

# FUDMA Journal of Sciences (FJS) ISSN online: 2616-1370 ISSN print: 2645 - 2944

Vol. 8 No. 1, February, 2024, pp 369 - 379



DOI: https://doi.org/10.33003/fjs-2024-0801-2219

# LOCAL AND GLOBAL STABILITY ANALYSIS OF MEASLES EPIDEMIC MODEL AT DISEASE-FREE EQUILIBRIUM

\*1Ochi, P.O., <sup>2</sup>Agada, Andrew A., <sup>3</sup>Nworah, Ifeoma B., <sup>4</sup>Nworah, Damascus Arinze and <sup>5</sup>Goni, A. N.

<sup>1</sup>Department of Mathematics, American University of Nigeria Academy, Yola

<sup>2</sup>Skyline University, Nigeria

<sup>3</sup>Federal Institute of Industrial Research (FIIRO), North-East centre, Yola, Adamawa State.

<sup>4</sup>Bioresources Development Centre, National Biotechnology Development Agency, Jalingo, Taraba State, Nigeria.

<sup>5</sup>Bethel Baptist High School, Kaduna, Nigeria

\*Corresponding authors' email: : <a href="mailto:onyema141@gmail.com">onyema141@gmail.com</a>

# ABSTRACT

In this study, a continuous mathematical model for the dynamics of Measles (rubeola) outbreak at constant recruitment rate  $\pi$  was formulated. In the model, we partitioned the population into Susceptible (S), Vaccinated (V), exposed (E), Infected (I) and recovered (R) individuals. We analyzed a SVEIR compartmental nonlinear deterministic mathematical model of measles epidemic in a community with constant population. Analytical studies were carried out on the model using the method of linearized stability. The basic reproductive number  $R_0$  that governs the disease transmission is obtained from the largest eigenvalue of the next-generation matrix. The disease-free equilibrium is computed and proved to be locally and globally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$  respectively. Finally, we simulate the model system in MATLAB and obtained the graphical behavior of each compartment. From the simulation, we observed that the measles infection was eradicated in the environment when  $R_0 < 1$ .

Keywords: Measles, SVEIR Model, Basic reproduction number, Local stability, Numerical simulation

#### INTRODUCTION

Measles are highly contagious infectious disease caused by measles virus (Guerra et al., 2017; Caserta, 2013; DHS, 2015). Symptoms usually develop 10-12 days after exposure to an infected person and last 7-10 days (Bope et al., 2014; WHO, 2014). Initial symptoms typically include fever, often greater than 40 °C (104 °F), cough, runny nose, and inflamed eyes (Caserta, 2013; CDC, 2014). Small white spots known as Koplik's spots may form inside the mouth two or three days after the start of symptoms (CDC, 2014). A red, flat rash which usually starts on the face and then spreads to the rest of the body typically begins three to five days after the start of 2014). Common (CDC, complications include diarrhea (in 8% of cases), middle ear infection (7%) and pneumonia (6%) (Atkinson, 2011). These occur in part due to measles-induced immuno-suppression (Rota et al., 2016). Less commonly seizures, blindness, or inflammation of the brain may occur (Atkinson, 2011; WHO, 2014). Other names include morbilli, rubeola, red measles, and English measles (Milner, 2015; Stanley, 2002). Both rubella, also known as German measles, and roseola are different diseases caused by unrelated viruses (Marx, 2010).

Measles is an airborne disease which spreads easily from one person to the next through the coughs and sneezes of infected people (WHO, 2014). It may also be spread through direct contact with mouth or nasal secretions (WHO, 2019). It is extremely contagious: nine out of ten people who are not immune and share living space with an infected person will be infected (Atkinson, 2011). Furthermore, measles's reproductive number estimates vary beyond the frequently cited range of 12 to 18 (Guerra et al., 2017). People are infectious to others from four days before to four days after the start of the rash (Atkinson, 2011). While often regarded as a childhood illness, it can affect people of any age (Chen, 2018). Most people do not get the disease more than once (WHO, 2014). Testing for the measles virus in suspected cases is important for public health efforts (Atkinson,

2011). Measles is not known to occur in other animals (WHO, 2019). Once a person has become infected, no specific treatment is available, (WHO, 2019) although supportive care may improve outcomes (WHO, 2014). Such care may include oral rehydration solution (slightly sweet and salty fluids), healthy food, and medications to control the fever (Bope et al., 2014; WHO, 2014). Antibiotics should be prescribed if secondary bacterial infections such as ear infections or pneumonia occur (WHO, 2014; WHO, 2019). Vitamin A supplementation is also recommended for children (WHO, 2019). Among cases reported in the U.S. between 1985 and 1992, death occurred in only 0.2% of cases, (Atkinson, 2011) but may be up to 10% in people with malnutrition (WHO, 2014). Most of those who die from the infection are less than five years old (WHO, 2019). The measles vaccine is effective at preventing the disease, is exceptionally safe, and is often delivered in combination with other vaccines (WHO, 2014; Russell et al., 2019). Vaccination resulted in an 80% decrease in deaths from measles between 2000 and 2017, with about 85% of children worldwide having received their first dose as of 2017 (WHO, 2019). Measles affects about 20 million people a year, (Caserta, 2013) primarily in the developing areas of Africa and Asia (WHO, 2014). It is one of the leading vaccinepreventable disease causes of death (Kabra & Lodha, 2013; WHO, 2019). In 1980, 2.6 million people died from measles, (WHO, 2014) and in 1990, 545,000 died due to the disease; by 2014, global vaccination programs had reduced the number of deaths from measles to 73,000 (GBD, 2013; GBD, 2015). Despite these trends, rates of disease and deaths increased from 2017 to 2019 due to a decrease in immunization (WHO, 2018; WHO, 2019; CDC, 2019). Measles can be prevented with measles-containing vaccine,

Measles can be prevented with measles-containing vaccine, which is primarily administered as the combination measles-mumps-rubella (MMR) vaccine. The combination measles-mumps-rubella-varicella (MMRV) vaccine can be used for children aged 12 months through 12 years for protection

against measles, mumps, rubella and varicella. Single-antigen measles vaccine is not available.

One dose of MMR vaccine is approximately 93% effective at preventing measles; two doses are approximately 97% effective. Almost everyone who does not respond to the measles component of the first dose of MMR vaccine at age 12 months or older will respond to the second dose.

Therefore, the second dose of MMR is administered to address primary vaccine failure (CDC, 2013). A vaccine failure is when an organism contracts a disease in spite of being vaccinated against it. Primary vaccine failure occurs when an organism's immune system does not produce antibodies when first vaccinated. Vaccines can fail when several series are given and fail to produce an immune response

In this paper, we also intend to see how basic reproduction number  $R_0$  affects the eradication of measles epidemic in a given area or population.

# MATERIALS AND METHODS

#### Model formulation

In this section, we developed a compartmental mathematical model of (SVEIR) to investigate the effect of vaccination in the dynamical spread of measles in the community. The human population is subdivided into four classes. These classes of individual are: Susceptible (S), Vaccinated (V), exposed (E), Infected (I) and recovered (R) individuals. The

formulation of the model is based on the following assumptions:

# Assumptions of the model

- i. the recruitment is through birth only.
- ii. The recruitment is constant.
- iii. all individuals are born susceptible.
- iv. an individual can be infected through coughing and sneezing, close personal contact or direct contact with infected nasal or throat secretions.
- v. infected individuals die either naturally or due to the disease.
- vi. vaccination is strictly on susceptible children.
- vii. individuals who received first dose of the vaccine return to the susceptible class when they lose immunity due to the fact that receiving first dose does not guarantee permanent immunity.
- viii. individuals who received first and second dose of the vaccine are approximately 97% immune to measles virus.
- ix. individuals who received first and second dose of the vaccine get infected at a minimum rate  $(1 \eta)\beta I$ , lower than the susceptible individuals, where  $\eta$  is the measles vaccine efficacy.

## Flow diagram of the model with constant control

We demonstrate the dynamical transfer of the population with the flow diagram in Figure 1 below:

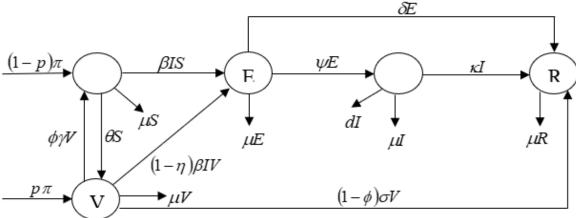


Figure 1: Flow diagram for the SVEIR Model

Table 1: Description of the variables of the models

Variables	Description
S(t)	Number of susceptible individuals at time $(t)$ .
V(t)	Number of vaccinated individuals at time (t).
E(t)	Number of exposed individuals at time $(t)$ .
I(t)	Number of infected individuals at time $(t)$ .
R(t)	Number of Recovered individuals at time ( <i>t</i> ).
N(t)	The total human population size at time $(t)$ .

Table 2: Description of the parameters of the models

Parameters	Description
π	The recruitment rate.
p	The proportion of newborn babies who are not vaccinated
1 - p	The proportion of newborn babies who are vaccinated
arphi	Proportion of individuals who are vaccinated once (not fully).
$1-\varphi$	The proportion of individuals who are vaccinated twice (fully).
heta	The rate at which the susceptible individuals move to the vaccinated class.
$\sigma$	The rate at which the vaccinated individuals moved to the recovered class.
δ	The rate at which the exposed individuals moved to the recovered class.

- β The probability of one infected individual to become infectious.
- The vaccine efficacy of the measles (which is the percentage reduction in a disease in a group of people η who received a vaccination in a clinical trial.).
- ψ The incubation rate (rate at which exposed individuals, E(t), progress to
  - infected I(t)).
- The rate of waning of first dose of vaccine. γ
- The rate at which the infected individuals moved to the recovered class. к
- d The death rate due to the disease in the infected class.
- The natural death rate. μ

# Equations of the model

$$\frac{dS}{dt} = (1 - p)\pi - \beta IS + \varphi \gamma V - (\theta + \mu)S \tag{1}$$

$$\frac{dV}{dt} = n\pi + \theta S - (1 - p)\beta IV - \alpha \gamma V - (1 - \alpha)\sigma V - \nu V$$

$$\frac{\partial V}{\partial t} = p\pi + \theta S - (1 - \eta)\beta IV - \varphi \gamma V - (1 - \varphi)\sigma V - \mu V \tag{2}$$

$$\frac{dE}{dt} = \beta IS + (1 - \eta)\beta IV - (\delta + \psi + \mu)E \tag{3}$$

$$\frac{dI}{dt} = \psi E - (\kappa + d + \mu)I \tag{4}$$

$$\frac{dR}{dt} = \delta E + \kappa I + (1 - \varphi)\sigma V - \mu R \tag{5}$$

$$\widetilde{N} = S + V + E + I + R \tag{6}$$

 $S(0) = S_0 > 0, V(0) = V_0 \geq 0, E(0) = E_0 \geq 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0, 0 \leq p \leq 1, 0 \leq \eta \leq 1, 0 \leq \varphi \leq 1$ The force of infection for human to human interaction  $\lambda = \beta I$ .

#### **Model Analysis**

# Existence and uniqueness of solution for the model

Consider the initial value problem (IVP)

$$y' = f(t, y), y(t_0) = y_0 (7)$$

whose solution exist and unique.

In this subsection, we shall establish conditions for the existence and uniqueness of solution for the system of equations.

Let 
$$y' = f(t, y) = f(y)$$
 (8)

such that

$$f_1(t, y) = f_1(y) = f_1 = (1 - p)\pi - \beta IS + \varphi \gamma V - (\theta + \mu)S$$
(9)

$$f_2(t, y) = f_2(y) = f_2 = p\pi + \theta S - (1 - \eta)\beta IV - \varphi \gamma V - (1 - \varphi)\sigma V - \mu V$$
 (10)

$$f_3(t,y) = f_3(y) = f_3 = \beta IS + (1-\eta)\beta IV - (\delta + \psi + \mu)E$$
(11)

$$f_4(t,y) = f_4(y) = f_4 = \psi E - (\kappa + d + \mu)I$$
 (12)

$$f_5(t,y) = f_5(y) = f_5 = \delta E + \kappa I + (1 - \varphi)\sigma V - \mu R$$
 (13)

# Theorem1: (Cauchy-Lipchitz theorem)

Consider the initial value problem (IVP)

$$y' = f(t, y_1, y_2, y_3, \cdots, y_n), y(t_0) = y_0, y_1(t_0) = y_1, y_2(t_0) = y_2, \cdots, y_1(t_0) = y_1$$
(14)

Let*R* denote the region

$$|t - t_0| \le a, ||y - y_0|| \le b, y = (y_1, y_2, y_3, \dots, y_n), y_0 = y_{1_0}, y_{2_0}, y_{3_0}, \dots, y_{n_0},$$
(15)

Suppose that f(t, y) satisfies the Lipchitz condition  $||f(t, y_n) - f(t, y_{n-1})|| \le L||y_n - y_{n-1}||, n = 1, 2, 3, \cdots$  (16)

whenever the pair  $(t, y_n)$  and  $(t, y_{n-1})$  belong to R, where L is a Lipchitz positive constant, then there exist a constant number  $\delta > 0$  such that there exists a unique continuous vector solution  $\bar{y}(t)$  of the system (14) in the interval  $|t - t_0| < \delta$ .

It is important to note that condition (16) is satisfied by the requirement that  $\frac{\partial f_i}{\partial v_i}$ ,  $\forall i, j = 1, 2, 3, \dots, n$  are continuous and bounded in the region R.

**Lemma 1.** If f(t,y) has continuous partial derivative  $\frac{\partial f_i}{\partial y_i}$  on a bounded closed convex domain R, then it satisfies a Lipchitz condition in R.

We are interested in the region

$$1 \le \varepsilon \le R. \tag{17}$$

We look for a bounded solution of the form

$$0 < R \le \infty. \tag{18}$$

We shall prove the following existence theorem.

**Theorem 2.** Let  $D^{\prime}$  denote the region defined in (16) such that (17) and (18) hold. Then there exist a solution of model system (9)-(13) which is bounded in the region  $D^{\prime}$ .

# **Proof:**

From (9)

$$\frac{dS}{dt} = (1 - p)\pi - \beta IS + \varphi \gamma V - (\theta + \mu)S$$
Let  $\frac{dS}{dt} = f_1(t, y_1) \equiv f_1(t, S)$ 

$$\begin{split} &f_1(t,S) = (1-p)\pi - \beta IS + \varphi \gamma V - (\theta + \mu)S \\ &\left|\frac{\partial f_1}{\partial S}\right| = \left|-(\beta I + \theta + \mu)\right| < \infty, \left|\frac{\partial f_1}{\partial V}\right| = \left|\varphi \gamma\right| < \infty, \left|\frac{\partial f_1}{\partial E}\right| = 0 < \infty, \left|\frac{\partial f_1}{\partial I}\right| = \left|-\beta S\right| < \infty, \left|\frac{\partial f_1}{\partial R}\right| = 0 < \infty \end{split}$$
 From (10) 
$$\frac{dV}{dt} = p\pi + \theta S - (1-\eta)\beta IV - \varphi \gamma V - (1-\varphi)\sigma V - \mu V \text{Let } \frac{dV}{dt} = f_2(t,y_2) \equiv f_2(t,V)$$
 
$$f_2(t,V) = p\pi + \theta S - \left(1-\eta\right)\beta IV - \phi \gamma V - \left(1-\phi\right)\sigma V - \mu V \left|\frac{\partial f_2}{\partial S}\right| = \left|\theta\right| < \infty \quad , \quad \left|\frac{\partial f_2}{\partial V}\right| = \left|-\left((1-\eta)\beta I + (1-\varphi)\sigma + \varphi \gamma + \mu\right)\right| < \infty, \left|\frac{\partial f_2}{\partial E}\right| = 0 < \infty, \left|\frac{\partial f_2}{\partial I}\right| = \left|-(1-\eta)\beta V\right| < \infty, \left|\frac{\partial f_2}{\partial R}\right| = 0 < \infty \end{split}$$

Similarly, we can also show that the remaining equations satisfy Lipchitz conditions.

This completes the proof.

Since all  $f_i$  and their partial derivatives of the model equations with respect to each dependent variables (i.e. S, V, E, I and R) are continuous and bounded in the interval  $0 < R < \infty$  by Lemma 1, there exists a unique solution of (9) to (13) in the region R.

# The positivity of solution of model

# Theorem 3:

Let the initial values of the parameters be

$$\{S(0) = S_0 > 0, V(0) = V_0 \geq 0, E(0) = E_0 \geq 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0, \in R_+^5.$$

Then, the solution set  $\{S(t), V(t), E(t), I(t), R(t)\}$  of the system (1) to (5) is non-negative for all t > 0.

# **Proof**

From (1)

$$\frac{dS}{dt} = (1 - p)\pi - \beta IS + \varphi \gamma V - (\theta + \mu)S$$

It follows by comparison theorem that

$$\frac{dS}{dt} \ge -(\beta I + \theta + \mu)S \tag{19}$$

Solving (19) with the aid of separation of variables, we have

$$\frac{dS}{S} \ge -(\beta I + \theta + \mu)dt$$

$$\int \frac{ds}{s} \ge -\int (\beta I + \theta + \mu) \, dt \tag{20}$$

Integrating (20), we have

 $S(t) \ge S(0)e^{-(\beta I + \theta + \mu)t} > 0$ 

From (2)

$$\frac{dV}{dt} = p\pi + \theta S - (1 - \eta)\beta IV - \phi \gamma V - (1 - \phi)\sigma V - \mu V \frac{dV}{dt} \ge -((1 - \eta)\beta I - \phi \gamma - (1 - \phi)\sigma - \mu)V$$
(21)

Solving (21) with the aid of separation of variables, we have

$$\frac{dV}{V} \ge -\left((1-\eta)\beta I - \varphi \gamma - (1-\varphi)\sigma - \mu\right)dt$$

$$\int \frac{dV}{V} \ge -\int \left( (1 - \eta)\beta I - \varphi \gamma - (1 - \varphi)\sigma - \mu \right) dt \tag{22}$$

Integrating (22), we have

 $V(t) \ge V(0)e^{-\int ((1-\eta)\beta I - \varphi \gamma - (1-\varphi)\sigma - \mu)dt}$ 

$$V(t) \ge V(0)e^{-((1-\eta)\beta I - \varphi \gamma - (1-\varphi)\sigma - \mu)t} > 0$$

Similarly, we can show that  $E(t) \ge 0$ ,  $I(t) \ge 0$  and  $R(t) \ge 0$ .

This completes the proof.

# The boundedness of solutions of the model

# Theorem 4:

The closed set

$$\Omega = \left\{ (S, V, E, I, R) \in R_+^5 : S + V + E + I + R = N; 0 < N(t) \le \frac{\pi}{\mu} \right\}$$
 (23)

is positively invariant.

## **Proof**

From the model equations (1) to (5), the total population is given by

$$N = S + V + E + I + R \tag{24}$$

Differentiating the total human population N(t) in (24) with respect to time t, we have

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \tag{25}$$

Substituting the differential equations (1) to (5) in (25), we have

$$\frac{dN}{dt} = \pi - \mu S - \mu V - \mu E - \mu I - \mu R - dI \tag{26}$$

$$\frac{dt}{dt} = \pi - \mu S - \mu V - \mu E - \mu I - \mu R - \alpha I \tag{20}$$

$$\frac{dN}{dt} = \pi - \mu (S + V + E + I + R) - dI \tag{27}$$

$$\text{Substitute (24) in (27) and have}$$

Substitute (24) in (27), we have

$$\frac{dN_h}{dt} = \pi - \mu N - dI \tag{28}$$

In the absence of measles virus, (i.e. d = 0) then (28) becomes

$$\frac{dN}{dt} = \pi - \mu N$$

$$\frac{dV}{dt} = \pi - \mu N$$

$$\frac{dN}{dt} + \mu N = \pi$$

$$I \cdot F = e^{\int \mu dt} = e^{\mu t}$$
(29)

$$I.F = e^{\int \mu dt} = e^{\mu t} \tag{30}$$

Multiply both sides of (29) by (30)

$$e^{\mu t} \frac{d\hat{N}}{dt} + \mu N e^{\mu t} = \pi e^{\mu t} \tag{31}$$

Equation (31) becomes

$$\frac{d}{dt}(Ne^{\mu t}) = \pi e^{\mu t} \Rightarrow d(Ne^{\mu t}) = \pi e^{\mu t} dt \implies \int d(Ne^{\mu t}) = \pi \int e^{\mu t} dt + C \Rightarrow N(t) = \frac{\pi}{\mu} + Ce^{-\mu t}$$

$$\lim_{t \to \infty} N(t) = \lim_{t \to \infty} \frac{\pi}{\mu} + C\lim_{t \to \infty} e^{-\mu t} \Longrightarrow \lim_{t \to \infty} N(t) = \frac{\pi}{\mu} + C(0)$$

$$\lim_{t \to \infty} N(t) = \frac{\pi}{\mu}$$
(32)

This result implies that if there is no disease,  $N = \frac{\pi}{\mu}$ . It also means that we have a steady state population. Therefore, the feasible solution set of the population of the system (29) exist in the region

$$\Omega = \left\{ (S, V, E, I, R) \in R_+^5 : S + V + E + I + R = N; \ 0 < N(t) \le \frac{\pi}{\mu} \right\}$$

This is a positive invariant set of the model which shows that the model is both biologically and mathematically meaningful in the domain  $\Omega$ .

# Disease-free equilibrium points of the model

The equilibrium points of the system of non-linear ordinary differential equation are obtained by setting the derivatives of the

model equation to zero (0).

$$\left(i.e.\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0\right)$$

Thus, at equilibrium point, the system of equation (1) to (5) becomes

$$(1-p)\pi - \beta I^{\circ}S^{\circ} + \varphi \gamma V^{\circ} - (\theta + \mu)S^{\circ} = 0$$

$$p\pi + \theta S^{\circ} - (1-\eta)\beta I^{\circ}V^{\circ} - \varphi \gamma V^{\circ} - (1-\varphi)\sigma V^{\circ} - \mu V^{\circ} = 0$$
(33)

$$p\pi + \theta S^{\circ} - (1 - \eta)\beta I^{\circ}V^{\circ} - \varphi \gamma V^{\circ} - (1 - \varphi)\sigma V^{\circ} - \mu V^{\circ} = 0$$

$$\tag{34}$$

$$\beta I^{\circ} S^{\circ} + (1 - \eta) \beta I^{\circ} V^{\circ} - (\delta + \psi + \mu) E^{\circ} = 0$$

$$\tag{35}$$

$$\psi E^{\circ} - (\kappa + d + \mu)I^{\circ} = 0 \tag{36}$$

$$\delta E^{\circ} + \kappa I^{\circ} + (1 - \varphi)\sigma V^{\circ} - \mu R^{\circ} = 0 \tag{37}$$

At disease-free equilibrium (in the absence of infection), there will be no exposed individuals

$$E^{\circ} = I^{\circ} = 0 \tag{38}$$

Substitute (38) in (33), we have

$$S^{\circ} = \frac{(1-p)\pi + \varphi \gamma V^{\circ}}{\theta + \mu} \tag{39}$$

Similarly, substitute (38) in (37), we have

$$R^{\circ} = \frac{(1 - \varphi)\sigma V^{\circ}}{\mu} \tag{40}$$

Substitute (39) in (34), we have

$$V^{\circ} = \frac{p\pi\mu + \theta\pi}{(\theta + \mu)(1 - \varphi)\sigma + \mu(\theta + \varphi\gamma + \mu)}$$

Therefore, the disease-free equilibrium point is denoted by  $\Phi^{\circ} = (S^{\circ}, V^{\circ}, E^{\circ}, I^{\circ}, R^{\circ})$ 

$$\Phi^{\circ} = \begin{cases}
\frac{(1-p)\pi[(\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu)]+\varphi\gamma[p\pi\mu+\theta\pi]}{(\theta+\mu)[(\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu)]}, & p\pi\mu+\theta\pi\\ \hline (\theta+\mu)[(\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu)], & (\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu), \\
\frac{(1-\varphi)\sigma[p\pi\mu+\theta\pi]}{\mu[(\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu)]}
\end{cases}$$
(41)

# Disease-endemic equilibrium points of the model

The equilibrium points of the system of non-linear ordinary differential equation are obtained by setting the derivatives of the

model equation to zero.

$$\left(i.e.\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0\right)$$

Thus, at equilibrium point, the system of equation (1) to (5) becomes

$$(1-p)\pi - \beta I^* S^* + \varphi \gamma V^* - (\theta + \mu) S^* = 0 \tag{42}$$

$$p\pi + \theta S^* - (1 - \eta)\beta I^* V^* - \varphi \gamma V^* - (1 - \varphi)\sigma V^* - \mu V^* = 0$$
(43)

$$\beta I^* S^* + (1 - \eta) \beta I^* V^* - (\delta + \psi + \mu) E^* = 0$$
(44)

$$\psi E^* - (\kappa + d + \mu)I^* = 0$$

$$\delta E^* + \kappa I^* + (1 - \varphi)\sigma V^* - \mu R^* = 0$$
(45)

At endemic equilibrium (i.e. in the presence of infection),

$$E^* \neq 0, I^* \neq 0 \tag{47}$$

From (45),

$$E^* = \frac{(\kappa + d + \mu)I^*}{I^*} \tag{48}$$

Substitute (48) in (44), we have
$$S^* = \frac{(\kappa + d + \mu)(\delta + \psi + \mu)I^*}{\beta \psi} - (1 - \eta)V^* \tag{49}$$

Substitute (49) in (43), we have

$$V^* = \frac{p\pi\beta\psi + \theta(\delta + \psi + \mu)(\kappa + d + \mu)}{\beta\psi[(1 - \eta)(\theta + \beta I^*) + \varphi\gamma + (1 - \varphi)\sigma + \mu]}$$

$$(50)$$

Substitute (50) in (49), we have

$$S^* = \frac{(\delta + \psi + \mu)(\kappa + d + \mu)[(1 - \eta)(\theta + \beta I^*) + \varphi \gamma + (1 - \varphi)\sigma + \mu - \beta \psi (1 - \eta)\theta] - (1 - \eta)p\pi(\beta \psi)^2}{\beta \psi[(1 - \eta)(\theta + \beta I^*) + \varphi \gamma + (1 - \varphi)\sigma + \mu]}$$
(51)

Substitute (48) and (50) in (46), we have

$$\frac{\delta(\kappa+d+\mu)I^*}{\psi} + \kappa I^* + (1-\varphi)\sigma \left[ \frac{p\pi\beta\psi + \theta(\delta+\psi+\mu)(\kappa+d+\mu)}{\beta\psi[(1-\eta)(\theta+\beta I^*) + \varphi\gamma + (1-\varphi)\sigma + \mu]} \right] - \mu R^* = 0$$

$$R^* = \frac{\delta(\kappa+d+\mu)I^*}{\psi} + \kappa I^* + (1-\varphi)\sigma \left[ \frac{p\pi\beta\psi + \theta(\delta+\psi+\mu)(\kappa+d+\mu)}{\beta\psi[(1-\eta)(\theta+\beta I^*) + \varphi\gamma + (1-\varphi)\sigma + \mu]} \right]$$

$$R^* = \frac{\delta(\kappa + d + \mu)I^* + \kappa \psi I^* + (1 - \varphi)\psi\sigma\left[\frac{p\pi\beta\psi + \theta(\delta + \psi + \mu)(\kappa + d + \mu)}{\beta\psi[(1 - \eta)(\theta + \beta I^*) + \varphi\gamma + (1 - \varphi)\sigma + \mu]}\right]}{h\mu}$$
(52)

Therefore, the endemic equilibrium point is denoted by  $\Phi^* = (S^*, V^*, E^*, I^*, R^*)$ 

$$\Phi^* = \begin{cases}
\frac{(\delta + \psi + \mu)(\kappa + d + \mu)[(1 - \eta)(\theta + \beta I^*) + \varphi \gamma + (1 - \varphi)\sigma + \mu - \beta \psi(1 - \eta)\theta] - (1 - \eta)p\pi(\beta \psi)^2}{\beta \psi[(1 - \eta)(\theta + \beta I^*) + \varphi \gamma + (1 - \varphi)\sigma + \mu]}, \\
\frac{p\pi\beta\psi + \theta(\delta + \psi + \mu)(\kappa + d + \mu)}{\beta\psi[(1 - \eta)(\theta + \beta I^*) + \varphi \gamma + (1 - \varphi)\sigma + \mu]}, \frac{(\kappa + d + \mu)I^*}{\psi}, I^*, \\
\frac{[\delta(\kappa + d + \mu) + \kappa\psi]I^* + (1 - \varphi)\psi\sigma\left[\frac{p\pi\beta\psi + \theta(\delta + \psi + \mu)(\kappa + d + \mu)}{\beta\psi[(1 - \eta)(\theta + \beta I^*) + \varphi \gamma + (1 - \varphi)\sigma + \mu]}\right]}{\psi\mu}
\end{cases}$$
(53)

# Computation of the basic reproduction Number $R_0$

The basic reproduction number  $R_0$  is the average number of new infections that one infected case will generate during their entire infectious lifetime (Heffernan et al., 2005). It is very important in determining whether the disease persists in the population or die out. We use the next generation matrix to compute the basic reproduction number  $R_0$  which is formulated in (Van den Driessche & Watmough, 2002; Guerra et al., 2017).

- . Let us assume that there are n compartments of which the first m compartments correspond to infected individuals. Let
- $F_i(y)$  be the rate of appearance of new infections in compartmenti,
- $V_i^+(y)$  be the rate of transfer of individuals into compartment i by all other means, and
- $V_i^-(y)$  be the rate of transfer of individuals out of compartments *i*.

It is assumed that each function is continuously differentiable at least twice in each variable. The disease transmission model consists of nonnegative initial conditions together with the following system of equations:

$$\frac{dy_i}{dt} = f_i(y) = F_i(y) - V_i(y), i = 1, 2, 3, \dots, n$$
(54)

where 
$$V_i(y) = V_i^-(y) - V_i^+(y)$$
. (55)

where 
$$V_i(y) = V_i^-(y) - V_i^+(y)$$
.
$$\frac{d}{dt} = F - V = \begin{pmatrix} \beta IS + (1 - \eta \beta IV) \\ 0 \end{pmatrix} - \begin{pmatrix} (\delta + \psi + \mu)E \\ (\kappa + d + \mu)I - \psiE \end{pmatrix}$$

$$R_0 = \rho(FV^{-1}) = \rho\left(\left(\frac{\partial F_i}{\partial y_j}\Big|_{E^0}\right)\left(\frac{\partial V_i}{\partial y_j}\Big|_{E^0}\right)^{-1}\right),\tag{56}$$

where F are the new infection transfer terms and V is the non-singular matrix of the remaining transfer terms. The basic reproduction number  $R_0$  of the model (1) – (5) is calculated using the next generation matrix (Van den Driessche & Watmough, 2002; Heffernan et al., 2005). In using their approach (Van den Driessche & Watmough, 2002; Heffernan et al., 2005), we

$$F = \left(\frac{\partial F_i}{\partial y_j}\Big|_{E^\circ}\right) = \begin{pmatrix} 0 & \beta S^\circ + (1-\eta)\beta V^\circ \\ 0 & 0 \end{pmatrix}$$
 (57)

Similarly, 
$$V = \begin{pmatrix} \frac{\partial v_i}{\partial y_j} \Big|_{E^s} \end{pmatrix} = \begin{pmatrix} (\delta + \psi + \mu) & 0 \\ -\psi & (\delta + \psi + \mu) \end{pmatrix}$$
$$|V| = \begin{vmatrix} (\delta + \psi + \mu) & 0 \\ -\psi & (\kappa + d + \mu) \end{vmatrix} \Rightarrow |V| = (\delta + \psi + \mu)(\kappa + d + \mu)$$
$$AdjV = \begin{pmatrix} (\kappa + d + \mu) & 0 \\ \psi & (\delta + \psi + \mu) \end{pmatrix}$$

$$V^{-1} = \frac{1}{(\delta + \psi + \mu)(\kappa + d + \mu)} \begin{pmatrix} (\kappa + d + \mu) & 0 \\ \psi & (\delta + \psi + \mu) \end{pmatrix}$$
Substitute (57) and (58) in (56), we have
$$FV^{-1} = \frac{1}{(\delta + \psi + \mu)(\kappa + d + \mu)} \begin{pmatrix} 0 & [S^{\circ} + (1 - \eta)V^{\circ}]\beta \end{pmatrix} \begin{pmatrix} (\kappa + d + \mu) & 0 \\ \psi & (\delta + \psi + \mu) \end{pmatrix}$$

$$FV^{-1} = \frac{1}{(\delta + \psi + \mu)(\kappa + d + \mu)} \begin{pmatrix} [S + (1 - \eta)V]\beta\psi & [\beta S + (1 - \eta)\beta V](\delta + \psi + \mu) \end{pmatrix}$$

$$|FV^{-1} = \frac{1}{(\delta + \psi + \mu)(\kappa + d + \mu)} \begin{pmatrix} [S^{\circ} + (1 - \eta)V^{\circ}]\beta\psi & [S^{\circ} + (1 - \eta)V^{\circ}]\beta \end{pmatrix} \\ |FV^{-1} - \lambda I| = \begin{pmatrix} [S^{\circ} + (1 - \eta)V^{\circ}]\beta\psi & [S^{\circ} + (1 - \eta)V^{\circ}]\beta \\ (\delta + \psi + \mu)(\kappa + d + \mu) & (\kappa + d + \mu) \end{pmatrix} - \begin{pmatrix} \lambda & 0 \\ 0 & \lambda \end{pmatrix} = 0$$

$$|FV^{-1} - \lambda I| = \begin{vmatrix} [S^{\circ} + (1 - \eta)V^{\circ}]\beta\psi & [S^{\circ} + (1 - \eta)V^{\circ}]\beta\psi \\ (\delta + \psi + \mu)(\kappa + d + \mu) & (\kappa + d + \mu) \end{pmatrix} = 0$$

$$-\lambda \begin{pmatrix} [S^{\circ} + (1 - \eta)V^{\circ}]\beta\psi & (\kappa + d + \mu) \\ (\delta + \psi + \mu)(\kappa + d + \mu) & (\kappa + d + \mu) \end{pmatrix}$$

$$\lambda_{2} = R_{e} = \frac{[S + (1 - \eta)V^{\circ}]\beta\psi}{(\delta + \psi + \mu)(\kappa + d + \mu)}$$

$$[(1 - p)\pi[(\theta + \mu)(1 - \varphi)\sigma + \mu(\theta + \varphi\gamma + \mu)] + \varphi\gamma[p\pi\mu + \theta\pi] + (1 - \eta) & p\pi\mu + \theta\pi$$

$$-\lambda \left( \frac{(\delta + (\lambda + \eta) + \beta + \mu)}{(\delta + \psi + \mu)(\kappa + d + \mu)} - \lambda \right) = 0 \Longrightarrow \lambda_1 = 0 \text{ and } \lambda_2 = \frac{(\delta + (\lambda + \eta) + \mu)}{(\delta + \psi + \mu)(\kappa + d + \mu)}$$

$$\lambda_1 = 0 \text{ and } \lambda_2 = \frac{(\delta + (\lambda + \eta) + \mu)}{(\delta + \psi + \mu)(\kappa + d + \mu)}$$

$$\lambda_2 = R_e = \frac{[S+(1-\eta)V^\circ]\beta\psi}{(\delta+\psi+\mu)(\kappa+d+\mu)}$$

$$R_{e} = \frac{\left[\frac{(1-p)\pi[(\theta+\mu)(\kappa+d+\mu)}{(\theta+\mu)[(\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu)]} + \varphi\gamma[p\pi\mu+\theta\pi]}{(\theta+\mu)[(\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu)]} + (1-\eta)\frac{p\pi\mu+\theta\pi}{(\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu)}\right]\beta\psi}{(\delta+\psi+\mu)(\kappa+d+\mu)}$$

$$R_{e} = \frac{\left[\frac{(1-p)\pi[(\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu)] + \varphi\gamma[p\pi\mu+\theta\pi] + (1-\eta)(p\pi\mu+\theta\pi)(\theta+\mu)}{(\theta+\mu)[(\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu)]}\right]\beta\psi}{(\delta+\mu)((\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu))}$$

$$R_{e} = \frac{\left[\frac{(1-p)\pi[(\theta+\mu)(1-\varphi)\sigma + \mu(\theta+\varphi\gamma+\mu)] + \varphi\gamma[p\pi\mu + \theta\pi] + (1-\eta)(p\pi\mu + \theta\pi)(\theta+\mu)}{(\theta+\mu)[(\theta+\mu)(1-\varphi)\sigma + \mu(\theta+\varphi\gamma+\mu)]}\right]\beta\psi}{(\delta+\psi+\mu)(\kappa+d+\mu)}$$

$$R_{e} = \frac{(\delta + \psi + \mu)(\kappa + \alpha + \mu)}{(\delta + \psi + \mu)(\kappa + \alpha + \mu)[\theta + \psi\gamma + \mu] + \psi\gamma[p\pi\mu + \theta\pi] + (1-\eta)(p\pi\mu + \theta\pi)(\theta + \mu)]\beta\psi}{(\delta + \psi + \mu)(\kappa + \alpha + \mu)(\theta + \mu)[(\theta + \mu)(1-\psi)\sigma + \mu(\theta + \psi\gamma + \mu)]}$$

$$R_{e} = \frac{[(1-p)\pi[(\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu)]+\varphi\gamma\pi[p\mu+\theta]+(1-\eta)\pi(p\mu+\theta)(\theta+\mu)]\beta\psi}{(\delta+\psi+\mu)(\kappa+d+\mu)(\theta+\mu)[(\theta+\mu)(1-\varphi)\sigma+\mu(\theta+\varphi\gamma+\mu)]}$$
(59)

# The local stability analysis of the disease-free equilibrium of the model

To examine the local stability of the disease-free  $E^{\circ}$  equilibrium, we obtain the Jacobian matrix by differentiating the functions  $(f_i: i = 1,2,3,4,5)$  partially with respect to the variables in the system of the modified equations.

The Jacobian matrix from the partial derivatives of (1) to (5) at disease-free  $(I_{F^{\circ}})$  is given by:

The Jacobian matrix from the partial derivatives of (1) to (5) at disease-free 
$$(J_{E^\circ})$$
 is given by 
$$J_{E^\circ} = \begin{pmatrix} -(\theta + \mu) & \varphi \gamma & 0 & \beta S^\circ & 0 \\ \theta & -(\varphi \gamma + (1 - \varphi)\sigma + \mu) & 0 & -(1 - \eta)\beta V^\circ & 0 \\ 0 & 0 & -(\delta + \psi + \mu) & \beta S^\circ + (1 - \eta)\beta V^\circ & 0 \\ 0 & 0 & \psi & -(\kappa + d + \mu) & 0 \\ 0 & (1 - \varphi)\sigma & \delta & \kappa & -\mu \end{pmatrix}$$

Let 
$$p = (\theta + \mu), q = (\varphi \gamma + (1 - \varphi)\sigma + \mu), r = (1 - \eta)\beta V^{\circ}, d = (\delta + \psi + \mu), e = \beta S^{\circ} + (1 - \eta)\beta V^{\circ},$$

$$|J_{E^{\circ}} - \lambda I| = \begin{vmatrix} -p - \lambda & \varphi \gamma & 0 & \beta S^{\circ} & 0 \\ \theta & -q - \lambda & 0 & -r & 0 \\ 0 & 0 & -d - \lambda & e & 0 \\ 0 & 0 & \psi & -f - \lambda & 0 \\ 0 & g & \delta & \kappa & -\mu - \lambda \end{vmatrix} = 0$$

$$[-\mu - \lambda] \begin{bmatrix} -p - \lambda & \varphi \gamma \\ \theta & -q - \lambda \end{bmatrix} [(d + \lambda)(f + \lambda) - e\psi] = 0$$

$$[-\mu - \lambda] \begin{bmatrix} -p - \lambda & \varphi \gamma \\ \theta & -q - \lambda \end{bmatrix} [(d + \lambda)(f + \lambda) - e\psi] = 0$$

 $[-\mu - \lambda][(d + \lambda)(f + \lambda) - e\psi][(p + \lambda)(q + \lambda) - \theta\varphi\gamma] = 0$ Therefore, the eigenvalues of the Jacobian matrix are:

$$-\mu - \lambda = 0 \Rightarrow \lambda_1 = -\mu$$

$$(d+\lambda)(f+\lambda) - e\psi = 0 \Rightarrow df + (d+f)\lambda + \lambda^2 - e\psi = 0 \Longrightarrow \lambda^2 + (d+f)\lambda + (df - e\psi) = 0$$

From quadratic equation: a = 1, b = d + f,  $c = df - e\psi$ 

$$\lambda_{2,3} = \frac{-b \pm \sqrt{b^2 - 4ac}}{2a} = \frac{-(d+f) \pm \sqrt{(d+f)^2 - 4(df - e\psi)}}{2}$$

$$\lambda_{2,3} = \frac{-(\delta + \psi + \kappa + d + 2\mu) \pm \sqrt{(\delta + \psi + \kappa + d + 2\mu)^2 - 4[(\delta + \psi + \mu)(\kappa + d + \mu) - \psi[\beta S^{\circ} + (1 - \eta)\beta V^{\circ}]]}}{2}$$

$$\lambda_{2,3} = \frac{-(\delta + \psi + \kappa + d + 2\mu) \pm \sqrt{(\delta + \psi + \kappa + d + 2\mu)^2 - 4[(\delta + \psi + \mu)(\kappa + d + \mu) - \psi[\beta S^{\circ} + (1 - \eta)\beta V^{\circ}]]}}{2}$$

$$\lambda_{2,3} = \frac{-(\delta + \psi + \kappa + d + 2\mu) \pm \sqrt{(\delta + \psi + \kappa + d + 2\mu)^2 - 4(\delta + \psi + \mu)(\kappa + d + \mu)[1 - \frac{\beta \psi[S^{\circ} + (1 - \eta)V^{\circ}]}{(\delta + \psi + \mu)(\kappa + d + \mu)}]}}$$

$$\lambda_{2,3} = \frac{-(\delta + \psi + \kappa + d + 2\mu) \pm \sqrt{(\delta + \psi + \kappa + d + 2\mu)^2 - 4(\delta + \psi + \mu)(\kappa + d + \mu)[1 - \frac{\beta \psi[S^{\circ} + (1 - \eta)V^{\circ}]}{(\delta + \psi + \mu)(\kappa + d + \mu)}]}}$$

$$\lambda_{2,3} = \frac{-(\delta + \psi + \kappa + d + 2\mu) \pm \sqrt{(\delta + \psi + \kappa + d + 2\mu)^2 - 4(\delta + \psi + \mu)(\kappa + d + \mu)[1 - R_e]}}{2}$$

$$\lambda_2 = \frac{-(\delta + \psi + \kappa + d + 2\mu) + \sqrt{(\delta + \psi + \kappa + d + 2\mu)^2 - 4(\delta + \psi + \mu)(\kappa + d + \mu)[1 - R_e]}}{2}$$

$$\lambda_{3} = \frac{-(\delta + \psi + \kappa + d + 2\mu) - \sqrt{(\delta + \psi + \kappa + d + 2\mu)^{2} - 4(\delta + \psi + \mu)(\kappa + d + \mu)[1 - R_{e}]}}{2}$$

Similarly,

$$(p+\lambda)(q+\lambda) - \theta \varphi \gamma = 0 \Rightarrow pq + (p+q)\lambda + \lambda^2 - \theta \varphi \gamma = 0 \Longrightarrow \lambda^2 + (p+q)\lambda + (pq - \theta \varphi \gamma) = 0$$

From quadratic equation: a = 1, b = p + q,  $c = pq - \theta \varphi \gamma$ 

$$\lambda_{4,5} = \frac{-b \pm \sqrt{b^2 - 4ac}}{2a} = \frac{-(p+q) \pm \sqrt{(p+q)^2 - 4(pq - \theta \varphi \gamma)}}{2a}$$

From quadratic equation: 
$$u = 1, b = p + q, c = pq - \theta \phi \gamma$$

$$\lambda_{4,5} = \frac{-b \pm \sqrt{b^2 - 4ac}}{2a} - \frac{-(p+q) \pm \sqrt{(p+q)^2 - 4(pq - \theta \phi \gamma)}}{2}$$

$$\lambda_4 = \frac{-(\theta + \phi \gamma + (1 - \phi)\sigma + 2\mu) + \sqrt{(\theta + \phi \gamma + (1 - \phi)\sigma + 2\mu)^2 - 4[(\theta + \mu)(\phi \gamma + (1 - \phi)\sigma + \mu) - \theta \phi \gamma]}}{2}$$

$$\lambda_5 = \frac{-(\theta + \phi \gamma + (1 - \phi)\sigma + 2\mu) - \sqrt{(\theta + \phi \gamma + (1 - \phi)\sigma + 2\mu)^2 - 4[(\theta + \mu)(\phi \gamma + (1 - \phi)\sigma + \mu) - \theta \phi \gamma]}}{2}$$

$$\lambda_5 = \frac{-(\theta + \varphi \gamma + (1 - \varphi)\sigma + 2\mu) - \sqrt{(\theta + \varphi \gamma + (1 - \varphi)\sigma + 2\mu)^2 - 4[(\theta + \mu)(\varphi \gamma + (1 - \varphi)\sigma + \mu) - \theta\varphi\gamma)}}{2}$$

# The global stability analysis of the disease-free equilibrium of the model

We investigated the global asymptotic stability of the disease- free equilibrium of measles infections using Castillo-Chavez theorem (Castillo-Chavez and Song, 2004; Delamater et al., 2019).

We write the model equations (1-5) in the form:

$$\frac{dX}{dt} = F(X, Z) \tag{60}$$

$$\frac{dt}{dt} = F(X, Z) \tag{60}$$

$$\frac{dY}{dt} = G(X, Z), G(X, 0) = 0 \tag{61}$$

Where  $X = (S, V, R) \in \mathbb{R}^3_+$  represents the uninfected individuals and  $Z = (E, I) \in \mathbb{R}^2_+$  represents the infected individuals. Let  $E^{\circ} = (X^{\circ}, 0)$  represents the disease-free equilibrium point of the system.

The disease-free equilibrium  $E^{\circ}$  to be globally asymptotically stable equilibrium for the model, the conditions (H1) and (H2)shown below should be satisfied:

H1: For 
$$\frac{dX}{dt} = F(X,0), X^{\circ}$$
, globally asymptotically stable.  
H2: For  $\frac{dZ}{dt} = D_Z G(X^{\circ}, 0) Z - \overline{G}(X, Z), \overline{G}(X, Z) \ge 0$  for all  $(X, Z) \in \Omega$ , where  $D_Z G(X^{\circ}, 0)$  is the Jacobian of  $G(X, 0)$  evaluated at  $(X^{\circ}, 0)$ 

Jacobian of G(X, 0) evaluated at  $(X^{\circ}, 0)$ 

#### **Theorem 5:**

The equilibrium point  $E^{\circ} = (X^{\circ}, 0)$  of the system (60) and (61) is globally asymptotically stable if  $R_e < 1$  and the conditions (H1)and (H2)are satisfied.

## **Proof:**

We partitioned the modified model system into two subsystems. These are  $X = (S, V, R) \in \mathbb{R}^3_+$  and  $Z = (E, I) \in \mathbb{R}^2_+$ . From equation (60) and (61) we have two functions: F(X,Z) and G(X,Z), where

equation (60) and (61) we have two functions: 
$$F(X,Z)$$
 and  $G(X,Z)$ , where

$$\frac{dS}{dt} = (1-p)\pi - \beta IS + \varphi \gamma V - (\theta + \mu)S$$

$$\frac{dV}{dt} = p\pi + \theta S - (1-\eta)\beta IV - \varphi \gamma V - (1-\varphi)\sigma V - \mu V$$

$$\frac{dR}{dt} = \delta E + \kappa I + (1-\varphi)\sigma V - \mu R$$

$$\frac{dV}{dt}\Big|_{E^o} = (1-p)\pi + \varphi \gamma V - (\theta + \mu)S$$

$$\frac{dV}{dt}\Big|_{E^o} = p\pi + \theta S - \varphi \gamma V - (1-\varphi)\sigma V - \mu V$$

$$\frac{dR}{dt}\Big|_{E^o} = (1-\varphi)\sigma V - \mu R$$
(62)

Therefore, the convergence of the solutions of the reduced system equation (62) is globally asymptotically stable in 
$$\Omega$$
.

Condition(H2):  $\frac{dZ}{dt} = G(X, Z) = \begin{pmatrix} \frac{dE}{dt} = \beta IS + (1 - \eta)\beta IV - (\delta + \psi + \mu)E \\ \frac{dI}{dt} = \psi E - (\kappa + d + \mu)I \end{pmatrix}$ 

$$\frac{dZ}{dt} = G(X, 0) = \begin{pmatrix} \frac{dE}{dt} |_{E^\circ} = 0 \\ \frac{dI}{dt} |_{E^\circ} = 0 \end{pmatrix} = 0$$

$$G(X,Z) = AZ - \overset{\wedge}{G}(X,Z) \Longrightarrow \overset{\wedge}{G}(X,Z) = AZ - G(X,Z)$$

where 
$$A = \frac{\partial G}{\partial Z}(X^{\circ}, 0) = D_Z(X^{\circ}, 0)$$
 is a metzler matrix.

$$\frac{dE}{dt} = f_1 = \beta IS + (1 - \eta)\beta IV - (\delta + \psi + \mu)E$$

$$\frac{dI}{dt} = f_2 = \psi E - (\kappa + d + \mu)I$$

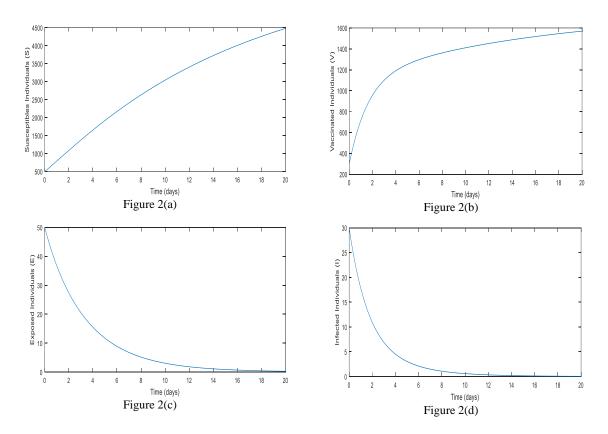
$$\frac{dl}{dt} = f_2 = \psi E - (\kappa + d + \mu) I$$

 $\frac{dt}{dt} = J_2 = \Psi B$  (K. 1 & 1 P.). The Jacobian matrix from the partial derivatives of (3) and (4) with respect to the infected variables at disease-free ( $A = J_{E^{\circ}}$ ) is given by:

$$\begin{split} A &= J_{E^{\circ}} = \begin{pmatrix} -(\delta + \psi + \mu) & \beta S^{\circ} + (1 - \eta)\beta V^{\circ} \\ \psi & -(\kappa + d + \mu) \end{pmatrix} \\ \overset{\wedge}{G}(X,Z) &= AZ - G(X,Z) \\ &= \begin{pmatrix} -(\delta + \psi + \mu) & \beta S^{\circ} + (1 - \eta)\beta V^{\circ} \\ \psi & -(\kappa + d + \mu) \end{pmatrix} \begin{pmatrix} E \\ I \end{pmatrix} - \begin{pmatrix} \beta IS + (1 - \eta)\beta IV - (\delta + \psi + \mu)E \\ \psi E - (\kappa + d + \mu)I \end{pmatrix} \\ \overset{\wedge}{G}_{1}(X,Z) &= -(\delta + \psi + \mu)E + [\beta S^{\circ} + (1 - \eta)\beta V^{\circ}]I - [\beta IS + (1 - \eta)\beta IV - (\delta + \psi + \mu)E] \\ \overset{\wedge}{G}_{1}(X,Z) &= [\beta S^{\circ} + (1 - \eta)\beta V^{\circ}]I - [\beta IS + (1 - \eta)\beta IV] \\ \overset{\wedge}{G}_{1}(X,Z) &= \beta I(S^{\circ} - S) + (1 - \eta)\beta I(V^{\circ} - V) \\ \overset{\wedge}{G}_{1}(X,Z) &= \beta I[(S^{\circ} - S) + (1 - \eta)(V^{\circ} - V)] \\ \overset{\wedge}{G}_{2}(X,Z) &= \psi E - (\kappa + d + \mu)I - [\psi E - (\kappa + d + \mu)I] = 0 \\ \overset{\wedge}{G}(X,Z) &= \begin{pmatrix} \overset{\wedge}{G}_{1}(X,Z) \\ \overset{\wedge}{G}_{2}(X,Z) \end{pmatrix} = \overset{\wedge}{\Longrightarrow} \overset{\wedge}{G}(X,Z) = \begin{pmatrix} \beta I[(S^{\circ} - S) + (1 - \eta)(V^{\circ} - V)] \\ 0 \end{pmatrix} \\ \overset{\wedge}{G}_{1}(X,Z) &= \beta I[(S^{\circ} - S) + (1 - \eta)(V^{\circ} - V)] \geq 0 \\ \overset{\wedge}{G}(X,Z) &\geq 0 \ \forall \ (X,Z) \in \Omega, \ \text{provided that } S^{\circ} \geq S \text{and} V^{\circ} \geq V. \end{split}$$

# RESULTS AND DISCUSSION

In this section, the numerical solution of the system (1) - (5) was carried out using the Runge-Kutta of order four scheme (RK4). The numerical results are shown in Figure 2. Figure 2, represented the graph of the model when the basic reproduction number is less than one.



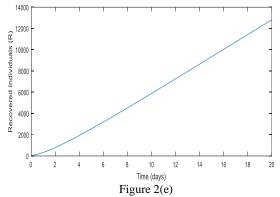


Figure 2: The graphical behaviour of the dynamic system (1) – (5) with a given initial conditions and parameter values: when  $R_e = 0.3452 < 1$ , the endemic equilibrium point is locally asymptotically stable, where  $\pi = 1000, \mu = 0.01, \eta = 0.95, p = 0.7, \varphi = 0.3, \beta = 0.0002, k = 0.6, \psi = 0.07, \theta = 0.06, \gamma = 0.25, d = 0.04, \delta = 0.30, \sigma = 0.75$ .

## Interpretation of the graphical results

In figure 2(a), the dynamics of the disease shows that there is rapid increase in the number of susceptible population from the initial population of 500 to 4500 throughout the remaining days.

In figure 2(b), the dynamics of the disease shows that there is rapid increase of the vaccinated population from the initial population of 500 to 1200 after t=4days and then a slow growth in the number of vaccinated population from 1200 to 4500 throughout the remaining days.

In figure 2(c), the dynamics of the disease shows that there is rapid decline in the exposed population from the initial population of 50 to 10 after t=6days and then a slow decline in the number of exposed population from 10 to its eradication point at t=18days.

In figure 2(d), the dynamics of the disease shows that there is rapid decline in the infected population from the initial population of 30 to 2 after t = 6days and then a slow decline in the number of infected population from 2 to its eradication point at t = 14days.

In figure 2(e), the dynamics of the disease shows that there is a rapid increase in the number of recovered population from the initial population of 25 to 12700 from t = 0 to t = 20days.

# Discussion of the results

From the numerical results above, Figures 2(a), 2(b), 2(c), 2(d) and 2(e) represents the graphs of the susceptible, vaccinated, exposed, infected and recovered individuals of the measles epidemic model. The decline in the number of exposed individuals is due to routine immunization program and as well as social distancing of the susceptible individuals from the infected individuals while the decline in the number of infected individuals may be due to early detection and treatment of the infectives in the community.

# CONCLUSION

In this Paper, we formulated mathematical model equations of measles infection with the aid of the system of ordinary differential equations to study the dynamics of measles infection with five compartments; susceptible class (S), vaccinated class (V), exposed class (E), infected class (I), and Recovered class (R) with their corresponding parameters. We investigated the existence and uniqueness of solution for the dynamic system using the Lipchitz condition to ascertain the effectiveness of the model as well as the positively invariant region of the system. The next generation matrix approach was used to determine the basic reproduction number  $R_0$ . The disease free equilibrium (DFE) was obtained. We also

obtained the local and global stability of the disease free equilibrium. We obtained the numerical solution of the model system in MATLAB. From the simulation, we observed that the measles infection eradicated in the environment with the original parameter values whose  $R_e = 0.3452 < 1$ .

# REFERENCES

Guerra, Fiona M.; Bolotin, Shelly; Lim, Gillian; Heffernan, Jane; Deeks, Shelley L.; Li, Ye; Crowcroft, Natasha S. (December 2017). "The basic reproduction number (R0) of measles: a systematic review". The Lancet. Infectious Diseases. **17** (12): e420–e428. doi:10.1016/ S1473-3099(17)30307-9. ISSN 1474-4457. PMID 28757186.

Rota PA, Moss WJ, Takeda M, de Swart RL, Thompson KM, Goodson JL (July 2016). "Measles". Nature Reviews. Disease Primers. 2: 16049. doi:10.1038/nrdp. 2016.49. PMID 27411684.

Bope, Edward T.; Kellerman, Rick D. (2014). Conn's Current Therapy 2015. Elsevier Health Sciences. p. 153. ISBN 9780323319560. Archived from the original on 8 September 2017.

Guerra FM, Bolotin S, Lim G, Heffernan J, Deeks SL, Li Y, Crowcroft NS (2017). "The basic reproduction number (R0) of measles: a systematic review". The Lancet Infectious Diseases. **17** (12): e420–e428. doi:10. 1016 /S1473-3099(17)30307-9. PMID 28757186.

Dept of Health, Saskatchewan (DHS) (2015) "Measles (Red Measles, Rubeola)". . Archived from the original on 10 February 2015. Retrieved 10 February 2015.

Milner DA (2015). Diagnostic Pathology: Infectious Diseases E-Book. Elsevier Health Sciences.p. 24. ISBN 9780323400374. Archived from the original on 8 September 2017.

Stanley J (2002). Essentials of Immunology & Serology. Cengage Learning. p. 323. ISBN 978-0766810648. Archived from the original on 8 September 2017.

Caserta, MT, ed. (2013). "Measles". Merck Manual Professional. Merck Sharp & DohmeCorp. Archived from the original on 23 March 2014. Retrieved 23 March 2014.

Atkinson W (2011). Epidemiology and Prevention of Vaccine-Preventable Diseases (12 ed.). Public Health Foundation. pp. 301–23. ISBN 9780983263135. Archived from the original on 7 February 2015. Retrieved 5 February 2015.

Marx JA (2010). Rosen's emergency medicine: concepts and clinical practice (7<sup>th</sup> ed.).Philadelphia: Mosby/Elsevier. p. 1541. ISBN 9780323054720. Archived from the original on 8 September 2017.

Delamater, P. L.; Street, E. J.; Leslie, T. F.; Yang, Y. T.; Jacobsen, K. H. (2019). "Complexity of the Basic Reproduction Number (R0)". Emerging Infectious Diseases. NIH website. **25** (1): 1–4. doi: 10.3201/eid2501.171901. PMC 6302597. PMID 30560777.

Chen (2018). Measles (Report). Medscape. Archived from the original on 25 September 2011. Retrieved 13 May 2020.

Russell, SJ; Babovic-Vuksanovic, D; Bexon, A; Cattaneo, R; Dingli, D; Dispenzieri, A; Deyle, DR; Federspiel, MJ; Fielding, A; Galanis, E (September 2019). "Oncolytic Measles Virotherapy and Opposition to Measles Vaccination". Mayo Clinic Proceedings. **94**(9): 1834–39. doi: 10. 1016 /j.mayocp.2019.05.006. PMC 6800178. PMID 31235278.

Kabra SK, Lodha R (August 2013). "Antibiotics for preventing complications in children with measles". The Cochrane Database of Systematic Reviews. **8**(8): CD 001477. doi:10. 1002/14651858. CD 0014 77.pub4. PMC 7055587. PMID 23943263.

GBD (2015) Mortality and Causes of Death Collaborators (October 2016). "Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015". Lancet. **388**(10053): 1459–1544. doi:10.1016/S0140-6736(16) 31012-1. PMC 5388903. PMID 27733281.

GBD 2013 Mortality Causes of Death Collaborators (January 2015). "Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of

death,1990-2013: a systematic analysis for the Global Burden of Disease Study 2013". Lancet. **385** (9963): 117–71. doi:10.1016/S0140-6736(14)61682-2. PMC 4340604. PMID 25530 442.

WHO (2014) "Measles Fact sheet N°286". Archived from the original on 3 February 2015. Retrieved 4 February 2015.

WHO (2018) "Measles cases spike globally due to gaps in vaccination coverage". World Health Organization . Retrieved 21 December 2018.

WHO (2019). "Measles – European Region". Archived from the original on 8 May 2019.

WHO (2019) "Measles fact sheet". Archived from the original on 1 June 2019. Retrieved 20 May 2019.

CDC (2013). Prevention of Measles, Rubella, Congenital Rubella Syndrome, and Mumps, 2013: Summary Recommendations of the Advisory Committee on Immunization Practices (ACIP) . *MMWR* 2013; 62 (RR04);1-34

CDC (2014) "Measles (Rubeola) Signs and Symptoms". Archived from the original on 2 February 2015. Retrieved 5 February 2015.

CDC (2019) "U.S. measles cases surge nearly 20 percent in early April, 2019. Retrieved 16 April 2019.

Heffernan, J., Smith, R. & Wahl, L. (2005). Perspectives on the basic reproductive ratio, Journal of the Royal Society Interface 2 (4) 281–293. 240.DOI:10.1098/rsif.2005.0042

Van den Driessche, P. & Watmough, J. (2002) Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission, Mathematical biosciences 180 (1), 29–48. https://doi.org/10.1016/S0025-5564(02)00108-6

Castillo-Chavez, C. and Song, B. (2004). Dynamical models of tuberculosis and their applications. Math. Biosci. Eng, 1(2):361–404.http://dx.doi.org/10.3934/mbe.2004.1.361



©2024 This is an Open Access article distributed under the terms of the Creative Commons Attribution 4.0 International license viewed via <a href="https://creativecommons.org/licenses/by/4.0/">https://creativecommons.org/licenses/by/4.0/</a> which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is cited appropriately.