoSONE. 13.

4. BT, SH, TS (2017) Rubella virus infection and associated factors among pregnant women attending the antenatal care clinics of public hospitals in Hawassa City, Southern Ethiopia: a cross-sectional study. BMJ open. 7.

5. MG, BB, KG, AA, BT, MT, et al. (2016) Epidemiology of rubella virus cases in the pre-vaccination era of Ethiopia, 2009–2015. BMC Public Health. 16: 1–7.

6. S MAM, S MM, KL, MA, A EBA, H RM (2021) Numerical solution and dynamical behaviors for solving fractional nonlinear Rubella ailment disease model. Results in Physics. 24.

7. Organization WH (2012) Surveillance guidelines for measles, rubella and congenital rubella syndrome in the WHO European Region. In Surveillance Guidelines for Measles, Rubella and Congenital Rubella Syndrome in the WHO European Region.

8. Y LM (2018) An introduction to mathematical modeling of infectious diseases. Cham, Switzerland:Springer. 2.

9. A HS, SB, NH (2017) Mathematical models used to inform study design or surveillance systems in infectious diseases: a systematic review. BMC infectious diseases. 17: 1–10.

10. PY, SL (2006) SEIR epidemic model with delay. The ANZIAM Journal. 48: 119–34.

11. ON (2016) Biological Models With Time Delay. (Doctoral dissertation, WORCESTER POLYTECHNIC INSTITUTE). 1: 1-15.

12. A RF, J AH, C R (2020) Stochastic SIRC epidemic model with time delay for COVID-19. Advances in difference equations. 1: 1-20.

13. AL, LO, DK (2011) Global analysis of a deterministic and stochastic nonlinear SIRS epidemic model. Nonlinear Analysis: Modelling and Control. 16: 59-76.

14. SG, IL, AL (1999) Small delay approximation of stochastic delay differential equations. Physical Review E, 59: 3970.

15. B GG, S D, L D, KK, E RS (2019) Progress toward rubella and congenital rubella syndrome control and elimination—Worldwide, 2000–2018. Morbidity and Mortality Weekly Report. 68: 855.

16. Qurashi A, M M (2020) Role of fractal-fractional operators in modeling of rubella epidemic with optimized orders. Open Physics. 18: 1111–20.

17. A A, T ABS (2016) Modeling the spread of Rubella disease using the concept of local derivative with fractional parameter. Beta-Derivative Complexity. 21: 442–51.

18. I K (2018) Analysis of rubella disease model with non-local and non-singular fractional derivatives. An International Journal of Optimization and Control: Theories and Applications (IJOCTA). 8: 17–25.

19. S R, S E, H M (2020) A mathematical analysis of a system of Caputo-Fabrizio fractional differential equations for the anthrax disease model in animals. Advances in Difference Equations. 2020: 481.

20. der Heijden O G V, van Spaendonck M A E C, D PA, E KME (1998)A model- based evaluation of the national immunization programme against rubella infection and congenital rubella syndrome in the Netherlands. Epidemiology & Infection. 121: 653–71.

21. E MCJ, J L, P K, F C, T GB. Impact of birth rate, seasonality and transmission rate on minimum levels of coverage needed for rubella vaccination. Epidemiology and Infection. 140: 2290–301.

22. M YH, R FAR (2019) Biological view of vaccination described by mathematical modeling: From rubella to dengue vaccines. Math BiosciEng. 16: 3195-214.

23. E EW, S O, M W (2019) Modelling and analysis of seir with delay differential equation. Global Journal of Pure and Applied Mathematics. 15: 365– 82.