

## Research Article

# Modelling, Analysis, and Simulation of Measles Disease Transmission Dynamics

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Measles is one of the top communicable diseases, which is still responsible for 2.6 million deaths every year. Due to this reason, the paper focuses on measles transmission dynamics concerning the impact of indirect contact rate (transmitted from the host of the virus to the healthy individual) and improving the SEVIR model into the SVIRP model. From the model, we first estimated the disease-free equilibrium, calculated the effective reproduction number ( $R_{\text{Eff}}$ ), and established the stability analysis. The Castillo–Chavez stability criterion is used to demonstrate the global stability of the disease-free equilibrium point, while the linearization method is used to justify its local stability analysis and gives a result  $R_{\text{Eff}} < 1$ . The stability analysis of endemic equilibrium point is explained by defining a Lyapunov function, and its global stability exists when  $R_{\text{Eff}} > 1$ . To identify the effect of parameters on the transmission dynamics, we performed sensitivity index and numerical simulation. From the result, we obtained that the indirect contact rate has the highest impact in maximizing the transmission dynamics of measles. Also, we found that working on prevention and treatment strategies brings a significant contribution in reducing the disease effect in the community.

## 1. Introduction

Measles is an infectious disease which was first acknowledged in Boston in 1675 [1]. It is an acute, highly infectious viral disease caused by morbillivirus (measles virus) for which humans are the only reservoirs [2]. In the last 2 decades, the global cases of measles have been declining before the emergence of COVID-19 pandemic. The number of measles infections increased in 2019, reaching 869,770 cases and 207,500 deaths, which is the highest incidence of the disease since 1996 [3]. Recently, there has been an increase in measles infections in sub-Saharan Africa, with 17,500 cases altogether as of January 2022, a 400% spike from cases reported in 2021 [4]. As shown in Table 1, Angola, Burundi, Cameroon, Chad, Democratic Republic of the Congo, Ethiopia, Somalia, South Sudan, and Togo are some few countries that have recently seen measles cases [3].

The primary source of transmission is through direct contact with the nose and throat secretions of an infected

person or by aerosolized droplets [2]. It spreads person to person at a high rate of over 90% among the vulnerable people due to the mode of transmission and infectious property [5]. When measles virus infects a nonimmune population, almost everyone will become infected and develop clinical illness [2, 6]. When an infected individual coughs or sneezes due to respiratory tissue or aerosol droplets, the virus can survive in the atmosphere for up to two hours [4].

The primary public health approaches to minimize measles prevalence internationally include mass immunization campaigns with routine MMR vaccination for children in nations with high rates of cases and fatalities. This is because some vaccines, including two doses of MMR, are nearly 100% effective at preventing measles (Measles, Mumps, and Rubella) vaccine will protect 99% of people from measles [1, 7–9].

Regardless of the availability of measles immunization, there were 207,500 predicted measles mortalities globally in

TABLE 1: Top 5 countries with reported measles cases in the last 12 months, until April 2022.

Country	Reported measles cases	Rate per million cases	First dose measles coverage (%), 2019	First dose measles coverage (%), 2020
Somalia	9068	554	46	46
Yemen	3629	119	67	68
Afghanistan	3628	91	64	66
Nigeria	12,341	58	54	54
Ethiopia	3039	26	60	58

2019, of which 147,900 (more than 70%) were in African nations [7, 10]. Globally, it was forecasted that 134,200 children would die from measles in 2015, making it the highest public health issue. Currently, crises in countries such as Afghanistan, Ethiopia, Somalia, and Ukraine are forcing millions of children to leave their homes. Immunization programs and other crucial children's health services have been interrupted in these countries. Children are crammed together in crowded conditions. These conditions improve the likelihood of a measles outbreak [1, 8, 11].

Mathematical modeling of infectious diseases can be used to comprehend the dynamics of communicable disease transmission and to inform public health policy makers on how to implement effective intervention programs to fight infections. To study the dynamics of measles illness spread, numerous researchers have designed various mathematical models. Among them, Momoh et al. [12] developed a mathematical model of measles transmission dynamics for measles epidemiology considering the impact of exposed individuals at the latent period and discussed through the stability analysis and numerical simulation. Edward et al. [7] formulated the SEIRV model for measles, and the model has shown importance of measles vaccination in preventing transmission within a population. They conclude from their findings that the spread of a disease largely depends on the contact rates and also the proportion of the population that is immune exceeds the herd immunity level of measles. The mathematical model set by Siam and Nasir [13] was an investigation of an infectious model of a measles outbreak without vaccination in which the population is divided into susceptible, latent, infected, post-infection, and recovered using ordinary differential equations.

The mathematical model of measles developed by Alhamami [14] used five ordinary differential equations with SEIRV compartmental model of the classes of the total population. The researcher tried to show that the impact of vaccination rate implementation using model simulation of measles infection is lower when higher measles vaccination rate is applied. An SEIR epidemic model for measles epidemic in human populations with vaccination and treatment on a homogeneously mixed population was proposed by Berhe et al. [15], and the susceptible individuals were recruited by immigration or birth at per capital rate.

Another simulation of measles transmission dynamics under the intervention of vaccination was performed by [5] to investigate the transmission of measles virus using the five categories of susceptible, vaccinated, exposed, infectious, and

recovered individuals with demographic factors using the deterministic compartment model. Tilahun and Alemneh [16] developed a model of measles transmission dynamics with double dose vaccination. The model was firstly developed as a deterministic approach and then converted into stochastic approach to determine the significant role of stochastic approach rather than deterministic approach, and the analysis of positivity of the solution, invariant region of the solution, the existence of equilibrium points, and their stability and sensitivity analysis of parameters of the basic reproductive number of both the models were analyzed and done in deterministic and stochastic approaches. A mathematical model of measles was formulated by Ogundare and Akingbade et al. [17] with an open population using ordinary differential equation over the three mutually exclusive classes of the population susceptible, infective, and recovered in the deterministic mathematical modeling approach. Recently, a mathematical model was developed by Rahmayani et al. [1] on measles transmission with vaccination and treatment intervention. In a similar manner, the model developed by Xue et al. [18] on the measles dynamics of network model tries to emphasize a transmission rate and theoretically examine the threshold dynamics to investigate the influence of heterogeneity and waning immunity of measles transmission dynamics. Pokharel et al. [19] recently developed a novel transmission dynamics model to evaluate the effects of monitored vaccination programs to control and eliminate measles.

In accordance with each of the most recent articles on the dynamics of measles transmission, all of the aforementioned studies do not develop an epidemiological or mathematical model of measles infection outbreak that emphasizes the persistence of the measles virus on objects, surfaces, or in the atmosphere. Therefore, the goal of this work is to construct an SVIRP deterministic mathematical model while also improving the SVIR deterministic mathematical model by introducing a compartment for the host of the measles virus's capability to survive in the atmosphere and other infected materials.

## 2. Model Description and Formulation

The model is proposed by considering five mutually exclusive divisions of subclasses of the total population size at time " $t$ " using compartments of deterministic ordinary differential equations in a mixed homogeneous population. The total population denoted by  $N(t)$  is the sum of the individual populations in each subclass. Susceptible ( $S(t)$ ) represents individuals who have not yet been infected with

the disease, but are susceptible to the disease and become infected. Vaccinated ( $V(t)$ ) represents individuals who have received a vaccine. Infected ( $I(t)$ ) represents individuals who have been infected with the disease and capable of spreading the disease to susceptible. Recovered ( $R(t)$ ) represents individual who have been infected and recovered from the disease. Pathogen population ( $P(t)$ ) is the host of measles virus.

The class of susceptible is increased by birth or immigration at the rate of  $\pi$  with fraction  $q$  of the newly recruited members of the community that are vaccinated, and from susceptible individuals vaccination at a rate of  $\eta$ . The susceptible class decreased following infection with measles forces of infections is given as  $=\beta_1 I + \beta_2 P$ .

The class of vaccinated individuals is decreased by the rate of waning vaccine for susceptible individuals at a rate of  $\alpha$ ; however, it is reduced by rate  $\varepsilon\lambda$ . The infected class is increased by the contact rate to the susceptible class by the rate of  $\lambda$  and it is also increased by reinfection of vaccinated at the rate of  $\varepsilon\lambda$ , but decreased by the recovery of an individual at a rate of  $\varphi$  and due to induced death rate of  $\delta$ . The infected individuals are contaminating the environment at the rate of  $\theta$ . The recovered class is increased because of the recovery of individuals from infection at the rate of  $\varphi$  and perfect vaccination at the rate of  $\gamma$ . The natural death rate of the human population is considered at a rate of  $\mu$ . The pathogen population is generated by the rate of  $\theta$  due to the sneezing and coughing of infected individual on the air and droplets of measles virus on different objects, and decay rate of the pathogen is assumed as  $\mu_p$ . The other parameters are described in Table 2.

Therefore, according to the relationship justified above, the transmission of measles dynamics is generalized in the following compartmental flow chart in Figure 1.

Using the above description of measles transmission compartmental flow chart, we get the following measles transmission dynamics model:

$$\begin{aligned} \frac{dS}{dt} &= (1-q)\pi - \lambda S + \alpha V - (\mu + \eta)S, \\ \frac{dV}{dt} &= \pi q - \varepsilon\lambda V + \eta S - (\mu + \gamma + \alpha)V, \\ \frac{dI}{dt} &= \lambda S + \varepsilon\lambda V - (\theta + \mu + \delta + \varphi)I, \\ \frac{dR}{dt} &= \varphi I + \gamma V - \mu R, \\ \frac{dP}{dt} &= \theta I - \mu_p P. \end{aligned} \quad (1)$$

With the initial conditions

$$\begin{aligned} S(0) &= S_0 \geq 0, V(0) = V_0 \geq 0, I(0) \\ &= I_0 \geq 0, R(0) = R_0 \geq 0, P(0) = P_0 \geq 0. \end{aligned} \quad (2)$$

TABLE 2: Description of parameters of the deterministic model.

Parameters	Interpretation
$\pi$	Recruitment of susceptible population
$\beta_1$	Disease transmission rate of susceptible individual with infectious individuals
$\beta_2$	Disease transmission rate of susceptible with pathogen in air and droplets on objects
$\mu$	Natural death rate of humans
$\eta$	The rate of susceptible individuals receive vaccination
$\alpha$	The rate of waning of vaccination
$\varepsilon$	Modification parameter
$\gamma$	The rate of recovery of a vaccine individuals
$\theta$	Contribution of infected individuals to the population of pathogen
$\varphi$	Recovery rate of infected individuals
$\delta$	Disease induced death rate of infected individuals
$\mu_p$	The rate dying of pathogen
$q$	Cohort vaccination rate

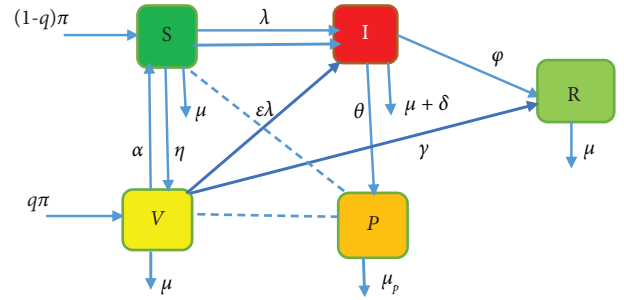


FIGURE 1: Compartmental model of measles disease transmission flow chart.

### 3. Basic Properties of the Model

#### 3.1. Invariant Region

**Theorem 1.** Suppose the system of equation in the model holds, then the feasible solution set  $\Omega = \{(S, V, I, R, P) \in \mathbb{R}^5\}$  of the model with initial condition  $S(0) = S_0 \geq 0, V(0) = V_0 \geq 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0, P(0) = P_0 \geq 0$ .

Then,  $\Omega = \{(S, V, I, R, P) \in \mathbb{R}^5 : 0 \leq N \leq \pi/N\}$  is a bounded region.

*Proof.* For the model in our assumption, the total population is given by the following equation:

$$N(t) = S(t) + V(t) + I(t) + R(t) + P(t). \quad (3)$$

Then, integrating  $N$  with respect to  $t$ , we have the following equation:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dI}{dt} + \frac{dR}{dt} + \frac{dP}{dt}. \quad (4)$$

After substituting the system of equation (1) into equation (4),

$$\frac{dN}{dt} = \pi - \mu(S + V + I + R + P) + \alpha V - \delta I = \pi - \mu N + \alpha V. \quad (5)$$

In the absence of death due to measles (i.e.,  $I = 0$  and  $P = 0$ ), equation (5) becomes as follows:

$$\frac{dN}{dt} \leq \pi - \mu N. \quad (6)$$

By integrating and taking limit as  $t \rightarrow \infty$  in equation (6) both sides, we get the following equation:

$$\Omega = \left\{ (S, V, I, R, P) \in \mathbb{R}^5 : 0 \leq N \leq \frac{\pi}{\mu} \right\}, \quad (7)$$

which is a non-negative invariant set for the system of the above given model.

### 3.2. Positivity of Solutions

**Theorem 2.** *If the initial values  $(S(0), V(0), I(0), R(0), P(0)) \geq 0$  belong to the invariant set  $\Omega$ , then the solution set  $\{S(t), V(t), I(t), R(t), P(t)\}$  of the model is positive for all time  $t \geq 0$ .*

*Proof.* From the third equation of the system in equation (1) we have the following equation:

$$\begin{aligned} \frac{dI}{dt} &= (\beta_1 I + \beta_2 P)S + \varepsilon \beta_1 I + \beta_2 P - (\theta + \mu + \delta + \varphi)I, \\ \frac{dI}{dt} &\geq -(\theta + \mu + \delta + \varphi)I, \end{aligned} \quad (8)$$

$$I(t) \geq I_0 e^{-(\theta + \mu + \delta + \varphi)t}.$$

From the last equation of the system in equation (1), we have the following equation:

$$\begin{aligned} \frac{dP}{dt} &= \theta I - \mu_P P, \\ \frac{dP}{dt} &\geq -\mu_P P, \end{aligned} \quad (9)$$

$$P(t) \geq P_0 (e^{-\mu_P t}).$$

From equation (1) of the system, we get the following equation:

$$\begin{aligned} \frac{dS}{dt} &= (1 - q)\pi - (\beta_1 I + \beta_2 P)S + \alpha V - (\mu + \eta)S, \\ \frac{dS}{dt} &\geq -(\beta_1 I + \beta_2 P + \mu + \eta)S, \end{aligned} \quad (10)$$

$$S(t) \geq S_0 \left( e^{-(\beta_1 I + \beta_2 P + \mu + \eta)t} \right),$$

$$S(t) \geq S_0 \left( e^{-(\beta_1 I_0 e^{-(\theta + \mu + \delta + \varphi)t} + \beta_2 P_0 (e^{-\mu_P t}) + \mu + \eta)t} \right) \geq 0.$$

Similarly, the solutions of the rest two are obtained as follows:

$$V(t) \geq V_0 e^{-\varepsilon(\beta_1 I + \beta_2 P + \mu + \gamma)t} \geq 0 \text{ and } R(t) \geq R_0 (e^{-\mu t}) \geq 0.$$

Thus, the solution of set of  $\{(S, V, I, R, P)\}$  for  $t \geq 0$  in the region  $\Omega$ , then we can deduced that the state variables of the system are all positive for all time  $t \geq 0$ .

**3.3. Disease-Free Equilibrium Point (DFEP).** The population is infection-free at the disease-free equilibrium point (DFEP), also known as the steady state solution (the endemic is eradicated from the population) [20], and obtained by letting all the right hand sided of the system of equations in the model to zero:

$$\frac{dS(t)}{dt} = \frac{dV(t)}{dt} = \frac{dI(t)}{dt} = \frac{dR(t)}{dt} = \frac{dP(t)}{dt} = 0. \quad (11)$$

In addition, the values of infected ( $I$ ) = pathogen population ( $P$ ) = 0.

Thus, from model (1), we have three disease-free equations. These are as follows:

$$\begin{cases} (1 - q)\pi - (\beta_1 I + \beta_2 P)S + \alpha V - (\mu + \eta)S + \omega P = 0, \\ q\pi - \varepsilon(\beta_1 I + \beta_2 P)V + \eta S - (\mu + \gamma)V = 0, \\ \varphi I + \gamma V - \mu R = 0, \end{cases} \quad \begin{cases} (1 - q)\pi + \alpha V - (\mu + \eta)S = 0, \\ q\pi + \eta S - (\mu + \gamma)V = 0, \\ \gamma V - \mu R = 0, \end{cases} \quad \because I = P = 0 \quad (12)$$

Then, solving the system in equation (12) simultaneously, we obtained the following equation:

$$\begin{aligned} V &= \frac{\pi(\eta + \mu q)}{(\mu + \eta)(\mu + \gamma) + \mu \alpha}, \\ S &= \frac{\pi(\mu + \gamma)(1 + \alpha - q)}{(\mu + \eta)(\mu + \gamma) + \mu \alpha}, \\ R &= \frac{\pi \gamma (\eta + \mu q)}{\mu(\mu + \eta)(\mu + \gamma) + \mu^2 \alpha}. \end{aligned} \quad (13)$$

Therefore, the disease-free equilibrium point denoted by  $E^*$  is as follows:

$$E^* = (S^*, V^*, I^*, R^*, P^*) = \left( \frac{\pi(\mu + \gamma)(1 + \alpha - q)}{(\mu + \eta)(\mu + \gamma) + \mu\alpha}, \frac{\pi(\eta + \mu q)}{(\mu + \eta)(\mu + \gamma) + \mu\alpha}, 0, \frac{\pi\gamma(\eta + \mu q)}{\mu(\mu + \eta)(\mu + \gamma) + \mu^2\alpha}, 0 \right), \quad (14)$$

which represents that there is no infection in the population.

**3.4. Effective Reproduction Number.** It can be determined using the method of next-generation matrix and represents

the spectral radius of the largest magnitude of the next-generation matrix [21], i.e.,  $R_{\text{Eff}} = \rho(FV^{-1})$ . According to the system in model (1), there are two infectious compartments with 2 by 2 matrices.

$$\begin{cases} \frac{dI}{dt} = (\beta_1 I + \beta_2 P)S + \varepsilon(\beta_1 I + \beta_2 P)V - (\theta + \mu + \delta + \varphi)I, \\ \frac{dP}{dt} = \theta I - \mu_p P. \end{cases} \quad (15)$$

Then,

$$\begin{aligned} \Rightarrow F &= \begin{pmatrix} \frac{df_1}{dI} & \frac{df_1}{dP} \\ \frac{df_2}{dI} & \frac{df_2}{dP} \end{pmatrix} = \begin{pmatrix} \beta_1(S + \varepsilon V) & \beta_2(S + \varepsilon V) \\ 0 & 0 \end{pmatrix}, \\ V &= \begin{pmatrix} \frac{dv_1}{dI} & \frac{dv_1}{dP} \\ \frac{dv_2}{dI} & \frac{dv_2}{dP} \end{pmatrix} = \begin{pmatrix} \theta + \mu + \delta + \varphi & 0 \\ -\theta & \mu_p \end{pmatrix}, \\ FV^{-1} &= \begin{pmatrix} \beta_1(S + \varepsilon V) & \beta_2(S + \varepsilon V) \\ 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{\theta + \mu + \delta + \varphi} & 0 \\ \frac{\theta}{\mu_p(\theta + \mu + \delta + \varphi)} & \frac{1}{\mu_p} \end{pmatrix} = \begin{pmatrix} \frac{b\beta_1(S + \varepsilon V) + \theta\beta_2(S + \varepsilon V)}{ab} & \frac{\beta_2(S + \varepsilon V)}{b} \\ 0 & 0 \end{pmatrix}, \end{aligned} \quad (16)$$

where  $a = \theta + \mu + \delta + \varphi$  and  $b = \mu_p$ .

Then, the effective reproduction number ( $R_{\text{Eff}}$ ) is obtained as follows:

$$R_{\text{Eff}} = \rho(FV^{-1}) = \left| \begin{array}{cc} \frac{b\beta_1(S + \varepsilon V) + \theta\beta_2(S + \varepsilon V)}{ab} - \lambda & \frac{\beta_2(S + \varepsilon V)}{b} \\ 0 & -\lambda \end{array} \right| = 0. \quad (17)$$

Thus, the mathematical definition of effective reproduction number  $\lambda_2$  is the largest eigenvalue of the matrix  $FV^{-1}$ . Therefore,

$$R_{\text{Eff}} = \frac{\pi[(\mu + \gamma)(1 + \alpha - q) + \varepsilon(\eta + q\mu)][\beta_1\mu_p + \theta\beta_2]}{\mu_p[(\mu + \eta)(\mu + \gamma) - \eta\alpha][(\theta + \mu + \delta + \varphi)]}. \quad (18)$$

### 3.5. Local Stability of Disease-Free Equilibrium (DFEP)

**Theorem 3.** *The disease-free equilibrium point DFEP  $E^* = (S^*, V^*, 0, R^*, 0)$  of system (1) is locally asymptotically stable if  $R_{\text{Eff}} < 1$  and unstable if  $R_{\text{Eff}} > 1$ .*

*Proof.* We linearized system (1), to get the Jacobian matrix:

$$J = \begin{pmatrix} -(\beta_1 I + \beta_1 P) - (\mu + \eta) & \alpha & -\beta_1 S \\ \eta & -\varepsilon(\beta_1 I + \beta_2 P) - (\mu + \alpha + \gamma) & -\varepsilon\beta_1 V \\ \beta_1 I + \beta_2 P & \varepsilon(\beta_1 I + \beta_2 P) & \beta_1 S + \varepsilon\beta_1 V - (\theta + \mu + \delta + \varphi) \\ 0 & \gamma & \varphi \\ 0 & 0 & \theta \\ 0 & -\beta_2 S \\ 0 & -\varepsilon\beta_2 V \\ 0 & \beta_2 (S + \varepsilon V) \\ -\mu & 0 \\ 0 & -\mu_p \end{pmatrix}. \quad (19)$$

After substituting the disease-free equilibrium point  $E^*$  in to the Jacobian matrix we obtained the following equation:

$$J(E^*) = \begin{pmatrix} -(\mu + \eta) & \alpha & -\beta_1 S^* \\ \eta & -(\mu + \alpha + \gamma) & -\varepsilon\beta_1 V^* \\ 0 & 0 & \beta_1 S^* + \varepsilon\beta_1 V^* - (\theta + \mu + \delta + \varphi) \\ 0 & \gamma & \varphi \\ 0 & 0 & \theta \\ 0 & -\beta_2 S^* \\ 0 & -\varepsilon\beta_2 V^* \\ 0 & \beta_2 (S^* + \varepsilon V^*) \\ -\mu & 0 \\ 0 & -\mu_p \end{pmatrix}. \quad (20)$$

Now, let

$$u = \mu + \eta v = \mu + \alpha + \gamma w = \beta_1 S^* + \varepsilon\beta_1 V^* - (\theta + \mu + \delta + \varphi). \quad (21)$$

$$x = \mu_p \text{ and } y = \beta_2 (S^* + \varepsilon V)$$

Then, the eigenvalues of  $|J(E^*) - \lambda I| = 0$  are determined as follows:

$$\begin{vmatrix} -u-\lambda & \alpha & -\beta_1 S^* & 0 & -\beta_2 S^* \\ \eta & -v-\lambda & -\varepsilon\beta_1 V^* & 0 & -\varepsilon\beta_2 V^* \\ 0 & 0 & w-\lambda & 0 & y \\ 0 & \gamma & \varphi & -\mu-\lambda & 0 \\ 0 & 0 & \theta & 0 & -x-\lambda \end{vmatrix} = 0. \quad (22)$$

Using cofactor expansion along the first column, we have the following equation:

$$\begin{aligned} & (-u-\lambda) \begin{vmatrix} -v-\lambda & -\varepsilon\beta_1 V^* & 0 & -\varepsilon\beta_2 V^* \\ 0 & w-\lambda & 0 & y \\ \gamma & \varphi & -\mu-\lambda & 0 \\ 0 & \theta & 0 & x-\lambda \end{vmatrix} - \eta \begin{vmatrix} \alpha & -\beta_1 S^* & 0 & -\beta_2 S^* + \omega \\ 0 & w-\lambda & 0 & y \\ \gamma & \varphi & -\mu-\lambda & 0 \\ 0 & \theta & 0 & -x-\lambda \end{vmatrix} \\ & = 0 = -(u+\lambda)(-v+\lambda) \begin{vmatrix} w-\lambda & 0 & y \\ \varphi & -\mu-\lambda & 0 \\ \theta & 0 & -x-\lambda \end{vmatrix} - \eta \left( \alpha \begin{vmatrix} w-\lambda & 0 & y \\ \varphi & -\mu-\lambda & 0 \\ \theta & 0 & -x-\lambda \end{vmatrix} + \gamma \begin{vmatrix} -\beta_1 S^* & 0 & -\beta_2 S^* + \omega \\ w-\lambda & 0 & y \\ \theta & 0 & -x-\lambda \end{vmatrix} \right) \\ & = 0 = (u+\lambda)(v+\lambda) \left[ (w-\lambda) \begin{vmatrix} -\mu-\lambda & 0 \\ 0 & -x-\lambda \end{vmatrix} - \varphi \begin{vmatrix} 0 & y \\ 0 & -x-\lambda \end{vmatrix} + \theta \begin{vmatrix} 0 & y \\ -\mu-\lambda & 0 \end{vmatrix} \right] \\ & \quad - \eta \left[ \alpha \left( (w-\lambda) \begin{vmatrix} -\mu-\lambda & 0 \\ 0 & -x-\lambda \end{vmatrix} - \varphi \begin{vmatrix} 0 & y \\ 0 & -x-\lambda \end{vmatrix} + \theta \begin{vmatrix} 0 & y \\ -\mu-\lambda & 0 \end{vmatrix} \right) \right. \\ & \quad \left. + \gamma \left( -\beta_1 S^* \begin{vmatrix} 0 & z \\ 0 & -x-\lambda \end{vmatrix} - (w-\lambda) \begin{vmatrix} 0 & -\beta_2 S^* + \omega \\ 0 & y-\lambda \end{vmatrix} + \theta \begin{vmatrix} 0 & -\beta_2 S^* + \omega \\ 0 & y \end{vmatrix} \right) \right] \\ & \quad - \eta \left[ \alpha \left( (w-\lambda) \begin{vmatrix} -\mu-\lambda & 0 \\ 0 & -x-\lambda \end{vmatrix} - \varphi \begin{vmatrix} 0 & y \\ 0 & -x-\lambda \end{vmatrix} + \theta \begin{vmatrix} 0 & y \\ -\mu-\lambda & 0 \end{vmatrix} \right) \right. \\ & \quad \left. + \gamma \left( -\beta_1 S^* \begin{vmatrix} 0 & z \\ 0 & -x-\lambda \end{vmatrix} - (w-\lambda) \begin{vmatrix} 0 & -\beta_2 S^* + \omega \\ 0 & y-\lambda \end{vmatrix} + \theta \begin{vmatrix} 0 & -\beta_2 S^* + \omega \\ 0 & y \end{vmatrix} \right) \right] \\ & = 0 = (u+\lambda)(v+\lambda)(-\mu-\lambda)[(w-\lambda)(-x-\lambda) - \theta y] - \eta[\alpha((w-\lambda)(-\mu-\lambda)(-x-\lambda) - \theta y(-\mu-\lambda))] \\ & = 0 = [(w-\lambda)(y-\lambda) - \theta y][(u+\lambda)(v+\lambda)(-\mu-\lambda) - \alpha\eta(-\mu-\lambda)] \\ & = 0 = (-\mu-\lambda)[(w-\lambda)(-x-\lambda) - \theta y][(u+\lambda)(v+\lambda) - \alpha\eta] \\ & = 0(-\mu-\lambda) = 0 \text{ or } (w-\lambda)(-x-\lambda) - \theta y = 0 \text{ or } (u+\lambda)(v+\lambda) - \alpha\eta = 0. \end{aligned} \quad (23)$$

From  $(-\mu-\lambda) = 0$ , we have  $\lambda_1 = -\mu < 0$ .

From equation  $(u+\lambda)(v+\lambda) - \alpha\eta = 0$ , we have obtained a quadratic equation with positive coefficient as follows:

$$\lambda^2 + a_1\lambda + a_2 = 0, \quad (24)$$

where  $a_1 = u + v = (\mu + \eta) + (\mu + \alpha + \gamma) > 0$  and  $a_2 = uv - \alpha\eta = (\mu + \eta)(\mu + \alpha + \gamma) > 0$

Thus, by Routh-Hurwitz stability criteria, the characteristic polynomial has negative eigenvalues. Hence,  $\lambda_2 < 0$  and  $\lambda_3 < 0$ , so they are stable.

In the last, from the second quadratic equation  $(w - \lambda)(-x - \lambda) - \theta y = 0$ , we have the following equation:

$$\lambda^2 + (x - w)\lambda - (wx + \theta y) = 0,$$

$$\begin{aligned} \lambda &= \frac{-(x - w) \pm \sqrt{(x - w)^2 + 4(wx + \theta y)}}{2}, \\ \lambda_4 &= \frac{-(x - w) - \sqrt{(x - w)^2 + 4(wx + \theta y)}}{2}, \\ &= -\frac{(x - w) + \sqrt{(x - w)^2 + 4(wx + \theta y)}}{2} < 0, \end{aligned} \quad (25)$$

this also satisfies for stability at the equilibrium point.

Now,

$$\lambda_5 = \frac{-(x - w) + \sqrt{(x - w)^2 + 4(wx + \theta y)}}{2}, \quad (26)$$

to be stable at the equilibrium point,  $wx + \theta y$  must be less than zero.

$$wx + \theta y < 0 \Rightarrow wx < -\theta y,$$

$$[\beta^1(S + \varepsilon V) - (\theta + \mu + \delta + \varphi)]\mu_p < -\theta\beta_2(S + \varepsilon V),$$

$$\beta_1(S + \varepsilon V)\mu_p - (\theta + \mu + \delta + \varphi) < \mu_p - \theta\beta_2(S + \varepsilon V),$$

$$\beta_1(S + \varepsilon V)\mu_p + \theta\beta_2(S + \varepsilon V) < (\theta + \mu + \delta + \varphi)\mu_p,$$

$$(S + \varepsilon V)(\beta_1\mu_p + \theta\beta_2) < (\theta + \mu + \delta + \varphi)\mu_p,$$

$$\frac{(S + \varepsilon V)(\beta_1\mu_p + \theta\beta_2)}{(\theta + \mu + \delta + \varphi)\mu_p} < \frac{(\theta + \mu + \delta + \varphi)(\omega + \mu_p)}{(\theta + \mu + \delta + \varphi)(\omega + \mu_p)}, \quad (27)$$

$$\frac{(S + \varepsilon V)(\beta_1\mu_p + \theta\beta_2)}{(\theta + \mu + \delta + \varphi)(\omega + \mu_p)} < 1,$$

$$(S + \varepsilon V) \frac{(\beta_1\mu_p + \theta\beta_2)}{\mu_p(\theta + \mu + \delta + \varphi)} < 1,$$

$$R_{\text{Eff}} < 1.$$

Thus, our finding shows that if  $R_{\text{Eff}} < 1$ , the disease-free equilibrium point is locally asymptotically stable.

**3.6. Global Stability of DFEP.** So to justify the DFEP is globally asymptotically stable, we use the Castillo--Chavez and Song [22] and Alemneh and Alemu [23] criterion. Based on the theorem, to investigate the global stability, we must rewrite the model in equation (1) as follows:

$$\begin{cases} \frac{dX}{dt} = F(X, Z), \\ \frac{dZ}{dt} = G(X, Z), G(X, 0) = 0, \end{cases} \quad (28)$$

where  $X \in R^3$  represents the number of uninfected population while  $Z \in R^2$  represents the number of infected population. For the disease-free equilibrium point to be globally stable, it should meet the following two axioms:

The following two conditions should be satisfied:

$A_1$   $dX/dt = F(X^*, 0)$ , where  $X^*$  is globally asymptotically stable.

$A_2$   $dZ/dt = \partial G/\partial Z(X^*, 0)Z - \hat{G}(X, Z), \hat{G}(X, Z) \geq 0$  for all  $(X, Z) \in \Omega$ .

**Theorem 4.** The disease-free equilibrium point  $E^0 = (X^*, 0)$  of the system in equation (1) is globally asymptotically stable if it satisfies the three conditions of Castillo--Chavez criterion.



*Proof.* To prove the disease-free equilibrium point is globally asymptotically stable, first of all we should identify  $F(X, Z)$  and  $G(X, Z)$ .

From our model,

$$F(X, Z) = \begin{cases} (1-q)\pi + \alpha V - (\mu + \eta)S, \\ q\pi + \eta S - (\mu + \gamma)V, \\ \gamma V - \mu R, \end{cases} \quad (29)$$

$$G(X, Z) = \begin{cases} (\beta_1 I + \beta_2 P)S + \varepsilon \beta_1 I + \beta_2 PV - (\theta + \mu + \delta + \varphi)I, \\ \theta I - (\omega + \mu_p)P. \end{cases} \quad (30)$$

The first condition is already proved in Theorem 3.

Now,

$$X^* = \left( \frac{\pi(\mu + \gamma)(1 + \alpha - q)}{(\mu + \eta)(\mu + \gamma) + \mu\alpha}, \frac{\pi(\eta + \mu q)}{(\mu + \eta)(\mu + \gamma) + \mu\alpha}, \frac{\pi\gamma(\eta + \mu q)}{\mu(\mu + \eta)(\mu + \gamma) + \mu^2\alpha} \right), \quad (31)$$

is satisfying the second condition of Castillo-Chavez criterion for the reduced system  $dX/dt = F(X^*, 0)$ . From the first equation in system (29), we have the following equation:

$$\frac{dS}{dt} = (1-q)\pi + \alpha V - (\mu + \eta)S. \quad (32)$$

Integrating both sides,

$$S = \frac{e^{-(\mu+\eta)(t+c)} - \pi(1-q) - \alpha V_0}{-(\mu + \eta)} = \frac{e^{-(\mu+\eta)(t+c)}}{-(\mu + \eta)} + \frac{\pi(1-q) + \alpha V_0}{\mu + \eta} \text{ Since as } t \rightarrow \infty, \quad (33)$$

$$e^{-(\mu+\eta)(t+c)} \rightarrow 0 \text{ and } S \rightarrow \frac{\pi(1-q) + \alpha V_0}{\mu + \eta} = \frac{\pi(\mu + \gamma)(1 + \alpha - q)}{(\mu + \eta)(\mu + \gamma) + \mu\alpha} = S_0.$$

From the second equation in system (29), we have the following equation:

$$\frac{dV}{dt} = q\pi + \eta S_0 - (\mu + \alpha + \gamma)V. \quad (34)$$

Integrating both sides by considering  $S = S_0$  using the method of separable variable,

$$V = \frac{e^{-(\mu+\alpha+\gamma)(t+c)}}{-(\mu + \alpha + \gamma)} + \frac{\pi(1-q) + \eta S_0}{\mu + \alpha + \gamma}. \quad (35)$$

Since  $t \rightarrow \infty$ ,  $e^{-(\mu+\alpha+\gamma)(t+c)} \rightarrow 0$  and

$$V \rightarrow \frac{\pi(1-q) + \eta S_0}{\mu + \alpha + \gamma} = \frac{\pi(\eta + \mu q)}{(\mu + \eta)(\mu + \gamma) + \mu\alpha} = V_0. \quad (36)$$

Using the same fashion, we have proved  $R$  by taking the third equation of system (29).

$$\frac{dR}{dt} = \gamma V - \mu R \Rightarrow \frac{dR}{dt} = \gamma V_0 - \mu R \Rightarrow \frac{e^{-\mu(t+c)}}{-\mu} + \frac{\gamma V_0}{\mu}. \quad (37)$$

Since  $t \rightarrow \infty$  then  $e^{-\mu(t+c)} \rightarrow 0$  and

$$R \rightarrow \frac{\gamma V_0}{\mu} = \frac{\pi\gamma(\eta + \mu q)}{\mu(\mu + \eta)(\mu + \gamma) + \mu^2\alpha} = R_0. \quad (38)$$

Thus, the second condition of Castillo-Chavez criterion holds true. Lastly, we must show the third criterion as follows:

$$\text{i.e. } \frac{\partial G}{\partial Z}(X^*, 0) \text{ is an } M\text{-matrix and } \hat{G}(X, Z) \geq 0 \text{ for all } (X, Y) \in \Omega. \quad (39)$$

Now,

$$\frac{\partial G}{\partial Z}(X^*, 0) = \begin{pmatrix} \frac{\partial G_1}{\partial I} & \frac{\partial G_1}{\partial P} \\ \frac{\partial G_2}{\partial I} & \frac{\partial G_2}{\partial P} \end{pmatrix}, \quad (40)$$

where

$$\begin{aligned}
G_1(X, 0) &= (\beta_1 I + \beta_2 P)S + \varepsilon(\beta_1 I + \beta_2 P)V, -(\theta + \mu + \delta + \varphi)I, \\
G_1(X, 0) &= \theta I - \mu_p P, \\
\frac{\partial G}{\partial Z}(X^*, 0) &= \begin{pmatrix} \frac{\partial G_1}{\partial I} & \frac{\partial G_1}{\partial P} \\ \frac{\partial G_2}{\partial I} & \frac{\partial G_2}{\partial P} \end{pmatrix} = \begin{pmatrix} (\beta_1 S + \varepsilon V) - (\theta + \mu + \delta + \varphi) & \beta_2(S + \varepsilon V) \\ \theta & -\mu_p \end{pmatrix},
\end{aligned} \tag{41}$$

Since the nondiagonal entries  $\theta$  and  $\beta_1(S + \varepsilon V)$  of  $\partial G/\partial Z(X^*, 0)$  are non-negative. Thus,  $\partial G/\partial Z(X^*, 0)$  is an  $M$ -matrix.

From

$$\frac{dZ}{dt} = \frac{\partial G}{\partial Z}(X^*, 0)Z - \hat{G}(X, Z), \tag{42}$$

we have obtained the following equation:

$$\begin{aligned}
\hat{G}(X, Z) &= \frac{\partial G}{\partial Z}(X^*, 0)Z - \frac{dZ}{dt} = \begin{pmatrix} \beta_1 I S_0 + (\varepsilon V_0 - (S + \varepsilon V)) + \beta_2 P S_0 + (\varepsilon V_0 - (S + \varepsilon V)) \\ 0 \end{pmatrix} \\
&= \begin{pmatrix} \beta_1 I + \beta_2 P(S_0 - S) + \varepsilon(V_0 - V) \\ 0 \end{pmatrix}.
\end{aligned} \tag{43}$$

Since  $S_0 \geq S$  and  $V_0 \geq V$ , it is clear that  $\hat{G}(X, Z) \geq 0$  for all  $(X, Z) \in \Omega$ . Thus, based on the above shown procedures, the theorem is satisfying the three conditions of Castillo–Chavez criterion. Therefore, the DFEP is globally asymptotically stable and the disease is die out plus the transmission dynamics is decreased and eradicated though time.

**3.7. Endemic Equilibrium Point (EEP).** The endemic equilibrium point (EEP) is calculated by considering all the state variables (i.e., susceptible, vaccinated, infected, recovered, and the pathogen classes) must not be zero at the equilibrium state, which means  $EEP = (S^*, V^*, I^*, R^*, P^*) \neq (0, 0, 0, 0, 0)$  and equating all equations in the system of the model to be zero.

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = \frac{dP}{dt} = 0. \tag{44}$$

From system (1) we have the following equation:

$$\begin{aligned}
\psi I^2 - \lambda I - \tau &= 0, \lambda = ef + \varepsilon bd + \frac{e(\pi(1-q)_{-ch})}{b}, \\
\tau &= d(\varepsilon h + \alpha) + \frac{f(\pi(1-q)_{-ch})}{b} \implies I \\
&= \frac{\lambda + \sqrt{\lambda^2 - 4\kappa\tau}}{2\kappa}.
\end{aligned} \tag{45}$$

Then, using back substitution, we have obtained the value of  $V$ ,  $S$ ,  $R$ , and  $P$  as follows:

Thus, the endemic equilibrium points (EEP) is as follows:

$$\begin{aligned}
S^* &= \frac{c}{b} - \frac{2d\epsilon\kappa}{e\left(\lambda + \sqrt{\lambda^2 - 4\kappa\tau}\right) - 2\kappa f}, \\
V^* &= \frac{2d\kappa}{e\left(\lambda + \sqrt{\lambda^2 - 4\kappa\tau}\right) - 2\kappa f}, \\
R^* &= \frac{\varphi\left(\lambda + \sqrt{\lambda^2 - 4\kappa\tau}\right)}{2\mu\kappa} + \frac{2d\gamma\kappa}{\mu\left[e\left(\lambda + \sqrt{\lambda^2 - 4\kappa\tau}\right) - 2\kappa f\right]}, \\
P^* &= \frac{\theta\left(\lambda + \sqrt{\lambda^2 - 4\kappa\tau}\right)}{2\kappa\mu_p},
\end{aligned} \tag{46}$$

where  $b = \beta_1\mu_p + \beta_2\theta/\mu_p$ ,  $c = (\theta + \mu + \delta + \varphi)$ ,  $d = \pi qb + \eta_{ce} = \epsilon b^2$ ,  $f = b(\alpha + \gamma + \mu + \eta\epsilon)$ , and  $h = \mu + \eta s$ .

**3.8. Global Stability of EEP.** An equilibrium point is said to be globally stable, which means for any possible starting points the system will come to the equilibrium point.

**Theorem 5.** *If  $R_{\text{Eff}} > 1$ , then the endemic equilibrium point ( $E'$ ) is globally asymptotically stable; otherwise, it is unstable.*

*Proof.* To prove the global stability of the endemic equilibrium point, we use the Lyapunov function defined as follows:

$$\begin{aligned}
\mathcal{O}(E') &= \left(S - \ln \frac{S}{S'}\right) + \left(V - \ln \frac{V}{V'}\right) + \left(I - \ln \frac{I}{I'}\right) \\
&\quad + \left(R - \ln \frac{R}{R'}\right) + \left(P - \ln \frac{P}{P'}\right).
\end{aligned} \tag{47}$$

After integrating  $\mathcal{O}$  with respect to time  $t$ , we have the following equation:

$$\begin{aligned}
\frac{d\mathcal{O}}{dt} &= \frac{d\mathcal{O}}{dS} \frac{dS}{dt} + \frac{d\mathcal{O}}{dV} \frac{dV}{dt} + \frac{d\mathcal{O}}{dI} \frac{dI}{dt} + \frac{d\mathcal{O}}{dR} \frac{dR}{dt} + \frac{d\mathcal{O}}{dP} \frac{dP}{dt} \\
&= \left(1 - \frac{S'}{S}\right) \frac{dS}{dt} + \left(1 - \frac{V'}{V}\right) \frac{dV}{dt} + \left(1 - \frac{I'}{I}\right) \frac{dI}{dt} + \left(1 - \frac{R'}{R}\right) \frac{dR}{dt} + \left(1 - \frac{P'}{P}\right) \frac{dP}{dt}.
\end{aligned} \tag{48}$$

Substituting the values of the system of equations of the model and collecting similar terms, we obtained the reduced equation given by the following equation:

$$\begin{aligned}
\frac{d\emptyset}{dt} = & (1-q)\pi + q\pi - (\beta_1 I + \beta_2 P)_S + (\beta_1 I + \beta_2 P)_S - \varepsilon(\beta_1 I + \beta_2 P)_{V+} \\
& \cdot \varepsilon(\beta_1 I + \beta_2 P)_{V+} \eta S - (\mu + \eta)S - (\mu + \alpha + \gamma)V + \alpha V + \gamma V \\
& - (\theta + \mu + \delta + \varphi)I + \theta I + \varphi I + \frac{S'}{S} \pi (1-q) + (\beta_1 I + \beta_2 P)S' \\
& - \frac{S'}{S} \alpha V + (\mu + \eta)S' - \frac{S'}{S} \omega P + \frac{V'}{V} \pi q + \varepsilon(\beta_1 I + \beta_2 P)V' - \frac{V'}{V} \eta S \\
& + (\mu + \alpha + \gamma)V' - \frac{I'}{I} (\beta_1 I + \beta_2 P)_S - \frac{I'}{I} \varepsilon(\beta_1 I + \beta_2 P)_V + (\theta + \mu + \delta + \varphi)I' \\
& - \mu R - \frac{R'}{R} \varphi I - \frac{R'}{R} \gamma V + \mu R' - \mu_p P - \frac{P'}{P} \theta I + \mu_p P' = \pi - \mu S - \mu V \\
& - (\mu + \delta)I - \mu R - \mu_p P + \frac{S'}{S} \pi (1-q) + (\beta_1 I + \beta_2 P)S' - \frac{S'}{S} \alpha V + (\mu + \eta)S' \\
& - \frac{S'}{S} \omega P - \frac{V'}{V} \pi q + \varepsilon(\beta_1 I + \beta_2 P)V' - \frac{V'}{V} \eta S + (\mu + \alpha + \gamma)V' - \frac{I'}{I} (\beta_1 I + \beta_2 P)_S \\
& - \frac{I'}{I} \varepsilon(\beta_1 I + \beta_2 P)_V + (\theta + \mu + \delta + \varphi)I' - \frac{R'}{R} \varphi I - \frac{R'}{R} \gamma V + \mu R' - \mu_p P - \frac{P'}{P} \theta I + \mu_p P'
\end{aligned} \tag{49}$$

Collecting together the positives and the negatives, we have obtained the following equation:

$$\begin{aligned}
& = \pi + \frac{S'}{S} \pi + (\beta_1 I + \beta_2 P)S' + (\mu + \eta)S' + \varepsilon(\beta_1 I + \beta_2 P)V' + (\mu + \alpha + \gamma)V' + (\theta + \mu + \delta + \varphi)I' + \mu_p P' \\
& - \left[ \mu S + \mu V + (\mu + \delta)I + \mu R + \mu_p P + \frac{S'}{S} \pi q + \frac{S'}{S} \alpha V + \frac{S'}{S} \omega P + \frac{V'}{V} \pi q + \frac{V'}{V} \eta S + \frac{I'}{I} (\beta_1 I + \beta_2 P)_S \right. \\
& \left. + \frac{I'}{I} \varepsilon(\beta_1 I + \beta_2 P)_{V+} \frac{R'}{R} \varphi I + \frac{R'}{R} \gamma V + \mu_p P + \frac{P'}{P} \theta I \right] \Rightarrow \frac{d\emptyset}{dt} = U - V.
\end{aligned} \tag{50}$$

where

$$U = \pi + \frac{S'}{S} \pi + (\beta_1 I + \beta_2 P)S' + (\mu + \eta)S' + \varepsilon(\beta_1 I + \beta_2 P)V' + (\mu + \alpha + \gamma)V' + (\theta + \mu + \delta + \varphi)I' + \mu_p P' \tag{51}$$

and

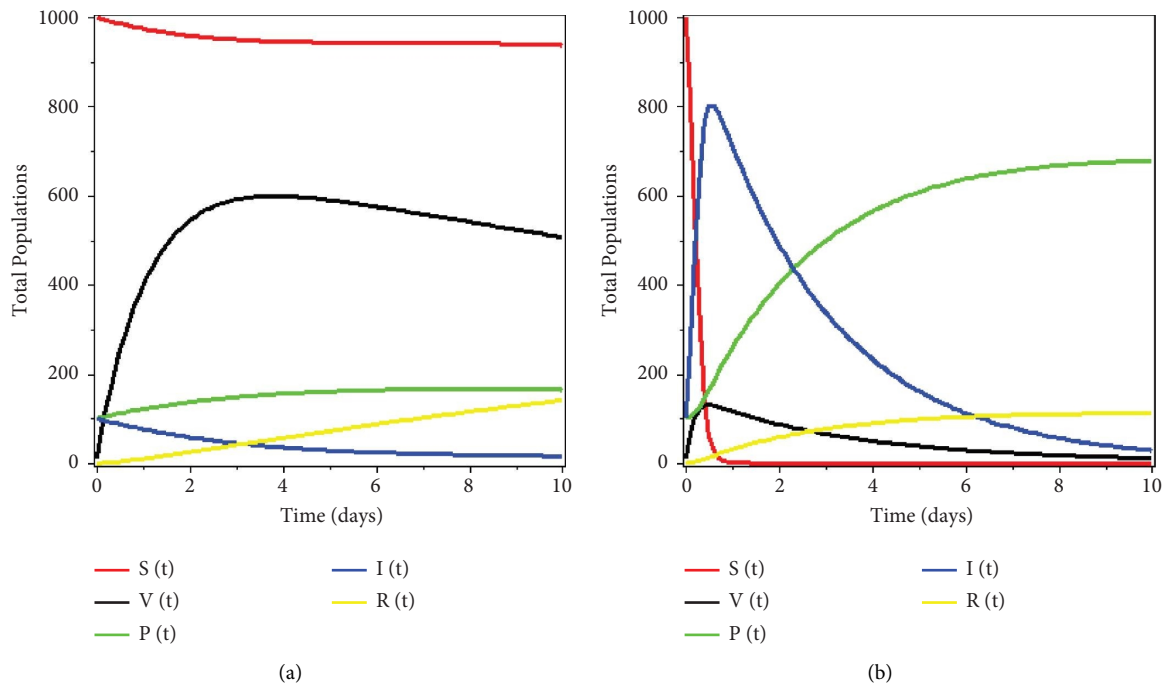
$$V = \left[ \mu S + \mu V + (\mu + \delta)I + \mu R + \mu_p P + \frac{S'}{S} \pi q + \frac{S'}{S} \alpha V + \frac{S'}{S} \omega P + \frac{V'}{V} \pi q + \frac{V'}{V} \eta S + \frac{I'}{I} (\beta_1 I + \beta_2 P)_S + \frac{I'}{I} \varepsilon(\beta_1 I + \beta_2 P)_{V+} \frac{R'}{R} \varphi I + \frac{R'}{R} \gamma V + \frac{P'}{P} \theta I \right]. \tag{52}$$

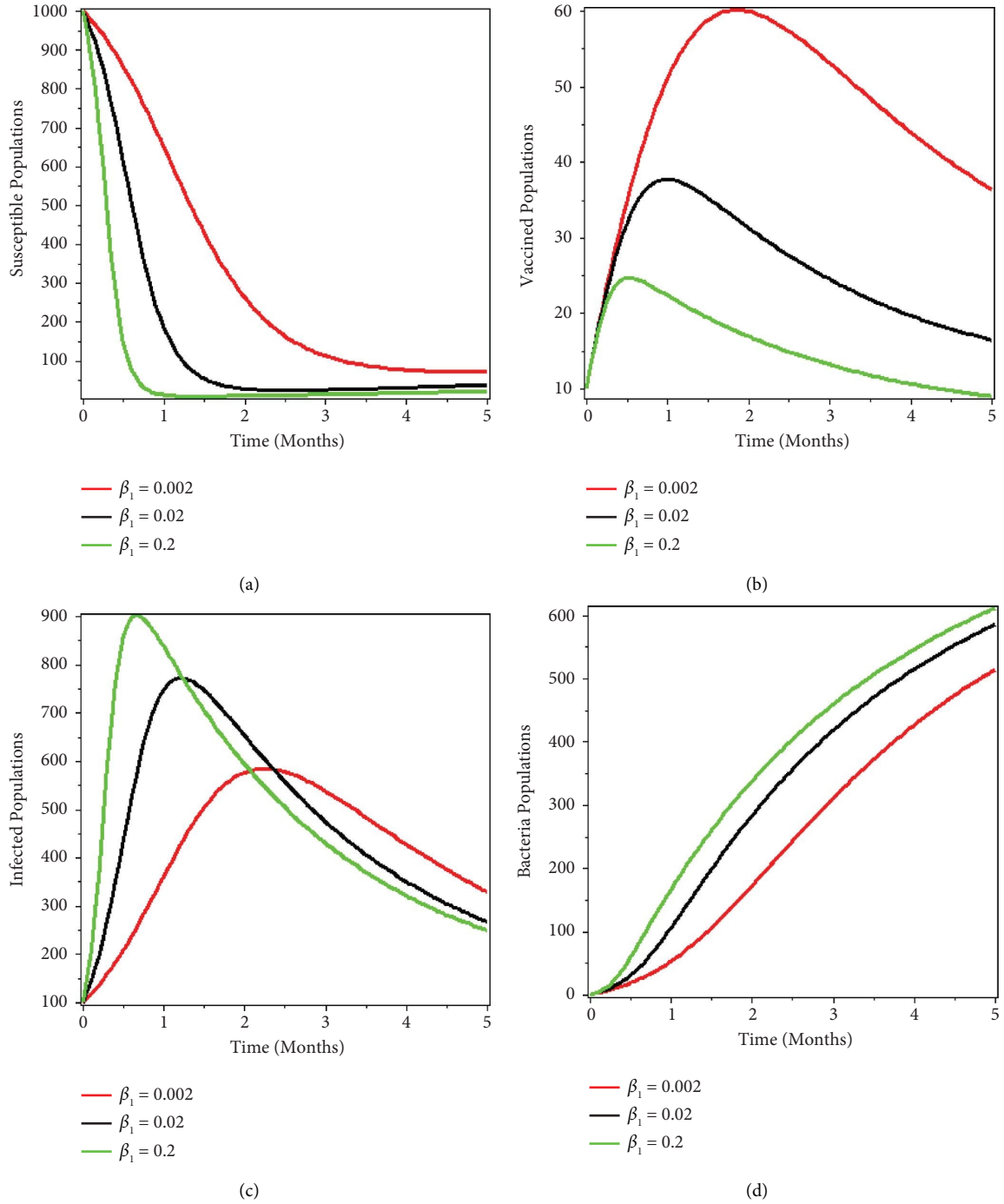
TABLE 3: Sensitivity indices of the parameters.

Parameters	Sensitivity value
$\beta_1$	+ve
$\beta_2$	+ve
$\mu$	-ve
$\eta$	-ve
$\alpha$	+ve
$\varepsilon$	+ve
$\gamma$	+ve
$\theta$	+ve
$\varphi$	-ve
$\delta$	-ve
$\mu_p$	-ve

TABLE 4: Parameter values and their sources.

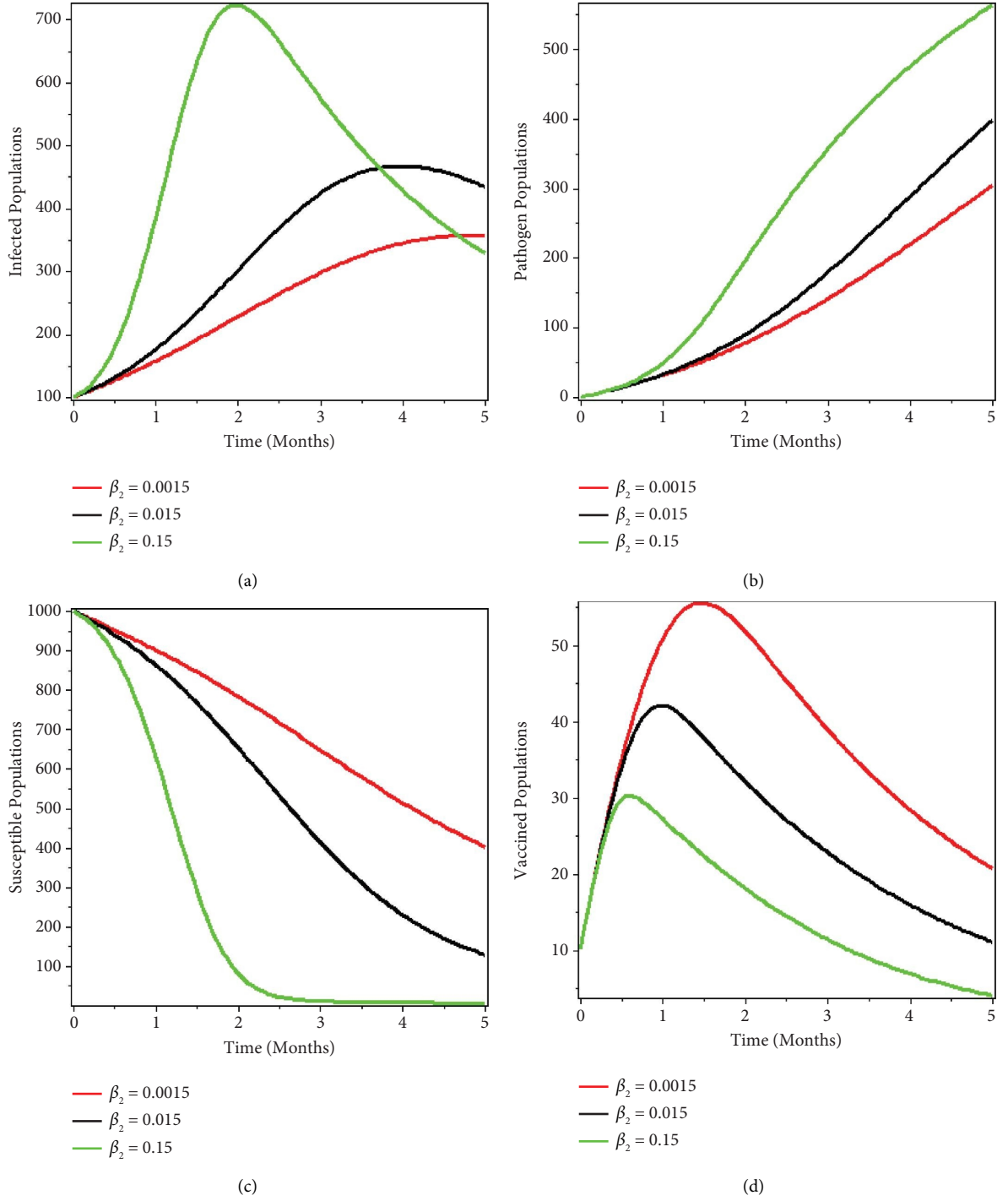
Parameter	Value	Source
$\pi$	100	Assumed
$\beta_1$	0.09091	Tilahun et al. [27]
$\beta_2$	0.052	Assumed
$\mu$	0.00875	Tilahun et al. [27]
$\eta$	0.6	Berhe and Makinde [15]
$\alpha$	0.167	Tilahun et al. [27]; Ethiopian Health and Nutrition Research Institute [28]
$\varepsilon$	0.0000003	Assumed
$\gamma$	0.8	Tilahun et al. [27]
$\theta$	0.04	Assumed
$\varphi$	0.14286	Tilahun et al. [27]; Ethiopian Health and Nutrition Research Institute [28]
$\delta$	0.125	Ethiopian Health and Nutrition Research Institute [28]
$\mu_p$	0.08	Assumed
$q$	0.037755	Ethiopian Health and Nutrition Research Institute [28]

FIGURE 2: Graphical representation of the total population when  $R_{\text{Eff}} < 1$  and when  $R_{\text{Eff}} > 1$ .

FIGURE 3: Effect of varying  $\beta_1$  on human population.

Hence, if  $U - V \leq 0$ , then  $d\mathcal{O}/dt \leq 0$  and  $d\mathcal{O}/dt = 0$  whenever  $S = S'$ ,  $V = V'$ ,  $I = I'$ ,  $R = R'$ , and  $P = P'$ . Therefore, the largest compact invariant set  $\Omega$ ,  $E'$  is the only set of the endemic equilibrium point. Thus, by LaSalle invariant principal  $E'$  is globally asymptotically stable on  $\Omega$  if  $U - V \leq 0$ .

**3.9. Sensitivity Analysis.** Sensitivity analysis is a tool of determining how different values of an independent variable affect a particular dependent variables under a given set of assumptions [24]. According to Alhamami [14] normalized sensitivity index is defined as follows: Let  $R$  be a variable that depends on a parameter  $p$ ; then the normalized sensitivity

FIGURE 4: Effect of varying  $\beta_2$  on human population.

index of variable  $R$  with respect to the parameter  $p$  given by the following equation:

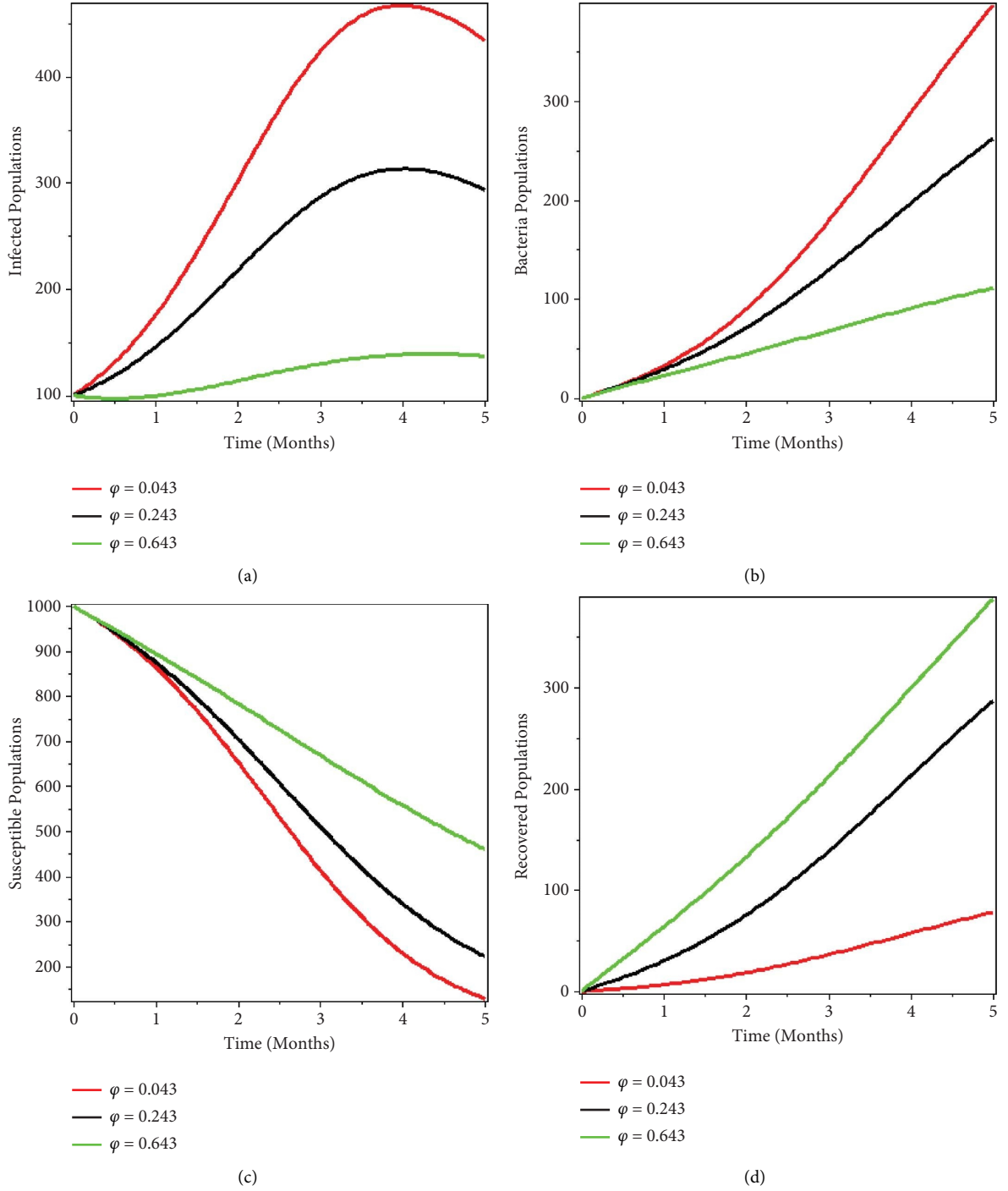
$$\Upsilon_p^R = \frac{\partial R}{\partial p} \times \frac{p}{R}. \quad (53)$$

Now, from our model the effective reproduction number is as follows:

$$R_{\text{Eff}} = (S + \epsilon V) \frac{(\beta_1(\omega + \mu_p) + \theta\beta_2)}{(\theta + \mu + \delta + \varphi)(\omega + \mu_p)}, \quad (54)$$

and the local sensitivity analysis of  $R_0$  with respect to each parameter is calculated as follows:

Sensitivity index of  $\beta_1$  is as follows:

FIGURE 5: Effect of varying  $\phi$  on human population.

$$\Upsilon_{\beta_1}^{R_{\text{Eff}}} = \frac{\partial R_{\text{Eff}}}{\partial \beta_1} \times \frac{\beta_1}{R_{\text{Eff}}} = \frac{\mu_p \beta_1}{\mu_p \beta_1 + \theta \beta_2} > 0. \quad (55)$$

Sensitivity index of  $\beta_2$  is as follows:

$$\Upsilon_{\beta_2}^{R_{\text{Eff}}} = \frac{\partial R_{\text{Eff}}}{\partial \beta_2} \times \frac{\beta_2}{R_{\text{Eff}}} = \frac{\theta \beta_2}{\mu_p \beta_1 + \theta \beta_2} > 0. \quad (56)$$

Sensitivity index of  $\theta$  is as follows:

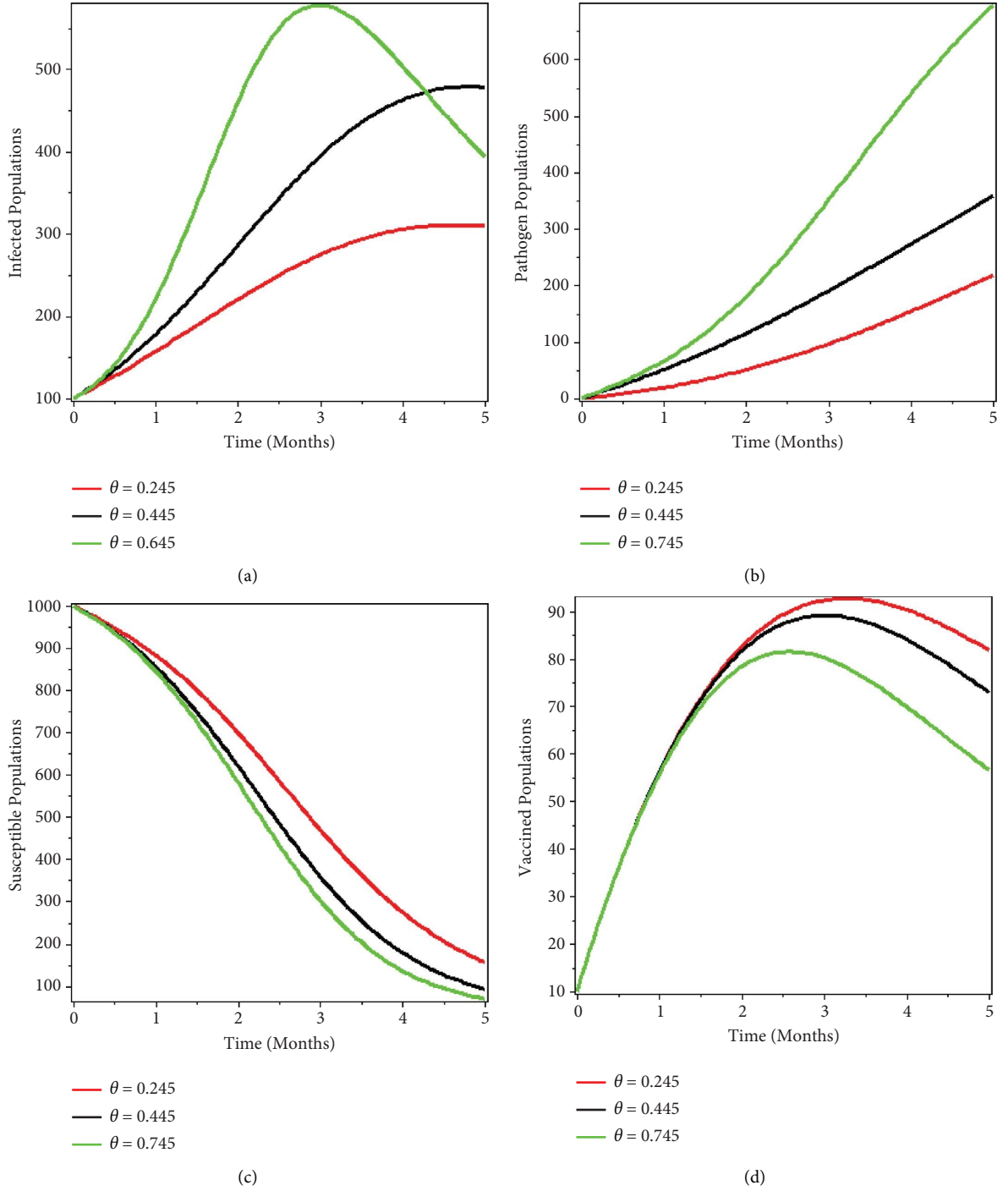
$$\Upsilon_{\theta}^{R_{\text{Eff}}} = \frac{\partial R_{\text{Eff}}}{\partial \theta} \times \frac{\theta}{R_{\text{Eff}}} = \frac{\theta [\beta_2 (\mu + \delta + \phi) - \beta_1 \mu_p]}{(\theta + \mu + \delta + \phi) (\beta_1 + \theta \beta_2)} > 0. \quad (57)$$

Sensitivity index of  $\delta$  is as follows:

$$\Upsilon_{\delta}^{R_{\text{Eff}}} = \frac{\partial R_{\text{Eff}}}{\partial \delta} \times \frac{\delta}{R_{\text{Eff}}} = \frac{-\delta}{(\theta + \mu + \delta + \phi)} < 0. \quad (58)$$

Sensitivity index of  $\phi$  is as follows:



FIGURE 6: Vaccinated population with different values of  $\theta$ .

$$\Upsilon_{\varphi}^{R_{\text{Eff}}} = \frac{\partial R_{\text{Eff}}}{\partial \varphi} \times \frac{\varphi}{R_{\text{Eff}}} = \frac{-\varphi}{(\theta + \mu + \delta + \varphi)} < 0. \quad (59)$$

Therefore, from the result, we generalized the sensitivity indices of the parameters in Table 3.

From Table 2, we conclude that the parameters  $\beta_1$ ,  $\beta_2$ ,  $\varepsilon$ , and  $\theta$  have a positive sensitivity indices. Thus, they have a great effect on the transmission dynamics and prevalence of measles. However,  $\mu_p$ ,  $\delta$ ,  $\eta$ , and  $\alpha$  have a negative sensitivity indices, and they have a high influence on controlling and

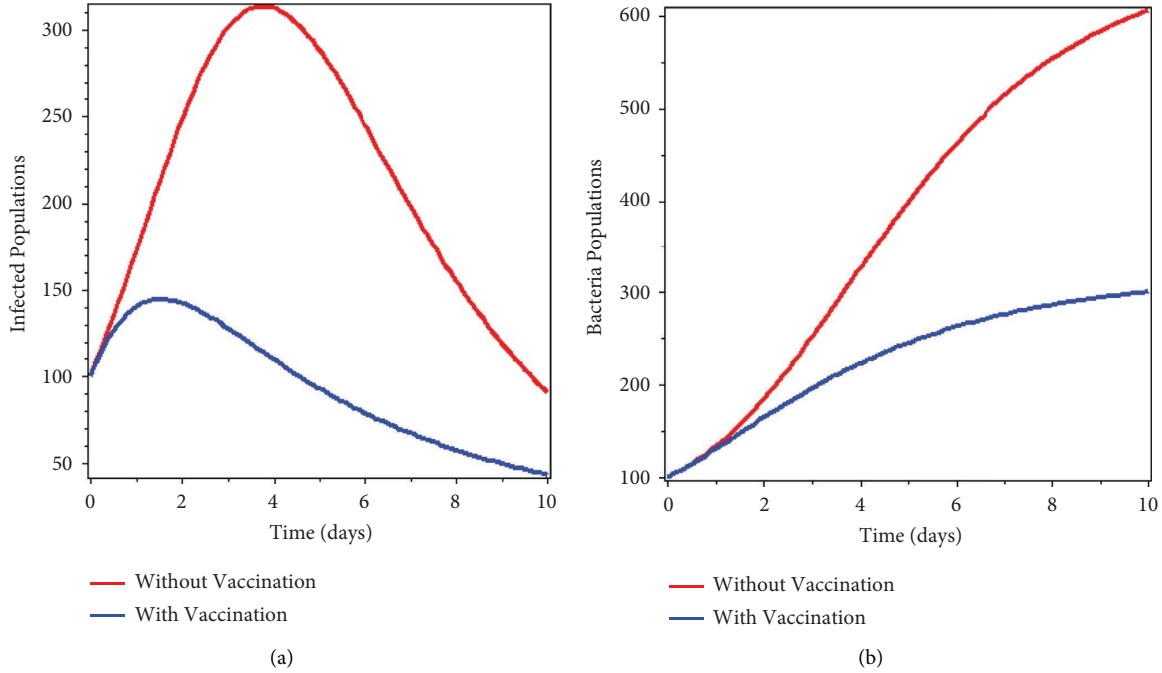


FIGURE 7: Effect of vaccination on the infected human population and bacteria population.

preventing transmission dynamics and prevalence of measles. Therefore, according to the results obtained on the sensitivity indices of the model parameters, we are assuming the values some of the parameters and also taken from related published articles.

#### 4. Numerical Simulation

In this section, we explore the behavior of measles what we have seen qualitatively in the previous chapter by conducting a numerical simulation and study the effects of some of the parameters and interpreting its effect graphically by considering different values. To conduct the simulation of the model, we used monthly time steps and solved it by a Maple 18 software with initial conditions  $S(t) = 1000$ ,  $V(t) = 10$ ,  $I(t) = 100$ ,  $R(t) = 0$ , and  $P(t) = 100$  and parameters in Table 4.

As we observe in Figure 2(a), if the disease is eradicated from the environment, there is no effect on the susceptible population, the vaccinated population, and the recovered population, and they are going to its steady state value (normal condition) while the number of infected population and the pathogen population is decreasing slow and approaching to zero. Thus, the transmission effect of measles has no impact when the reproduction number is much less than one on the human population due to a decrease in the number of infectious subpopulation..

In Figure 2(b), the graph shows the number of susceptible, vaccinated, and recovered population is decreasing in a fast rate; on the other hand, the number of infected population is increasing rapidly in a short period of time and reached at the highest point and then decreasing gradually. Additionally, the pathogen population is still increasing up due the persistent measles virus in the environment when the infected population sneezes, cough, and so on. So in the

endemic equilibrium point, the basic reproduction number is greater than one because of the rapid increment of infection and the pathogen population on the graph.

Figure 3 illustrates that when the transmission rate  $\beta_1$  increases, the infection of susceptible (Figure 3(a)), and vaccine population (Figure 3(b)) decreases, and this causes an increase in the number of infected population (Figure 3(c)), and this again increases bacteria population in the environment (Figure 3(d)).

In Figure 4, letting all the other parameters constant and changing the value of  $\beta_2$  in the human population. When we observe the graph in different values of the indirect contact rate ( $\beta_2$ ), the number of infected population and pathogen population increases as  $\beta_2$  increases as shown in Figures 4(a) and (b), respectively. Also the increase in the value of contact rate  $\beta_2$  has decreased the number of susceptible and vaccinated populations as shown in Figures 4(a) and 4(b), respectively. Therefore, it is important to work on prevention methods of indirect contact of susceptible individual to the disease to combat against the disease.

In Figure 5, we illustrate the effect of varying the value of the recovery rate of infected individuals  $\varphi$ , letting all the other parameters constant, in the human population. As it is observed in the figure, increasing the treatment rates of the infected individuals decreased the number of infected individuals as shown in Figure 5(a) in population which again decreases the bacteria population as put in the environment (Figure 5(b)). This recovery increment has also a positive effect in the susceptible in Figure 5(c) and recovered human population in Figure 5(d). Therefore, it is important to work on treatment of infected individuals to fight against the measles.

In Figure 6, we observed the effect of infected individuals to the population of pathogen in different value of  $\theta$ . As shown in Figure 6(a), the number of infected population

increases as  $\theta$  increases. Similarly as depicted in Figure 6(b), the pathogen population increased in number as infected individuals increase the concentration of bacteria in the environment. Figure 6(c) also shows a decrease in the number of susceptible human as the bacteria increase due to infected individuals. Furthermore, this increment of pathogen has a negative relation with a vaccinated human population as shown in Figure 6(d).

Figure 7 illustrates the effect of vaccination in the population. From Figure 7(a), the absence of vaccination in the population increases the susceptible individuals to join the infected population, which leads an increase in the infected subpopulation. The amount of diseased people drastically reduces if vaccination is made available to the general public. According to Figure 7(b), the shortage of a vaccination resulted in a rise in the number of bacterial infections among humans. If vaccination is made widely available, the number of bacteria that cause disease will substantially decrease. Therefore, stakeholders should work in increasing the vaccination rate of susceptible individuals to reduce the disease in the community.

## 5. Conclusion

In this study, we have formulated and analyzed a mathematical model of transmission dynamics of measles considering the indirect contact that causes infection on the susceptible individuals due to touching infected objects, inhaling polluted air, and other modes of transmissions of measles virus. To formulate the model, we first reviewed some of recent published papers and articles that worked on measles disease and modified the SEVIR model by SVIR considering a new compartment called pathogen population P that has a highest impact on the transmission of the disease. Then, we have analyzed the model by calculating the diseases free equilibrium point, the  $t$  endemic equilibrium point, and the reproduction number using the next-generation matrix. The stability of the equilibrium points are conducted in different ways. First of all, the local stability analysis of the DFEP is justified using linearization and its global stability is shown by Castillo–Chavez stability criterion. To perform the stability analysis of the EEP, we used Lyapunov function and show its global stability. The sensitivity of the parameters was computed using sensitivity index formula to identify the effects on the transmission dynamics of measles. Thus, the parameters  $\beta_1$ ,  $\beta_2$ ,  $\epsilon$ , and  $\theta$  have a positive sensitivity indices. They have a great effect on the transmission dynamics and prevalence of measles. However,  $\mu_p$ ,  $\delta$ ,  $\mu$ , and  $\alpha$  have a negative sensitivity indices and they have a positive influence on controlling and preventing transmission dynamics and prevalence of measles. The model analysis and the behavior of the parameters sensitivity analysis performed using numerical simulation are strengthened. So to study the sensitivity, Maple 18 is used which gives the same qualitative result to the sensitivity index. So the simulation graphs indicated that the parameters  $\beta_1$ ,  $\beta_2$ ,  $\epsilon$ , and  $\theta$  have the highest impact for maximizing the

transmission dynamics of the measles. From numerical simulation, we obtain that the indirect contact rate ( $\beta_2$ ) has an extreme impact on the transmission dynamics of measles.

Therefore, we conclude that the rate of indirect contact must be minimized to reduce the number of infected individuals. Also, we found that working on reducing the infection rates ( $\beta_1$  and  $\beta_2$ ) and infective contribution in the pathogen concentration in the environment ( $\theta$ ). In addition, enhancing the treatment rate of infective ( $\varphi$ ) and vaccination ( $\eta$ ) brings a significant contribution in reducing the disease effect in the community.

## Data Availability

The data supporting to this model are from previous published articles and they have been duly cited in this paper.

## Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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