

## On mathematical modeling and stability analysis of chickenpox models in the presence of weakened-immune individuals in a population

Charles Iwebuke Nkeki<sup>†\*</sup>, Imuwahen Anthonia Mbarie<sup>‡</sup>

<sup>†</sup>*Department of Mathematics, Faculty of Physical Sciences, University of Benin, Benin City, Edo State, Nigeria*

<sup>‡</sup>*Institute of Child Health, College of Medical Sciences, University of Benin, Benin City, Edo State, Nigeria*

Email(s): <sup>†</sup> [iwebuke.nkeki@uniben.edu](mailto:iwebuke.nkeki@uniben.edu), <sup>†</sup> [nkekicharles2003@yahoo.com](mailto:nkekicharles2003@yahoo.com),  
<sup>‡</sup> [imuwahen.mbarie@uniben.edu](mailto:imuwahen.mbarie@uniben.edu)

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**Abstract.** This paper considers susceptible-exposed-infected-weakened immune-recovered-vaccinated (SEIWRV) epidemic model for chickenpox infectious disease, in the presence of treatment. By using the new generation matrix, the basic reproduction number, denoted by  $\mathcal{R}_0$  for the model is obtained, and found to be re-enforced by two classes of individuals:-spread by first-time infected individuals who are unvaccinated, and spread by the weakened-immune individuals. The basic reproduction number is found to depend on incidence rate of the susceptible and weakened-immune individuals as well as the treatment rate. It is shown in this paper that the model exhibits two equilibriums, which include, the disease-free and the endemic equilibriums. By constructing a suitable Lyapunov function, it is observed that the global asymptotic stability of the disease-free equilibrium depends on number of infectious,  $\mathcal{R}_0$  and the treatment rate. As a result, it is found that chickenpox will remain endemic as long as weakened-immune individuals remain in the population. Also, the global endemic equilibrium is established using geometric approach. This approach is applied to a five-dimensional system of ordinary differential equations. Numerical simulations are also presented to illustrate our main results in this paper, using real data from Phuket Province, Thailand. It is found that it is possible to eradicate chickenpox from the population, only if the medical practitioners and researchers understand the role of weakened-immune individuals in the spread of chickenpox in our population.

**Keywords:** Mathematical model, SEIWRV, chickenpox, weakened-immune individuals, vaccination, stability analysis, endemic equilibrium, basic reproduction number.

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\*Corresponding author

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## 1 Introduction

The varicella-zoster virus (VZV) also known as chickenpox is one of the most contagious diseases in the world today. Individuals who have never had VZV, have never been vaccinated, or have a compromised immune (which is refers to as immunocompromised) systems, stand the highest risk of VZV infection.

The evidence of the existence of chickenpox is dated back to ancient era, that is, in 1691. Since then and till now, the disease continue to persist in our population, even with the presence of vaccine ranked to have above 95% efficacy. In 2014, the WHO estimated that over 4 million hospitalizations and over 4000 death occurred globally each year due to complications associated with VZV infection, see [20]. The question is: What are the complications? Between 2006 and 2022, according [29], a total of 11,990 varicella outbreaks are reported in china resulting in 354,082 cases. According to CDC 2024, in a report published Thursday May 30, 2024 by Mary Kekatos, in October 2022, the New York City Department of Health and Mental Hygiene discovered a varicella outbreak among people who migrated from Central and South American to New York City; the outbreak is still ongoing, see [13]. It was found that more than 90% of chickenpox cases in New York City outbreak is among unvaccinated people, and 1.4% of the cases are among people who are vaccinated with two vaccine doses. It simply implies that 8.6% of the cases arises from the weakened-immune people in the population, which are yet to be identified in the literature of medicine and epidemiology. It is now glaring that chickenpox complications must have arisen from the spread by weakened-immune persons. In July 2024, according to [29], 29 varicella cases are reported in 12 counties. Chickenpox outbreaks continue to occur among school children and adults due to the fact that some children and adults are unvaccinated. In Slovakia where vaccination against VZV is not widely used, 15,000-30,000 cases are reported annually, see [33]. According to [9], chickenpox is a widespread and reported in large numbers annually in Nineveh province in Iraq with a large increase in the number of cases which is recorded in 2008 as there was no immunization program against chickenpox in Iraq. Also, several outbreaks of chickenpox have been reported in different parts of the world and India is not left out. There was outbreak of chickenpox reported from remote villages of Chatra and Gumla district of Jharkhand State, where there was a clustering of cases having a similar presentation, in May 4, 2022.

The VZV is a highly contagious disease and causes chickenpox among children and adults. This disease causes an itchy rash, fever, and fluid-filled blisters. Chickenpox disease primarily affects children and adults, it can be severe, especially among pregnant women, babies, unvaccinated and those with a weakened immune system. There is no cure for chickenpox, but vaccine against VZV is available. This chickenpox vaccine is about 90% effective at preventing the chickenpox disease for most individuals. Though, most individuals who get VZV develop immunity to it, some individuals can get it more than once. Again, some infections may still occur among unvaccinated or immunocompromised individuals, which we refer to in this paper as weakened-immune individuals. In [5], it was stated that immune dysfunction can occur as a result of malnutrition and can also be characterized by recurrent infections and chronic inflammation. It was asserted in [27] that healthy immune system helps in enhancing the quality of life and reduces the risk of infectious disease in the population. They stated that chronic disease increases the risk of immune system impairment. Also, [2] emphasized clinically that stress significantly has negative impact on immune system of every individuals in the population. It was stated that stress can trigger molecular and immune modulation, affecting the distribution and trafficking of immune cells in various organs and altering their composition in the blood, which will has practical implications for and disease prevention. It has also been proven clinically that aging can result to weakened immune

system, see [30]. According to [17], other factors that bring about weakened immune system include lack of sleep, sedentary lifestyle, smoking, excessive alcohol consumption, lack of hygiene or excessive cleanliness, environmental factors, vitamin deficiencies, and lack of exercise or physical activity. A new fractional-order mathematical model for a tumor-immune surveillance mechanism was studied in [3], and they carried out analysis of the interaction between various cell populations and immune system using fractional differential equations. For a system of nonlinear dynamics, see [10] and [4].

Today, some vaccinated individuals can still get chickenpox, so also some people who are unvaccinated or weakened-immune, see [12]. It is imperative to note that when a person has chickenpox and recovers from it, the virus stays in their body and becomes dormant. After some time in life, people that will face a situation of having weakened immunity, will see the virus reactivate in their body. However, they can as well infect others who are unvaccinated or have weakened immunity. The question now is: Why do some individuals get chickenpox more than once? We deduced from [12] that, one may be susceptible to getting the chickenpox virus twice if: (i) an individual was less than 6 months old, when he or she had the first case of chickenpox; (ii) the first case of chickenpox was extremely mild; and (iii) an individual has a weakened-immune system. Hence, no matter the vaccine or immunity an individual has, it does not kill the virus, it only rendered the virus inactive in their nerve tissue, which can be reactivate later in life, especially if an individual developed weakened immunity [7]. In the United States, chickenpox was rampant before the development of VZV vaccines. About four million people contacted chickenpox in the early 1990s before the availability of vaccine in 1995. Today, chickenpox rates decreased by more than 97% in the United States. According to CDC, the chickenpox vaccine has prevented over 91 million cases of VZV for over 25 years.

Some of the fundamental questions now are: Can chickenpox be eradicated from our population? Why is chickenpox still prevalence in our population even in the presents of vaccine? What can we possibly do to eradicate this disease? This paper intend to provide answers to the above raised questions. Historically, there are numerous number of research papers that studies the spread dynamics of chickenpox using mathematical models. For instance, for the study of the effects of the adoption of vaccination strategies in the control of the spread of chickenpox using SIR and SVEIR models, see [11], and [1]. In [1], the SVEITR models of chickenpox was studied and analyzed in the presence of control measures which include vaccination strategy, and the use of medical assistance for infected individuals. In [14], the SEIR model was used to quantitatively describe the significance of immunization of newborn infants against the spread of chickenpox and treatment of both latent and active infected individuals. For more on the spread and management of chickenpox disease, see [18], [19] and [33]. Also, for the spread of chickenpox in Phuket Province, Thailand, see [21].

In [6], they considers and apply a dynamic mathematical model of chickenpox transmission to forecast the effect of various vaccination strategies on the age-specific incidence and outcome of infection. They further developed a deterministic age-structured model that put into consideration the increase in potential for transmission within school aged groups. Also investigated in their paper, was the various vaccine efficacy scenarios, vaccine coverage and vaccination strategies. Sensitivity analysis of varicella incidence was also carried out in their paper. A mathematical dynamic models for the spread of varicella through non-sexual social contacts was considered in [23]. Three popular model estimation methods on how well they fitted seroprevalence data are considered, and they further gives estimates for the basic reproduction number in their paper. Again, [26] developed a mathematical model to compare a school-based vaccination intervention scenario with a baseline scenario and stipulated that from 2010 to 2015, there are two epidemic waves of chickenpox displayed annually among school children in Shenzhen,

China. They depicted that the transmission dynamics of chickenpox among school children in Shenzhen remain vague. The work of [24] stated that in 2013 in Shenzhen City of China, there was a deadly unprecedented outbreak of chickenpox disease among school children, which led to several hospitalizations within the space of one week of its first emergence. For the prevalence of chickenpox, [9] examines the prevalence of chickenpox in Nineveh province sectors between 2005 and 2018, by adopting statistical analysis of all sections of the province. It was stipulated that isolation or quarantine of patients may reduce the spread of chickenpox in the area. In the work of [32], they adopted a conventional SEIR model to study the optimal strategies for the prevention and control of the spread of chickenpox outbreak in a school in a central city of China. Furthermore, [25] developed a mathematical compartmental model that deal with the optimal schedule for chickenpox vaccination in Jiangsu Province of China using SIR model. A mathematical model that depicts the transmission dynamics of chickenpox by incorporating a new parameter that captured the rate of precautionary measures was examined in [15]. The influence and the importance of precautionary measures by applying the real data collected at Phuket province, Thailand was examined. Chickenpox model that involves the Caputo fractional derivative was studied in [16]. They computed equilibrium points and the fundamental reproduction number of their chickenpox model.

In this paper, we consider six compartment model SEIWRV for the spread, control and management of chickenpox in our societies, where  $S$  stands for the susceptible population which increase by the net inflow of unvaccinated individuals into the population and individuals that fails vaccine, and decrease by natural death, vaccinated individuals and by infection that is acquired by contact between a susceptible and an infected individual who may be exposed or infected,  $E$ , stands for the exposed individuals to chickenpox, which increases by infection that is acquired by contact between a susceptible and an infected individual who may be exposed or infected and by contact between a weakened-immune and an infected individual who may be exposed or infected, and decrease by natural death and by infected individuals,  $I$ , stands for the number of infected individuals, which may increase by exposed individuals, and diminished by natural death, chickenpox induce death, weakened-immune and recovered individuals. The population,  $W$ , is the number of individuals who have weakened-immune system, which increase by inflow from the infected class, inflow of weakened-immune individuals (immigrates) and vaccination class, and decrease by contact between a weakened-immune and an infected individual who may be exposed or infected and natural death,  $R$  stands for individuals who recovered from chickenpox with strong immunity, which increases by infected persons that are free from the disease after recovery, but diminished by natural death, and  $V$  stands for number of individuals who are vaccinated, which increase by susceptible who are vaccinated and inflow of vaccinated individuals from outside the population, but decreases by natural death, number of individuals who fails vaccine and number of vaccinated individuals who are weakened-immune system.

### Highlights

- Mathematical model is developed to give insight on the management and control of chickenpox.
- The impact of weakened-immune person in the spread of chickenpox is studied.
- The basic reproduction number that depends on weakened-immunity is studied.
- The introduction and study of treatment strategy in our models is considered.
- Stability of equilibrium points are examined.

- The global asymptotic stability for endemic equilibrium point is studied.

**The following are the paper's main contributions:**

- The mathematical model was adopted to study the control and management of the spread of chickenpox in order to help stakeholders to have best possible understanding of the nature of spread and control of the disease.
- Expose medical practitioners and researchers to understanding the role of weakened-immune individuals on the spread of chickenpox in our population.
- Give the stakeholders an insight on how to reduce or possibly eradicate the chickenpox in our society.
- Demonstrate the disease transmission dynamics involving weakened-immune persons in the population as well as treatment strategy.
- The basic reproduction number that depends on weakened-immunity is obtained.
- The local and global asymptotic stability of equilibrium points are examined via Lyapunov function and Routh Hurwitz's.
- The global asymptotic stability for endemic equilibrium point was established using Li and Muldowney (1996) geometric technique.

The rest of the paper is structured as follows: In Section 2, we present the mathematical model formulation of chickenpox. The stability analysis of SEIWRV epidemic model is presented in Section 3. Section 4 presents the global stability of the disease-free equilibrium for the SEIWRV model. In Section 5, we present the stability analysis of the endemic equilibrium for the SEIWRV model. Section 6 presents the numerical simulation of the model. Finally, Section 7 presents the summary of the paper.

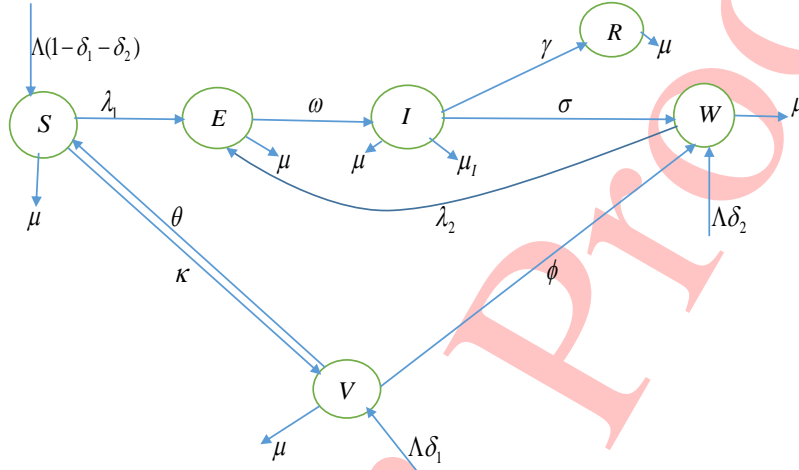
## 2 Model formulation

In this section, we consider the transmission dynamics for an outbreak of chickenpox in the presence of individuals with weakened-immune system and treatment strategy. The population structure comprises of six main compartments, which include the susceptible,  $S(t)$ , exposed,  $E(t)$ , infective,  $I(t)$ , weakened-immune,  $W(t)$ , recovered,  $R(t)$  and vaccinated,  $V(t)$ , and the sum of these compartments is equal to the total population,  $N(t)$  at time  $t$ .

We now give the following definitions.

**Definition 1.** *A weakened-immune (also known as immunocompromised) individual is someone whose immune system is compromised or malfunction at any time, making such individual more susceptible to chickenpox infections and other diseases.*

In this paper,  $\Lambda(1 - \delta_1 - \delta_2)$  represents the net inflows of unvaccinated individuals into the susceptible class,  $S(t)$  at time  $t$ , where  $\Lambda$  is the total inflow of individuals into the population,  $\delta_1$  is the fraction of inflow of individuals who are vaccinated into the vaccinated class,  $V(t)$  at time  $t$  and  $\delta_2$  is the fraction



**Figure 1:** The schematic diagram for SEIWRV model with vaccination and weakened-immune individuals, where  $\lambda_1 = \frac{\beta_S SI}{N}$  and  $\lambda_2 = \frac{\beta_W WI}{N}$

of inflow of weakened-immune individuals into the weakened-immune class,  $W(t)$  at time  $t$ . The rate at which the susceptible individuals who are vaccinated move to vaccinated class given by  $\kappa$  and  $\theta$  is the rate at which susceptible individuals fail vaccination.  $\beta_S$  is a contact rate of susceptible individual with infected individual,  $\beta_W$  is a contact rate of weakened-immune individual with the infected individual, and  $\mu$  represents natural death rate for the population. The rate at which the vaccinated class moves to weakened-immune class is given by  $\phi$ . The rate at which the exposed class moves to the infectious class is given by  $\omega$ . An infectious individual moves to recovered class with strong immunity at the rate  $\gamma$ , also moves to the weakened-immune class given that the individual recovered with weak immunity at the rate (i.e., weakened-immune transmission rate)  $\sigma$  and the disease induced death for the infectious class is given by the rate  $\mu_I$ . The incidence rates for the susceptible and weakened-immune classes are given by  $\lambda_1$  and  $\lambda_2$ , respectively. It is observed that the growth of weakened-immune individuals corresponds to some portion  $\delta_2$  of the inflow population  $\Lambda$  who arrive with weak immunity, some portion  $\phi$  of the vaccinated who only gain weak immunity, some portion  $(\sigma + r_2/N)$  of the infected who recover but only with weak immunity; at the same time, reduction of these weakened-immune individuals corresponds to some portion  $\mu$  of their population (due to deaths) and the conversion of weakened-immune to the exposed category due to their interaction with the infected ( $\beta_W WI/N$ ).

Figure 1 represents the schematic diagram for the SEIWRV model designed for chickenpox in the presence of weakened-immune persons, from which, we derived the following nonlinear system of dif-

ferential equations:

$$\left\{ \begin{array}{l} \frac{dS}{dt} = \Lambda(1 - \delta_1 - \delta_2) - \frac{\beta_S SI}{N} + \theta V - m_1 S, \quad t \geq 0, \\ \frac{dE}{dt} = \frac{\beta_S SI}{N} + \frac{\beta_W WI}{N} - m_2 E, \quad t \geq 0, \\ \frac{dI}{dt} = \omega E - m_3 I - \frac{rI}{N}, \quad t \geq 0, \\ \frac{dW}{dt} = \Lambda\delta_2 + \phi V + \sigma I - \frac{\beta_W WI}{N} + \frac{r_2 I}{N} - \mu W, \quad t \geq 0, \\ \frac{dR}{dt} = \gamma I - \mu R + \frac{r_1 I}{N}, \quad t \geq 0, \\ \frac{dV}{dt} = \Lambda\delta_1 + \kappa S - m_4 V, \quad t \geq 0, \end{array} \right. \quad (1)$$

subject to the initial values  $S(0) = S_0, E(0) = E_0, I(0) = I_0, W(0) = W_0, R(0) = R_0, V(0) = V_0$ , where  $r$  is the treatment rate, and is the sum of  $r_1$  being the treatment rate for  $R$  class and  $r_2$  is the treatment rate for the  $W$  class, that is,  $r = r_1 + r_2, m_1 = \mu + \kappa, m_2 = \mu + \omega, m_3 = \mu + \mu_I + \gamma + \sigma, m_4 = \mu + \theta + \phi$ .

### 3 Stability analysis of our SEIWRV epidemic model

In this section, we determine the stability analysis of SEIWRV chickenpox epidemic model. First, we consider the invariant set of the system of differential equations (1) in the following theorem:

**Theorem 1.** *The set*

$$D = \left\{ \begin{array}{l} (S, E, I, W, R, V) \in \mathbb{R}^6 | 0 < S(t) + E(t) \\ + I(t) + W(t) + R(t) + V(t) \leq N(t) \leq \frac{\Lambda}{\mu}, \\ S \geq 0, E \geq 0, I \geq 0, W \geq 0, R \geq 0, V \geq 0 \end{array} \right\}$$

is a positively invariant set for the system of differential equations (1).

*Proof.* We now determine the dynamics of the total population,  $N(t)$  at time  $t$  as follows:

$$N(t) = S(t) + E(t) + I(t) + W(t) + R(t) + V(t). \quad (2)$$

The derivative of  $N(t)$  is given as

$$\dot{N}(t) = \Lambda - \mu(S(t) + E(t) + I(t) + W(t) + R(t) + V(t)) - \mu_I I(t). \quad (3)$$

Simplifying (3), yields

$$\dot{N}(t) = \Lambda - \mu N(t) - \mu_I I(t). \quad (4)$$

It then follows that

$$\dot{N}(t) \leq \Lambda - \mu N(t). \quad (5)$$

Solving the differential inequality (5), we have

$$N(t) \leq N(0) \exp(-\mu t) + \frac{\Lambda}{\mu} (1 - \exp(-\mu t)). \quad (6)$$

Hence,  $\lim_{t \rightarrow \infty} \sup N(t) \leq \frac{\Lambda}{\mu}$ . Moreover if  $N(t) > \frac{\Lambda}{\mu}$ ,  $\dot{N}(t) < 0$  the solution of (1) either enters  $D$  in a finite time or  $N(t)$  approaches  $\frac{\Lambda}{\mu}$  asymptotically. As a result, the region  $D$  attracts all solutions to (1) in  $\mathbb{R}_+$ . Hence, since all solutions of (1) will remain or allow to tends to the field of  $D$ , then the set  $D$  is positive invariant for the system of differential equation (1), that is, all initial solutions belonging to  $D$ , remain in  $D$  for all  $t > 0$ .  $\square$

We now outline our model's main features before determining its mathematical results in terms of its stability analysis. The equilibrium values corresponding to  $S$ ,  $W$  and  $V$  are given respectively, as  $S_o$ ,  $W_o$  and  $V_o$ . Hence, the disease free equilibrium (DFE) point  $P_0$  of model (1) is obtained as  $P_0 = (S_o, 0, 0, W_o, 0, V_o)$ , where

$$S_o = \frac{m_1 m_4}{m_1 m_4 - \theta \kappa} \left( \frac{\Lambda(1 - \delta_1 - \delta_2)}{m_1} + \frac{\theta \Lambda \delta_1}{m_1 m_4} \right), \quad (7)$$

$$V_o = \frac{\Lambda \delta_1}{m_4} + \frac{\kappa m_1 m_4}{m_4 (m_1 m_4 - \theta \kappa)} \left( \frac{\Lambda(1 - \delta_1 - \delta_2)}{m_1} + \frac{\theta \Lambda \delta_1}{m_1 m_4} \right), \quad (8)$$

$$W_o = \frac{\Lambda \delta_2}{\mu} + \frac{\phi \Lambda \delta_1}{\mu m_4} + \frac{\phi \kappa m_1 m_4}{\mu m_4 (m_1 m_4 - \theta \kappa)} \left( \frac{\Lambda(1 - \delta_1 - \delta_2)}{m_1} + \frac{\theta \Lambda \delta_1}{m_1 m_4} \right). \quad (9)$$

One of the fundamental factor in the study of mathematical epidemiology is to determine the rate of spread and management strategies of an epidemic disease, which is referred to as the basic reproduction number, denoted by  $\mathcal{R}_o$ . Consequently, we derive the basic reproduction number of the model (1), and it is obtained as

$$\mathcal{R}_o = \frac{\mu \omega (\beta_S S_o + \beta_W W_o)}{m_2 (m_3 \Lambda + r \mu)} = \mathcal{R}_{o1} + \mathcal{R}_{o2}, \quad (10)$$

where  $\mathcal{R}_{o1} = \frac{\omega \mu \beta_S S_o}{m_2 (m_3 \Lambda + r \mu)}$  and  $\mathcal{R}_{o2} = \frac{\omega \mu \beta_W W_o}{m_2 (m_3 \Lambda + r \mu)}$ .

From (10), we observed that  $\mathcal{R}_o$  is made up of two parts,  $\mathcal{R}_{o1}$  and  $\mathcal{R}_{o2}$ . Hence,  $\mathcal{R}_{o1}$  is the expected number of secondary infections produced in compartment  $E$  by an infected individual originally in compartment  $E$ , and  $\mathcal{R}_{o2}$  is the expected number of secondary infections produced in compartment  $E$  by an infected individual originally in compartment  $W$ . It simply implies that chickenpox will continue to have additional reproduction rate  $\mathcal{R}_{o2}$ , as long as the weakened-immune individuals remain in the population. Medical practitioners and epidemiologist should now know that there is an additional factor that has remains unknown but doing avoke in the spread and retention of chickenpox in our society. Hence, more attention should be paid to individuals who are weakened-immune rather than susceptible individuals alone, if we must eradicate this disease in our population.

In order to determine the positive equilibriums of model (1), we need to determine the stationary values corresponding to  $S$ ,  $E$ ,  $I$ ,  $W$ ,  $R$  and  $V$  define respectively, as  $S^*$ ,  $E^*$ ,  $I^*$ ,  $W^*$ ,  $R^*$  and  $V^*$ , thus, we

set

$$\begin{cases} \Lambda(1 - \delta_1 - \delta_2) - \frac{\beta_S S^* I^*}{N} + \theta V^* - m_1 S^* = 0, \\ \frac{\beta_S S^* I^*}{N} + \frac{\beta_W W^* I^*}{N} - m_2 E^* = 0, \\ \omega E^* - m_3 I^* - \frac{r I^*}{N} = 0, \\ \Lambda \delta_2 + \phi V^* + \sigma I^* - \frac{\beta_W W^* I^*}{N} + \frac{r_2 I^*}{N} - \mu W^* = 0, \\ \gamma I^* - \mu R^* + \frac{r_1 I^*}{N} = 0, \\ \Lambda \delta_1 + \kappa S^* - m_4 V^* = 0. \end{cases} \quad (11)$$

It then follows that by using the fact that

$$\frac{dS^*}{dt} + \frac{dE^*}{dt} + \frac{dW^*}{dt} = 0, \quad (12)$$

we have

$$\begin{aligned} & \Lambda(1 - \delta_1) + (\theta + \phi) \frac{\Lambda \delta_1}{m_4} - \frac{m_2(m_3 N + r) I^*}{\omega N} + \frac{r_2 I^*}{N} + \sigma I^* \\ & + \left( \frac{(\theta + \phi) \kappa}{m_4} - m_1 \right) S^* - \mu W^* = 0. \end{aligned} \quad (13)$$

Also,

$$\frac{\beta_S S^* I^*}{N} + \frac{\beta_W W^* I^*}{N} = m_2 E^* = \frac{m_2(m_3 N + r) I^*}{\omega N}. \quad (14)$$

We therefore have the following simultaneous equations:

$$\begin{cases} x S^* + \mu W^* = B(I^*), \\ \beta_S S^* + \beta_W W^* = C, \end{cases} \quad (15)$$

where  $x = m_1 - \frac{(\theta + \phi) \kappa}{m_4}$ ,  $B(I^*) = A - C I^* + \Sigma I^*$ ,  $\Sigma = \sigma + \frac{r_2 \mu}{\Lambda}$  and  $C = \frac{m_2(m_3 \Lambda + r \mu)}{\mu \omega}$ ,  $A = \Lambda(1 - \delta_1) + (\theta + \phi) \frac{\Lambda \delta_1}{m_4}$ .

Solving (15) simultaneously for  $S^*$  and  $W^*$  in terms of  $I^*$ , we have

$$S^* = \frac{B(I^*)}{x} - \frac{\mu(\beta_S B(I^*) - xC)}{x(\mu\beta_S - \beta_W x)}, \quad (16)$$

$$W^* = \frac{\beta_S B(I^*) - xC}{\mu\beta_S - \beta_W x}. \quad (17)$$

It then follows that if  $\mathcal{R}_0 > 1$ , then system (1) has a positive equilibrium point  $P^* = (S^*, E^*, I^*, W^*, R^*, V^*)$ , where

$$\begin{aligned} S^* &= \frac{B(I^*)}{x} - \frac{\mu(\beta_S B(I^*) - xC)}{x(\mu\beta_S - \beta_W x)}, W^* = \frac{\beta_S B(I^*) - xC}{\mu\beta_S - \beta_W x}, V^* = \frac{\Lambda \delta_1}{m_4} + \frac{\kappa}{m_4} \left( \frac{B(I^*)}{x} - \frac{\mu(\beta_S B(I^*) - xC)}{x(\mu\beta_S - \beta_W x)} \right), \\ E^* &= \frac{C I^*}{m_2}, R^* = \frac{(\gamma \Lambda + \mu r) I^*}{\mu \Lambda}. \end{aligned}$$

Now, let us consider the following relation  $G(I^*)$ , define as follows:

$$G(I^*) = \frac{\beta_S S^* I^*}{N} + \frac{\beta_W W^* I^*}{N} - \frac{CI^*}{N}. \quad (18)$$

Finding the derivative with respect to  $I^*$  of (18), we have

$$G'(I^*) = \frac{\beta_S S^*}{N} + \frac{\beta_W W^*}{N} - \frac{C}{N} \quad (19)$$

$$= \frac{C}{N}(\mathcal{R}_o - 1). \quad (20)$$

Using the fact that  $N = \frac{\Lambda}{\mu}$ , we have

$$G'(I^*) = \frac{\mu C}{\Lambda}(\mathcal{R}_o - 1).$$

Hence,  $G'(I^*) \leq 0$ , if and only if  $\mathcal{R}_o \leq 1$ , and  $G'(I^*) > 0$ , if and only if  $\mathcal{R}_o > 1$ .

Again, we observe that at  $I^* = 0$ , we can obtain that if the model must be free from the disease, the following must also holds:

$$G'(0) = \frac{\mu(A\beta_S - C)}{\Lambda} + \frac{\mu(\beta_S A - xC)}{\Lambda(\mu\beta_S - \beta_W x)} \left( \beta_W - \frac{\mu\beta_S}{x} \right).$$

**Assumption 1.** We therefore make the following assumptions: Intuitively,

- $\frac{I^*}{I^*} \leq \frac{W^*}{W^*} < \frac{V^*}{V^*}$ .
- $\frac{\beta_S S^*}{N} > \frac{\phi V^*}{W^*}$ .

**Remark 1.** We remark that for  $S^* > 0$ ,  $E^* > 0$ ,  $I^* > 0$ ,  $W^* > 0$  and  $V^* > 0$ , the following ratios hold:

$$\begin{aligned} \frac{E^*}{I^*} &= \frac{m_3\Lambda + \mu r}{\mu\omega}, \quad \frac{W^*}{E^*} = \frac{\mu\omega(\beta_S B(I^*) - xC)}{(\mu\beta_S - \beta_W x)(m_3\Lambda + \mu r)}, \\ \frac{W^*}{V^*} &= \frac{m_4(\beta_S B(I^*) - xC)}{(\mu\beta_S - \beta_W x)(\Lambda\delta_1 + \kappa S^*)}, \\ \frac{V^*}{E^*} &= \frac{\mu\omega[(\mu\beta_S - \beta_W x)(x\Lambda\delta_1 + \kappa B(I^*)) - \mu\kappa(\beta_S B(I^*) - xC)]}{m_4(m_3\Lambda + \mu r)(\mu\beta_S - \beta_W x)}, \\ \frac{S^*}{E^*} &= \frac{\mu\omega[B(I^*)(1 - \mu\beta_S) + \mu xC]}{x(m_3\Lambda + \mu r)I^*}. \end{aligned}$$

## 4 Global stability of the disease-free equilibrium

In this section, we consider the global stability of the disease-free equilibrium  $P_0$  for system (1). We now have the following theorem:

**Theorem 2.** The system of differential equation (1) has a disease-free equilibrium point  $P_0$  given by

$$P_0 (S_o, 0, 0, W_o, 0, V_o)$$

which exists for all the values of the parameters. If  $\mathcal{R}_o > 1$ , then the endemic equilibrium  $P^* (S^*, E^*, I^*, W^*, R^*, V^*)$  admits the unique positive equilibrium point for the system of differential equation (1).

*Proof.* We are to show that  $I^* = 0$ , which will implies that  $E^* = R^* = 0$ . Now, it is clear that

$$\beta_S S^* + \beta_W W^* = C. \quad (21)$$

It now follows that

$$I^* = \left( 1 + \frac{\beta_W x - \mu \beta_S}{\mu \beta_S - \beta_W x} \right) \frac{A \beta_S - x C}{x(C - \Sigma)} = 0, \quad (22)$$

which is the desire result.  $\square$

**Theorem 3.** If  $\mathcal{R}_o \leq 1$ , then the disease-free equilibrium  $P_0$  of the system of differential equation (1) is globally asymptotically stable in the feasible region  $D$ . If  $\mathcal{R}_o > 1$ , then  $P_0$  is unstable.

*Proof.* The Jacobian matrix of the system (1) in the point  $P^*$  is obtained as

$$J|_{P^*} (S^*, E^*, I^*, W^*, R^*, V^*) = \begin{pmatrix} -m_1 & 0 & \frac{\mu \beta_S S^*}{\Lambda} & 0 & 0 & \theta \\ 0 & -m_2 & \frac{\mu (\beta_S S^* + \beta_W W^*)}{\Lambda} & 0 & 0 & 0 \\ 0 & \omega & -m_3 - \frac{r \mu I^*}{\Lambda} & 0 & 0 & 0 \\ 0 & 0 & \Sigma - \frac{\mu \beta_W W^*}{\Lambda} & -\mu & 0 & \phi \\ 0 & 0 & \gamma & 0 & -\mu & 0 \\ \kappa & 0 & 0 & 0 & 0 & -m_4 \end{pmatrix}.$$

We now find the characteristics equation of  $J|_{P^*}$  by using the relation  $\text{Det} (\lambda \mathbf{I}_J - J|_{P^*}) = 0$ , where  $\mathbf{I}_J$  is an identity matrix which has the dimension with matrix  $J|_{P^*}$  and  $\lambda$ 's are the eigenvalues of the system. It then follows that

$$\begin{aligned} & (\lambda + \mu)^2 \left( (\lambda + m_2) \left( \lambda + m_3 + \frac{r \mu}{\Lambda} \right) - \frac{\mu \omega (\beta_S S^* + \beta_W W^*)}{\Lambda} \right) \\ & \times [(\lambda + m_1) (\lambda + m_4) - \kappa \theta] = 0. \end{aligned} \quad (23)$$

Clearly,  $\lambda = -\mu$  twice are two eigenvalues of  $J|_{P^*}$ . The other eigenvalues of  $J|_{P^*}$  are determined by the following equations:

$$\lambda^2 + \lambda (m_1 + m_4) + m_1 m_4 - \kappa \theta = 0 \quad (24)$$

and

$$\lambda^2 + \lambda \left( m_2 + m_3 + \frac{r \mu}{\Lambda} \right) + m_2 \left( m_3 + \frac{r \mu}{\Lambda} \right) (1 - \mathcal{R}_o) = 0. \quad (25)$$

If  $\mathcal{R}_o > 1$ , then one eigenvalue is positive. Thus,  $P_0$  is unstable. We now consider the Lyapunov function  $F(E, I; t) = \omega E(t) + m_2 I(t)$ . Differentiating with respect to  $t$ , we have

$$\dot{F}(E, I; t) = \omega \dot{E}(t) + m_2 \dot{I}(t). \quad (26)$$

Adopting system (1), we have

$$\begin{aligned} \dot{F}(E, I; t) &= \omega \left( \frac{\beta_S S I}{N} + \frac{\beta_W W I}{N} \right) - m_2 \left( m_3 I + \frac{r I}{N} \right) \\ &= I \left[ \omega \left( \frac{\beta_S S}{N} + \frac{\beta_W W}{N} \right) - m_2 \left( m_3 + \frac{r}{N} \right) \right]. \end{aligned}$$

In the set  $D$ , we have  $N = \frac{\Lambda}{\mu}$ ,  $W = W_o$  and  $S = S_o$ , then

$$\begin{aligned} \dot{F}(E, I; t) &= I \left[ \omega \left( \frac{\mu \beta_S S_o}{\Lambda} + \frac{\mu \beta_W W_o}{\Lambda} \right) - m_2 \left( m_3 + \frac{r \mu}{\Lambda} \right) \right] \\ &= \frac{I}{\Lambda} [\omega (\mu \beta_S S_o + \mu \beta_W W_o) - m_2 (m_3 \Lambda + r \mu)] \\ &= \frac{I m_2 (m_3 \Lambda + r \mu)}{\Lambda} \left[ \frac{\mu \omega (\beta_S S_o + \beta_W W_o)}{m_2 (m_3 \Lambda + r \mu)} - 1 \right] \\ &= \frac{I m_2 (m_3 \Lambda + r \mu)}{\Lambda} (\mathcal{R}_o - 1). \end{aligned}$$

Hence,

$$\dot{F}(E, I; t) = \frac{I m_2 (m_3 \Lambda + r \mu)}{\Lambda} (\mathcal{R}_o - 1) \leq 0, \quad (27)$$

if and only if  $\mathcal{R}_o \leq 1$ . Moreover,  $\dot{F}(E, I; t) = 0$  if and only if  $I = 0$ . Therefore, the largest compact invariant set in  $D$ , when  $\mathcal{R}_o \leq 1$ , is the singleton  $P_0$ . By the LaSalle's (1976) extension to Lyapunov's method, the disease-free equilibrium  $P_0$  is globally asymptotically stable if  $\mathcal{R}_o \leq 1$ , and  $P_0$  is unstable in  $D$  if  $\mathcal{R}_o > 1$ .  $\square$

## 5 The stability analysis of the endemic equilibrium points

In this section, we analyze the stability of the endemic equilibrium  $P^*$ .

### 5.1 Local stability of the endemic equilibrium point

We first, show the local stability of the endemic equilibrium of system (1) around the endemic equilibrium  $P^*$ .

**Theorem 4.** *If  $\mathcal{R}_o > 1$ , then the endemic equilibrium  $P^*$  exists and is locally asymptotically stable in  $D$  for the system (1).*

*Proof.* The Jacobian matrix of the system (1) in the point  $P^*$  is obtained as

$$J|_{P^*}(S^*, E^*, I^*, W^*, R^*, V^*) = \begin{pmatrix} -m_1 - \frac{\beta_S S^*}{N} & 0 & -\frac{\beta_S S^*}{N} & 0 & 0 & \theta \\ \frac{\beta_S S^*}{N} & -m_2 & \frac{\beta_S S^*}{N} + \frac{\beta_W W^*}{N} & \frac{\beta_W W^*}{N} & 0 & 0 \\ 0 & \omega & -m_3 - \frac{r}{N} & 0 & 0 & 0 \\ 0 & 0 & \sigma + \frac{r_2}{N} - \frac{\beta_W W^*}{N} & -\mu - \frac{\beta_W W^*}{N} & 0 & \phi \\ 0 & 0 & \gamma + \frac{r_1}{N} & 0 & -\mu & 0 \\ \kappa & 0 & 0 & 0 & 0 & -m_4 \end{pmatrix},$$

$$J|_{P^*}(S^*, E^*, I^*, W^*, R^*, V^*) = \begin{pmatrix} -a_{11} & 0 & -a_{13} & 0 & 0 & \theta \\ a_{21} & -m_2 & a_{23} & a_{24} & 0 & 0 \\ 0 & \omega & -a_{33} & 0 & 0 & 0 \\ 0 & 0 & a_{43} & -a_{44} & 0 & \phi \\ 0 & 0 & a_{53} & 0 & -\mu & 0 \\ \kappa & 0 & 0 & 0 & 0 & -m_4 \end{pmatrix},$$

$$a_{11} = m_1 + \frac{\beta_S I^*}{N}, a_{13} = \frac{\beta_S S^*}{N}, a_{21} = \frac{\beta_S I^*}{N}, a_{23} = \frac{\beta_S S^*}{N} + \frac{\beta_W W^*}{N}, a_{24} = \frac{\beta_W I^*}{N}, a_{33} = m_3 + \frac{r}{N}, a_{43} = \sigma + \frac{r_2}{N} - \frac{\beta_W W^*}{N}, a_{44} = \mu + \frac{\beta_W I^*}{N}, a_{53} = \gamma + \frac{r_1}{N}.$$

Clearly, one of the roots of  $J|_{P^*}$  is negative, that is  $\lambda = -\mu$ . The remaining roots can be obtained from the following equation

$$\lambda^5 + A_4 \lambda^4 + A_3 \lambda^3 + A_2 \lambda^2 + A_1 \lambda + A_0 = 0,$$

where

$$\begin{aligned} A_4 &= a_{11} + a_{33} + a_{44} + m_2 + m_4, \\ A_3 &= a_{44}m_2 + (a_{44} + m_2)m_4 + a_{33}(a_{44} + m_2 + m_4) \\ &\quad + a_{11}(a_{33} + a_{44} + m_2 + m_4) - a_{23}\omega - \kappa\theta, \\ A_2 &= a_{33}a_{44}(m_2 + m_4) + (a_{33} + a_{44})m_2m_4 - (a_{13}a_{21} + a_{24}a_{43})\omega \\ &\quad - a_{23}\omega(a_{44} + m_4) + a_{11}(a_{44}m_2 + (a_{44} + m_2)m_4 + a_{33}(a_{44} + m_2 + m_4) \\ &\quad - a_{23}\omega) - \kappa(a_{33} + a_{44} + m_2)\theta, \\ A_1 &= a_{33}a_{44}m_2m_4 - ((a_{24}a_{43} + a_{23}a_{44})m_4 + a_{13}a_{21}(a_{44} + m_4))\omega \\ &\quad + a_{11}(a_{33}a_{44}m_2 + a_{44}m_2m_4 + a_{33}(a_{44} + m_2)m_4 \\ &\quad - (a_{24}a_{43} + a_{23}(a_{44} + m_4))\omega) - a_{33}\kappa(a_{44} + m_2)\theta + \kappa(-a_{44}m_2 + a_{23}\omega)\theta, \\ A_0 &= a_{11}m_4(a_{33}a_{44}m_2 - (a_{24}a_{43} + a_{23}a_{44})\omega) - a_{13}\omega(a_{21}a_{44}m_4 + a_{24}\kappa\phi) \\ &\quad + \kappa(a_{24}a_{43}\omega + a_{23}a_{44}\omega - a_{33}a_{44}m_2)\theta. \end{aligned}$$

Clearly,  $A_4 > 0$ . We now show that  $A_3 > 0$  as follows: Since  $a_{23} = \frac{\mathcal{R}_0 m_2 (m_3 \Lambda + r \mu)}{\mu N} > 0$ , then  $a_{23}\omega + \kappa\theta > 0$ . It then follows that  $A_3 > 0$  if and only if  $a_{44}m_2 + (a_{44} + m_2)m_4 + a_{33}(a_{44} + m_2 + m_4) +$

$a_{11}(a_{33} + a_{44} + m_2 + m_4) > a_{23}\omega + \kappa\theta$ . Again, we show that that  $A_2 > 0$ . But,  $a_{33}a_{44}(m_2 + m_4) + (a_{33} + a_{44})m_2m_4 + a_{11}(a_{44}m_2 + (a_{44} + m_2)m_4 + a_{33}(a_{44} + m_2 + m_4)) > 0$  and  $(a_{13}a_{21} + a_{24}a_{43})\omega + a_{23}\omega(a_{44} + m_4) + a_{11}a_{23}\omega + \kappa(a_{33} + a_{44} + m_2)\theta > 0$ . It therefore follows that  $A_2 > 0$  if and only if  $a_{33}a_{44}(m_2 + m_4) + (a_{33} + a_{44})m_2m_4 + a_{11}(a_{44}m_2 + (a_{44} + m_2)m_4 + a_{33}(a_{44} + m_2 + m_4)) > (a_{13}a_{21} + a_{24}a_{43})\omega + a_{23}\omega(a_{44} + m_4) + a_{11}a_{23}\omega + \kappa(a_{33} + a_{44} + m_2)\theta > 0$ . Similarly, we have that  $A_1 > 0$ , and  $A_0 > 0$ .

It follows from the Routh–Hurwitz criteria that all the eigenvalues associated to  $J|_{P^*}$  have negative real parts if and only if  $A_i > 0$  for  $i = 0, 1, 2, 3, 4$ ,

$$A_4A_3A_2 > A_2^2 + A_4^2A_1$$

and

$$(A_4A_1 - A_0)(A_4A_3A_2 - A_2^2 - A_4^2A_1) > A_0(A_4A_3 - A_2)^2 + A_4A_0^2.$$

Hence, the conditions of Routh–Hurwitz stability criterion are satisfied by our characteristic polynomial. We therefore conclude that  $P^*(S^*, E^*, I^*, W^*, R^*, V^*)$ , is locally asymptotically stable in  $D$  for our system of differential equations (1) when  $\mathcal{R}_0 > 1$ .  $\square$

## 5.2 Global stability of the endemic equilibrium point using geometric approach

To find the global stability of system (1), it is necessary to reduce system (1) by removing the recovered class, since the recovered class  $R$  does not appear anywhere on the dynamics of  $S, E, I, W$  and  $V$  classes. Hence, we discuss the following system of differential equations:

$$\begin{cases} \frac{dS}{dt} = \Lambda(1 - \delta_1 - \delta_2) - \frac{\beta_S SI}{N} + \theta V - m_1 S, & t \geq 0, \\ \frac{dE}{dt} = \frac{\beta_S SI}{N} + \frac{\beta_W WI}{N} - m_2 E, & t \geq 0, \\ \frac{dI}{dt} = \omega E - m_3 I - \frac{rI}{N}, & t \geq 0, \\ \frac{dW}{dt} = \Lambda\delta_2 + \phi V + \sigma I + \frac{r_2 I}{N} - \frac{\beta_W WI}{N} - \mu W, & t \geq 0, \\ \frac{dV}{dt} = \Lambda\delta_1 + \kappa S - m_4 V, & t \geq 0. \end{cases} \quad (28)$$

The solutions to the system of differential equations (28) associated with the nonnegative initial values remain nonnegative for all  $t$ . Since  $R$  class has been removed from the total population, so we now have subpopulation, given by (28). We therefore have the total population size of (28) denoted by  $N_s(t)$ , and it satisfies  $N_s(t) = S(t) + E(t) + I(t) + W(t) + V(t)$ , we therefore discuss the model in the set:

$$D_s = \left\{ (S, E, I, W, V) \in \mathbb{R}^5 \mid 0 < S(t) + E(t) + I(t) + W(t) + V(t) \leq N_s(t) \leq \frac{\Lambda}{\mu}, \right. \\ \left. S \geq 0, E \geq 0, I \geq 0, W \geq 0, V \geq 0 \right\},$$

which is also a positively invariant subset of  $D$  for the system of differential equations (28). It then follows that  $N(t) = N_s(t) + R(t)$  and  $D_s \subset D$ .

Next, we establish global stability of our model by adopting the Li and Muldowney (1996). In their work, they proposed a geometric approach of obtaining a global stability problem based on the criteria of Bendixson and Dulac, see [8] through the following Lemma 1

**Lemma 1.** Assume that the region  $\Pi$  is simply connected, and there is a compact absorbing set  $\Pi_0 (\subset \Pi)$ , and  $y$  is the only equilibrium in  $\Pi_0$ , if the quantity  $q_a$  satisfies

$$q_a = \limsup_{t \rightarrow \infty} \sup_{y_0 \in \Pi_0} \frac{1}{t} \int_0^t \rho_a(Q(y(s, y_0))) ds < 0, \quad (29)$$

where  $Q = Z_g Z^{-1} + Z \frac{\partial g^{[2]}(y)}{\partial y} Z^{-1}$ ,  $Z(y)$  is a matrix valued function,  $Z_g = \frac{\partial z_{ij}}{\partial y} g$ ,  $\frac{\partial g^{[2]}}{\partial y}$  is the second additive compound matrix of  $\frac{\partial g}{\partial y}$ , and  $\rho_a(Q) = \lim_{h \rightarrow 0^+} \frac{\|I_0 + hQ\| - 1}{h}$ , then equilibrium  $\bar{y}$  is global asymptotically stable in  $\Pi_0$ .

Hence, the global asymptotic stability is obtained, according to Lemma 1, is to find a norm  $\|\cdot\|$  such that  $q_a < 0$  for all  $y$  in the interior of  $D_s$ .

**Theorem 5.** If  $\mathcal{R}_o > 1$ , the endemic equilibrium  $P^*$  of the system of differential equations (1) is globally asymptotically stable in  $D_s (\subset D)$ .

*Proof.* It is obvious to see that there exists a compact absorbing set  $D_s (\subset D)$ , and  $P^*$  the only equilibrium point in  $D$ . Using Lemma 1, we establish that  $q_a < 0$ .

Then, the Jacobian matrix of the system (28) in the point  $P^*$  is obtained as

$$J|_{P^*}(S^*, E^*, I^*, W^*, V^*) = \begin{pmatrix} A_{11} & A_{12} & A_{13} & A_{14} & A_{15} \\ A_{21} & A_{22} & A_{23} & A_{24} & A_{25} \\ A_{31} & A_{32} & A_{33} & A_{34} & A_{35} \\ A_{41} & A_{42} & A_{43} & A_{44} & A_{45} \\ A_{51} & A_{52} & A_{53} & A_{54} & A_{55} \end{pmatrix},$$

$$= \begin{pmatrix} -m_1 - \frac{\beta_S I^*}{N} & 0 & -\frac{\beta_S S^*}{N} & 0 & \theta \\ \frac{\beta_S I^*}{N} & -m_2 & \frac{\beta_S S^* + \beta_W W^*}{N} & \frac{\beta_W I^*}{N} & 0 \\ 0 & \omega & -m_3 - \frac{r}{N} & 0 & 0 \\ 0 & 0 & \sigma + \frac{r_2}{N} - \frac{\beta_W W^*}{N} & -\mu - \frac{\beta_W I^*}{N} & \phi \\ \kappa & 0 & 0 & 0 & -m_4 \end{pmatrix}.$$

Therefore, the second additive compound matrix of  $J|_{P^*}$  is given as follows:

$$J^{[2]}|_{P^*}(S^*, E^*, I^*, W^*, V^*) = \begin{pmatrix} A_{11} + A_{22} & A_{23} & A_{24} & A_{25} & -A_{13} & -A_{14} & -A_{15} & 0 & 0 & 0 \\ A_{32} & A_{11} + A_{33} & A_{34} & A_{35} & A_{12} & 0 & 0 & -A_{14} & -A_{15} & 0 \\ A_{42} & A_{43} & A_{11} + A_{44} & A_{45} & 0 & A_{12} & 0 & A_{13} & 0 & -A_{15} \\ A_{52} & A_{53} & A_{54} & A_{11} + A_{55} & 0 & 0 & A_{12} & 0 & A_{13} & A_{14} \\ -A_{31} & A_{21} & 0 & 0 & A_{22} + A_{33} & A_{34} & A_{35} & -A_{24} & -A_{25} & 0 \\ -A_{41} & 0 & A_{21} & 0 & A_{43} & A_{22} + A_{44} & A_{45} & A_{23} & 0 & -A_{25} \\ -A_{51} & 0 & 0 & A_{21} & A_{53} & A_{54} & A_{22} + A_{55} & 0 & A_{23} & A_{24} \\ 0 & -A_{41} & A_{31} & 0 & -A_{42} & A_{32} & 0 & A_{33} + A_{44} & A_{45} & -A_{35} \\ 0 & -A_{15} & 0 & A_{31} & -A_{52} & 0 & A_{32} & A_{54} & A_{33} + A_{55} & A_{34} \\ 0 & 0 & -A_{51} & A_{41} & 0 & -A_{52} & A_{42} & -A_{53} & A_{43} & A_{44} + A_{55} \end{pmatrix}$$

$$= \begin{pmatrix} K_{11} & K_{12} & K_{13} & 0 & K_{15} & 0 & -\theta & 0 & 0 & 0 \\ \omega & K_{22} & 0 & 0 & 0 & 0 & 0 & 0 & -\theta & 0 \\ 0 & K_{32} & K_{33} & \phi & 0 & 0 & 0 & K_{38} & 0 & -\theta \\ 0 & 0 & 0 & K_{44} & 0 & 0 & 0 & 0 & K_{49} & 0 \\ K_{51} & K_{52} & 0 & 0 & K_{55} & 0 & 0 & K_{58} & 0 & 0 \\ 0 & 0 & K_{63} & 0 & K_{65} & K_{66} & \phi & K_{68} & 0 & 0 \\ -\kappa & 0 & 0 & K_{74} & 0 & 0 & K_{77} & 0 & K_{79} & K_{7,10} \\ 0 & 0 & 0 & 0 & 0 & \omega & 0 & K_{88} & \phi & 0 \\ 0 & -\theta & 0 & 0 & 0 & 0 & \omega & 0 & K_{99} & 0 \\ 0 & 0 & -\kappa & 0 & 0 & 0 & 0 & 0 & K_{10,9} & K_{10,10} \end{pmatrix},$$

where  $K_{11} = -m_1 - m_2 - \frac{\beta_S I^*}{N}$ ,  $K_{12} = \frac{\beta_S S^*}{N} + \frac{\beta_W W^*}{N}$ ,  $K_{13} = \frac{\beta_W I^*}{N}$ ,  $K_{15} = \frac{\beta_S S^*}{N}$ ,  $K_{22} = -m_1 - m_3 - \frac{r}{N} - \frac{\beta_S I^*}{N}$ ,  $K_{32} = \sigma + \frac{r_2}{N} - \frac{\beta_W W^*}{N}$ ,  $K_{33} = -\mu - m_1 - \frac{\beta_S I^*}{N} - \frac{\beta_W I^*}{N}$ ,  $K_{38} = -\frac{\beta_S S^*}{N}$ ,  $K_{44} = -m_1 - m_4 - \frac{\beta_S I^*}{N}$ ,  $K_{49} = -\frac{\beta_S S^*}{N}$ ,  $K_{51} = \frac{\beta_S S^*}{N}$ ,  $K_{52} = \frac{\beta_S I^*}{N}$ ,  $K_{55} = -m_2 - m_3 - \frac{r}{N}$ ,  $K_{58} = -\frac{\beta_W I^*}{N}$ ,  $K_{63} = \frac{\beta_S I^*}{N}$ ,  $K_{65} = \sigma + \frac{r_2}{N} - \frac{\beta_W W^*}{N}$ ,  $K_{66} = -\mu - m_2 - \frac{\beta_W I^*}{N}$ ,  $K_{68} = \frac{\beta_S S^*}{N} + \frac{\beta_W W^*}{N}$ ,  $K_{74} = \frac{\beta_S I^*}{N}$ ,  $K_{77} = -m_2 - m_4$ ,  $K_{79} = \frac{\beta_S S^*}{N} + \frac{\beta_W W^*}{N}$ ,  $K_{7,10} = \frac{\beta_W I^*}{N}$ ,  $K_{88} = -\mu - m_3 - \frac{r}{N} - \frac{\beta_W I^*}{N}$ ,  $K_{99} = -m_3 - m_4 - \frac{r}{N}$ ,  $K_{10,9} = \sigma + \frac{r_2}{N} - \frac{\beta_W W^*}{N}$ ,  $K_{10,10} = -\mu - m_4 - \frac{\beta_W I^*}{N}$ .

Next, we choose a suitable function

$$Z = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{E^*}{I^*} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{E^*}{I^*} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{E^*}{I^*} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{E^*}{W^*} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{E^*}{W^*} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{E^*}{W^*} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{E^*}{V^*} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{E^*}{V^*} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{E^*}{V^*} \end{pmatrix}.$$

We then obtain the matrix  $Q = Z_g Z^{-1} + ZJ^{[2]}|_{P^*} Z^{-1}$ , where  $Z_g$  is the derivative of  $Z$  in the direction of the given vector field  $g$ , such that

$$Z_g Z^{-1} = \text{diag} \left[ \begin{array}{c} 0, \frac{\dot{E}^*}{E^*} - \frac{\dot{I}^*}{I^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{I}^*}{I^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{I}^*}{I^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{I}^*}{I^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{W}^*}{W^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{W}^*}{W^*}, \\ \frac{\dot{E}^*}{E^*} - \frac{\dot{W}^*}{W^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{V}^*}{V^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{V}^*}{V^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{V}^*}{V^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{V}^*}{V^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{W}^*}{W^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{W}^*}{W^*}, \\ \frac{\dot{E}^*}{E^*} - \frac{\dot{W}^*}{W^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{V}^*}{V^*}, \frac{\dot{E}^*}{E^*} - \frac{\dot{V}^*}{V^*} \end{array} \right]$$

and

$$Q = Z_g Z^{-1} + Z \frac{\partial f^{[2]}}{\partial x} Z^{-1} = \begin{pmatrix} B_{11} & B_{12} & B_{13} & B_{14} \\ B_{21} & B_{22} & B_{23} & B_{24} \\ B_{31} & B_{32} & B_{33} & B_{34} \\ B_{41} & B_{42} & B_{43} & B_{44} \end{pmatrix},$$

where

$$\begin{aligned} B_{11} &= -\frac{\beta_S I^*}{N} - m_1 - m_2, B_{12} = \left( \frac{\beta_S S^* I^*}{NE^*} + \frac{\beta_W W^* I^*}{NE^*}, \frac{\beta_W I^{*2}}{NE^*} \right), \\ B_{13} &= \left( 0, \frac{\beta_S S^* I^*}{NE^*}, 0 \right), \\ B_{14} &= \left( 0, -\frac{\theta V^*}{E^*}, 0, 0 \right), B_{21} = \left( \frac{\omega E^*}{I^*}, 0 \right)', \\ B_{22} &= \begin{pmatrix} \frac{\dot{E}^*}{E^*} - \frac{i^*}{I^*} - \frac{r}{N} - m_1 - m_3 & 0 \\ \sigma + \frac{r_2}{N} - \frac{\beta_W W^*}{N} & \frac{\dot{E}^*}{E^*} - \frac{i^*}{I^*} - \frac{\beta_S I^*}{N} - \frac{\beta_W W^*}{N} - \mu - m_1 \end{pmatrix}, B_{23} = \begin{pmatrix} 0 & 0 & 0 \\ 0 & \frac{\phi W^*}{I^*} & 0 \end{pmatrix}, \\ B_{24} &= \begin{pmatrix} 0 & 0 & -\frac{\theta V^*}{I^*} & 0 \\ -\frac{\beta_S S^* W^*}{I^* N} & 0 & 0 & -\frac{\theta V^*}{I^*} \end{pmatrix}, B_{31} = \left( \frac{\beta_S S^* E^*}{I^* N}, 0, 0 \right)', B_{32} = \begin{pmatrix} \frac{\beta_S I^*}{N} & 0 \\ 0 & 0 \\ 0 & \frac{\beta_S I^{*2}}{NW^*} \end{pmatrix}, \\ B_{33} &= \begin{pmatrix} \frac{\dot{E}^*}{E^*} - \frac{i^*}{I^*} - \frac{r}{N} - m_2 - m_3 & 0 & 0 \\ 0 & \frac{\dot{W}^*}{W^*} - \frac{i^*}{I^*} - \frac{\beta_S I^*}{N} - m_1 - m_4 & 0 \\ \frac{I^*}{W^*} \left( \sigma + \frac{r_2}{N} - \frac{\beta_W W^*}{N} \right) & 0 & \frac{\dot{W}^*}{W^*} - \frac{i^*}{I^*} - \frac{\beta_W I^*}{N} - \mu - m_2 \end{pmatrix}, \\ B_{34} &= \begin{pmatrix} -\frac{\beta_W W^*}{N} & 0 & 0 & 0 \\ 0 & 0 & -\frac{\beta_S S^* V^*}{NW^*} & 0 \\ \frac{\beta_S S^*}{N} + \frac{\beta_W W^*}{N} & \frac{\phi V^*}{W^*} & 0 & 0 \end{pmatrix}, B_{41} = \left( 0, -\frac{\kappa E^*}{V^*}, 0, 0 \right)', \\ B_{42} &= \begin{pmatrix} 0 & 0 \\ 0 & 0 \\ -\frac{\theta I^*}{V^*} & 0 \\ 0 & -\frac{\kappa I^*}{V^*} \end{pmatrix}, B_{43} = \begin{pmatrix} 0 & 0 & \omega \\ 0 & \frac{\beta_S I^* W^*}{NV^*} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \\ B_{44} &= \begin{pmatrix} \frac{\dot{E}^*}{E^*} - \frac{\dot{W}^*}{W^*} - \frac{r}{N} - \mu - m_3 & 0 & \frac{\phi V^*}{W^*} & 0 \\ 0 & \frac{\dot{E}^*}{E^*} - \frac{\dot{V}^*}{V^*} - m_2 - m_4 & \frac{\beta_S S^*}{N} + \frac{\beta_W W^*}{N} & \frac{\beta_W I^*}{N} \\ 0 & \omega & \frac{\dot{E}^*}{E^*} - \frac{\dot{V}^*}{V^*} - \frac{r}{N} - m_3 - m_4 & 0 \\ 0 & 0 & \sigma + \frac{r_2}{N} - \frac{\beta_W W^*}{N} & \frac{\dot{E}^*}{E^*} - \frac{\dot{V}^*}{V^*} - \frac{\beta_W I^*}{N} - \mu - m_4 \end{pmatrix}. \end{aligned}$$

The estimation of the Lozinskil measure  $\rho_a(Q)$  corresponding to the vector norm  $\|\cdot\|$  in  $\mathbb{R}^5 \cong \mathbb{R} \begin{pmatrix} 5 \\ 2 \end{pmatrix}$  can be obtained as follows:

$$\rho_a(B) = \sup \left\{ \begin{array}{l} (B_{11}) + \|B_{12}\| + \|B_{13}\| + \|B_{14}\|, \\ \|B_{21}\| + \rho_{a_2}(B_{22}) + \|B_{23}\| + \|B_{24}\|, \\ \|B_{31}\| + \|B_{32}\| + \rho_{a_3}(B_{33}) + \|B_{34}\|, \\ \|B_{41}\| + \|B_{42}\| + \|B_{43}\| + \rho_{a_4}(B_{44}) \end{array} \right\}, \quad (30)$$

where, there exists  $t^* > t$  such that

$$\|B_{12}\| = \max \left\{ \frac{\beta_S S^* I^*}{NE^*} + \frac{\beta_W W^* I^*}{NE^*}, \frac{\beta_W I^{*2}}{NE^*} \right\} = \frac{\beta_S S^* I^*}{NE^*} + \frac{\beta_W W^* I^*}{NE^*}, \|B_{13}\| = \max \left\{ 0, \frac{\beta_S S^* I^*}{NE^*}, 0 \right\} = \frac{\beta_S S^* I^*}{NE^*}, \|B_{14}\| = \max \left\{ 0, -\frac{\theta V^*}{E^*}, 0, 0 \right\} = 0, \|B_{21}\| = \max \left\{ \frac{\omega E^*}{I^*}, 0 \right\} = \frac{\omega E^*}{I^*},$$

$$\begin{aligned} \rho_{a_2}(B_{22}) &= \max \left\{ \frac{\dot{E}^*}{E^*} - \frac{\dot{I}^*}{I^*} - \frac{r}{N} - \frac{\beta_W W^*}{N} - m_1 - m_3 + \sigma + \frac{r_2}{N}, \frac{\dot{E}^*}{E^*} - \frac{\dot{I}^*}{I^*} - \frac{\beta_S I^*}{N} - \frac{\beta_W W^*}{N} - \mu - m_1 \right\} \\ &= \frac{\dot{E}^*}{E^*} - \frac{\dot{I}^*}{I^*} - \frac{\beta_S I^*}{N} - \frac{\beta_W W^*}{N} - \mu - m_1, \end{aligned}$$

$$\|B_{23}\| = \max \left\{ 0, \frac{\phi W^*}{I^*}, 0 \right\} = \frac{\phi W^*}{I^*}, \|B_{24}\| = \max \left\{ -\frac{\beta_S S^* W^*}{I^* N}, 0, -\frac{\theta V^*}{I^*}, -\frac{\theta V^*}{I^*} \right\} = 0,$$

$$\|B_{31}\| = \max \left\{ \frac{\beta_S S^* E^*}{I^* N}, 0, 0 \right\} = \frac{\beta_S S^* E^*}{I^* N}, \|B_{32}\| = \max \left\{ \frac{\beta_S I^*}{N}, \frac{\beta_S I^{*2}}{NW^*} \right\} = \frac{\beta_S I^*}{N},$$

$$\begin{aligned} \rho_{a_3}(B_{33}) &= \max \left\{ \begin{array}{l} \frac{\dot{E}^*}{E^*} - \frac{\dot{I}^*}{I^*} - \frac{r}{N} - m_2 - m_3 + \frac{I^*}{W^*} \left( \sigma + \frac{r_2}{N} - \frac{\beta_W W^*}{N} \right), \\ \frac{\dot{E}^*}{E^*} - \frac{\dot{W}^*}{W^*} - \frac{\beta_S I^*}{N} - m_1 - m_4, \frac{\dot{E}^*}{E^*} - \frac{\dot{W}^*}{W^*} - \frac{\beta_W I^*}{N} - \mu - m_2 \end{array} \right\} \\ &= \frac{\dot{E}^*}{E^*} - \frac{\dot{I}^*}{I^*} - \frac{r}{N} - m_2 - m_3 + \frac{I^*}{W^*} \left( \sigma + \frac{r_2}{N} - \frac{\beta_W W^*}{N} \right), \end{aligned}$$

$$\|B_{34}\| = \max \left\{ \frac{\beta_S S^* \phi V^*}{N}, \frac{\phi V^*}{W^*}, -\frac{\beta_S S^* V^*}{NW^*}, 0 \right\} = \frac{\beta_S S^* \phi V^*}{N}, \|B_{41}\| = \max \left\{ 0, -\frac{\kappa E^*}{V^*}, 0, 0 \right\} = 0,$$

$$\|B_{42}\| = \max \left\{ -\frac{\theta I^*}{V^*}, -\frac{\kappa I^*}{V^*} \right\} = -\frac{\theta I^*}{V^*}, \|B_{43}\| = \max \left\{ 0, \frac{\beta_S I^* W^*}{NV^*}, \omega \right\} = \omega,$$

$$\begin{aligned} \rho_{a_4}(B_{44}) &= \max \left\{ \begin{array}{l} \frac{\dot{E}^*}{E^*} - \frac{\dot{W}^*}{W^*} - \frac{r}{N} - \mu - m_3, \frac{\dot{E}^*}{E^*} - \frac{\dot{V}^*}{V^*} - m_2 - m_4, \\ \frac{\dot{E}^*}{E^*} - \frac{\dot{V}^*}{V^*} - \frac{r}{N} - m_3 - m_4 + \frac{\phi V^*}{W^*} + \frac{\beta_S S^*}{N} + \sigma + \frac{r_2}{N}, \\ \frac{\dot{E}^*}{E^*} - \frac{\dot{V}^*}{V^*} - \mu - m_4 \end{array} \right\} \\ &= \frac{\dot{E}^*}{E^*} - \frac{\dot{W}^*}{W^*} - \frac{r}{N} - \mu - m_3. \end{aligned}$$

It then follows that when  $t^* > t$  and using Assumption 1 and Remark 1, we have

$$\begin{aligned}
 \|B_{11}\| + \|B_{12}\| + \|B_{13}\| + \|B_{14}\| &= \frac{\beta_S S^* I^*}{NE^*} + \frac{\beta_W W^* I^*}{NE^*} + \frac{\beta_S S^* I^*}{NE^*} - \frac{\beta_S I^*}{N} - m_1 - m_2 \\
 &= \frac{\dot{E}^*}{E^*} + \frac{\beta_S S^* I^*}{NE^*} - \frac{\beta_S I^*}{N} - m_1 \\
 &= \frac{\dot{E}^*}{E^*} + \frac{m_2(m_3 I^* N + r I^*)}{\omega E^*} - \frac{\beta_W W^* I^*}{NE^*} - \frac{\beta_S I^*}{N} - m_1 \\
 &= \frac{\dot{E}^*}{E^*} + m_2 - m_1 - \frac{\beta_W W^* I^*}{NE^*} - \frac{\beta_S I^*}{N} \\
 &= \frac{\dot{E}^*}{E^*} + m_2 - m_1 - \frac{\mu \omega \beta_W (\beta_S B(I^*) - xC)}{N(\mu \beta_S - \beta_W x)(m_3 \Lambda + r\mu)} - \frac{\beta_S I^*}{N}
 \end{aligned} \tag{31}$$

$$\begin{aligned}
 \|B_{21}\| + \rho_{a_2}(B_{22}) + \|B_{23}\| + \|B_{24}\| &= \frac{\dot{E}^*}{E^*} - \frac{I^*}{I^*} - \frac{\beta_S I^*}{N} - \frac{\beta_W W^*}{N} - \mu - m_1 + \frac{\omega E^*}{I^*} + \frac{\phi W^*}{I^*} \\
 &= \frac{\dot{E}^*}{E^*} - \frac{I^*}{I^*} - \frac{\beta_S I^*}{N} - \frac{\beta_W W^*}{N} - \mu - m_1 + \frac{\omega E^*}{I^*} + \frac{\phi W^*}{I^*} \\
 &= \frac{\dot{E}^*}{E^*} + \frac{m_3 \Lambda + r\mu}{\mu} + \frac{\beta_S B(I^*) - xC}{\mu \beta_S - \beta_W x} \left( \frac{\phi}{I^*} - \frac{\beta_W}{N} \right) - \frac{\beta_S I^*}{N} - \mu - m_1.
 \end{aligned} \tag{32}$$

$$\begin{aligned}
 \|B_{31}\| + \rho_{a_1}(B_{32}) + \rho_{a_3}(B_{33}) + \|B_{34}\| &= \frac{\dot{E}^*}{E^*} - \frac{I^*}{I^*} - \frac{r}{N} - m_2 - m_3 + \frac{\sigma N + r_2 I^*}{NW^*} - \frac{\beta_W I^*}{N} + \frac{\beta_S S^* E^*}{I^* N} + \frac{\beta_S I^*}{N} + \frac{\beta_S S^*}{N} \\
 &= \frac{\dot{E}^*}{E^*} - \frac{r}{N} - m_2 - m_3 + \frac{(\sigma N + r_2 I^*)(\mu \beta_S - \beta_W x)}{N(\beta_S B(I^*) - xC)} - \frac{\beta_W I^*}{N} + \frac{\beta_S S^* E^*}{I^* N} + \frac{\beta_S I^*}{N} + \frac{\beta_S S^*}{N} \\
 &\quad + \frac{m_2(m_3 \Lambda + r\mu)}{\mu \omega N} \left[ \frac{m_3 \Lambda + r\mu}{\mu \omega} + 1 \right] - \left[ \frac{m_3 \Lambda + r\mu}{\mu \omega} + 1 \right] \frac{\beta_W W^*}{N}.
 \end{aligned} \tag{33}$$

$$\|B_{41}\| + \|B_{42}\| + \|B_{43}\| + \rho_{a_4}(B_{44}) = \frac{\dot{E}^*}{E^*} - \frac{\dot{W}^*}{W^*} - \frac{r}{N} - \mu - m_3 + \omega - \frac{\theta I^*}{V^*}. \tag{34}$$

So when  $t^* > t$  and setting  $N = \frac{\Lambda}{\mu}$ , we have

$$\begin{aligned}
 \rho_a(B) &= \sup \left\{ \begin{array}{l} \|B_{11}\| + \|B_{12}\| + \|B_{13}\| + \|B_{14}\|, \\ \|B_{21}\| + \rho_{a_2}(B_{22}) + \|B_{23}\| + \|B_{24}\|, \\ \|B_{31}\| + \|B_{32}\| + \rho_{a_3}(B_{33}) + \|B_{34}\|, \\ \|B_{41}\| + \|B_{42}\| + \|B_{43}\| + \rho_{a_4}(B_{44}) \end{array} \right\} \\
 &= \|B_{31}\| + \|B_{32}\| + \rho_{a_3}(B_{33}) + \|B_{34}\| \\
 &= \frac{\dot{E}^*}{E^*} - \frac{r\mu}{\Lambda} - m_2 - m_3 + \frac{(\sigma \Lambda + r_2 \mu)(\mu \beta_S - \beta_W x)}{\Lambda(\beta_S B(I^*) - xC)} I^* - \frac{\mu \beta_W I^*}{\Lambda} \\
 &\quad + \frac{m_2(m_3 \Lambda + r\mu)}{\omega \Lambda} \left[ \frac{m_3 \Lambda + r\mu}{\mu \omega} + 1 \right] - \left[ \frac{m_3 \Lambda + r\mu}{\mu \omega} + 1 \right] \frac{\mu \beta_W W^*}{\Lambda} + \frac{\mu \beta_S I^*}{\Lambda} \\
 &\leq \frac{\dot{E}^*}{E^*} - \frac{r\mu}{\Lambda} - m_2 - m_3 + \frac{m_2(m_3 \Lambda + r\mu)}{\omega \Lambda} \left[ \frac{m_3 \Lambda + r\mu}{\mu \omega} + 1 \right],
 \end{aligned} \tag{35}$$

provided that  $\left[ \frac{(\sigma \Lambda + r_2 \mu)(\mu \beta_S - \beta_W x)}{\beta_S B(I^*) - xC} - \mu \beta_W + \mu \beta_S \right] I^* \leq \left[ \frac{m_3 \Lambda + r\mu}{\mu \omega} + 1 \right] \mu \beta_W W^*$ . Hence,

$$\rho_a(B) \leq \frac{\dot{E}^*}{E^*} - \beta, \tag{36}$$

where  $\beta = \frac{r\mu}{\Lambda} + m_2 + m_3 - \frac{m_2(m_3\Lambda + r\mu)}{\omega\Lambda} \left[ \frac{m_3\Lambda + r\mu}{\mu\omega} + 1 \right] > 0$ .

Now, when  $t^* > t$ , it then follows that by substituting (36) into (29), we have

$$\begin{aligned} q_a &= \lim_{t \rightarrow \infty} \sup_{x_0 \in \Pi_0} \sup \frac{1}{t} \int_0^t \rho_a(B) ds \\ &< \lim_{t \rightarrow \infty} \sup_{x_0 \in \Pi_0} \sup \left[ \frac{1}{t} \int_0^{t^*} \rho_a(B) ds + \frac{1}{t} \int_{t^*}^t \left( \frac{\dot{E}^*}{E^*} - \beta \right) ds \right] \\ &< \lim_{t \rightarrow \infty} \sup_{x_0 \in \Pi_0} \sup \left[ \frac{1}{t} \int_0^{t^*} \rho_a(B) ds + \frac{1}{t} \ln \frac{E(t)}{E(t^*)} - \frac{\beta(t-t^*)}{t} \right]. \end{aligned}$$

Since the subsystem (28) is uniformly persistent, there exists  $t > t^*$  such that  $\frac{1}{t} \ln E(t) < \frac{\beta}{2}$ , then

$$q_a < -\frac{\beta}{2} < 0. \quad (37)$$

Hence, we have that the equilibrium  $(S^*, E^*, I^*, W^*, R^*, V^*)$  is globally asymptotically stable for the subsystem (28) in  $D_s$ . We now solve  $R(t)$  as follows:

$$\dot{R} = \left( \gamma + \frac{r_1\mu}{\Lambda} \right) I^* - \mu R. \quad (38)$$

Solving (38), we have

$$R(t) = \Pi e^{-\mu t}, \quad (39)$$

where  $\Pi = \left( \gamma + \frac{r_1\mu}{\Lambda} \right) I^* \int_0^t e^{\mu\tau}$ . It implies that as  $t \rightarrow \infty$ , we have  $R(t) \rightarrow R^*(t) = \frac{1}{\mu} \left( \gamma + \frac{r_1\mu}{\Lambda} \right) I^*$ . Hence, the endemic equilibrium  $P^*$  of the system of differential equations (1) is globally asymptotically stable in  $D_s(\subset D)$  provided that  $\mathcal{R}_0 > 1$ .  $\square$

## 6 Numerical simulations

In this section, we investigate the empirical results of our analytical experiments of chickenpox model with treatment and vaccination, and in the presence of weakened-immune individuals in a population. In order to validate our analytical results, we carryout numerical results of the analytical experiments using the real data presented in [15] for Phuket Province, Thailand in 2021 which has a total population of 418,785 people. According to [15], in Phuket Province, Thailand, there are 83,812 susceptible individuals, 604 exposed individuals, 534 infected individuals, and 178 total recovered individuals. We assume in this paper that 20% of the recovered individuals are with weakened-immune system, which gave a total of 36 persons, while the remaining 142 recovered with strong immunity. All these values are taken to be the initial population of the model.

We assume that 20% of 0.000106 (recovered rate from Jose *et.al* (2023)) recovered with weakened immunity. We also assume that 20% of 0.000504 (vaccinated rate from [15]) moves to weakened immunity compartment. Again, we assume that 30% of infected persons recovered without treatment, 40% recovered with strong immunity and 30% recovered with weakened-immune system. The experimental

**Table 1:** The baseline values of the parameters for the *SEIWRV* disease model

Parameter	Baseline value	References
$\Lambda$	9 day <sup>-1</sup>	[15]
$\beta_S$	0.000010778 day <sup>-1</sup>	[15]
$\beta_W$	0.00001 day <sup>-1</sup>	Assumed
$\kappa$	0.000504 day <sup>-1</sup>	[15]
$\omega$	0.000318 day <sup>-1</sup>	[15]
$\gamma$	0.0000848 day <sup>-1</sup>	[15]
$\mu$	0.000012814 day <sup>-1</sup>	[28]
$\mu_I$	0.00001694 day <sup>-1</sup>	[15]
$\theta$	0.000429 day <sup>-1</sup>	[15]
$\delta_1$	0.1 day <sup>-1</sup>	Assumed
$\delta_2$	0.4 day <sup>-1</sup>	Assumed
$\sigma$	0.0000212 day <sup>-1</sup>	Estimated from [15]
$\phi$	0.0001008 day <sup>-1</sup>	Estimated from [15]
$r_1$	0.000391 day <sup>-1</sup>	Estimated
$r_2$	0.000086 day <sup>-1</sup>	Estimated

**Table 2:** Initial values for model's state variables for the *SEIWRV* disease model

Variable	Initial values	References
$\Lambda$	9	[15]
$N$	418,785	[15]
$S$	83,312	[15]
$E$	604	[15]
$I$	534	[15]
$W$	36	Estimated
$V$	573	[15]
$R$	142	[15]

baseline values of the parameters for the *SEIWRV* disease model can be found in Table 1, and the experimental initial values for model's state variables for the *SEIWRV* disease model can be found in Table 2.

Since it has been established clinically that people with weakened immune system can spread chickenpox through the same primary process as a healthy person, see [22], it is imperative for us to determine how contact rate with those with weakened immune system can spread the chickenpox disease in a population. Table 3 depicts the relationship between  $\beta_W$  and  $\mathcal{R}_{o1}$ ,  $\mathcal{R}_{o2}$ , and  $\mathcal{R}_o$ . From the table, we observed that slight increase in  $\beta_W$ , will lead to a high proportional increase in  $\mathcal{R}_{o2}$ , and in turn leads to increase in  $\mathcal{R}_o$ . The result shows that the  $\mathcal{R}_o$  for the Phuket Province, Thailand to be 0.0344,

**Table 3:** Impact of  $\beta_W$  in  $\mathcal{R}_{o1}$ ,  $\mathcal{R}_{o2}$  and  $\mathcal{R}_o$  for the SEIWRV disease model

$\beta_W$	$\mathcal{R}_{o1}$	$\mathcal{R}_{o2}$	$\mathcal{R}_o$
0.00001	0.0048	0.0296	0.0344
0.00002	0.0048	0.0593	0.0641
0.00003	0.0048	0.0889	0.0937
0.00004	0.0048	0.1186	0.1233
0.00005	0.0048	0.1482	0.1530
0.00006	0.0048	0.1778	0.1826
0.00010	0.0048	0.2964	0.3012
0.00020	0.0048	0.5928	0.5976
0.00030	0.0048	0.8892	0.8940
0.00040	0.0048	1.1856	1.1904
0.00050	0.0048	1.4820	1.4868
0.00060	0.0048	1.7784	1.7831

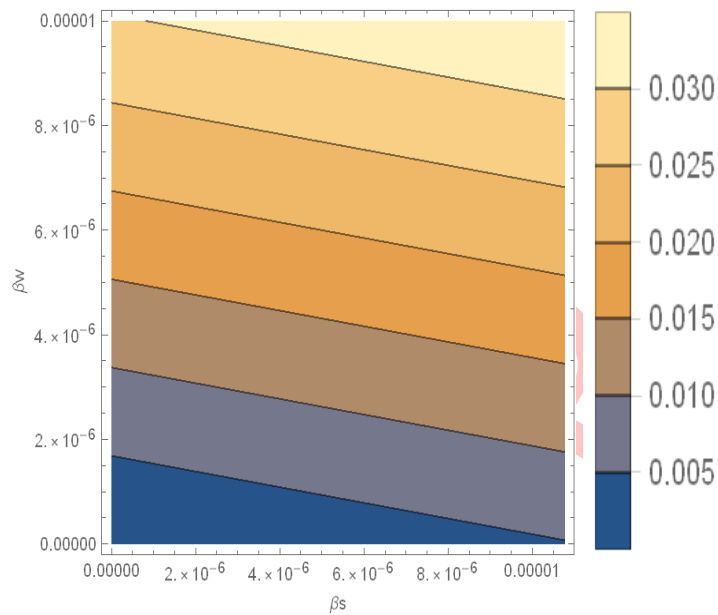
given that  $\beta_W = 0.00001$  and  $\beta_S = 0.000010778$ . Hence, if  $\beta_W = 0.0$  and  $\beta_S = 0.000010778$ , then  $\mathcal{R}_o = 0.0048$ , which will be obtained when the conventional approach is adopted, hence the rate of spread,  $\mathcal{R}_{o2} = 0.0296$  arising from the weakened-immune, are overlooked or better still, not discovered or unrecorded, though [31] reported that persons with weakened immune system can spread the disease more than the healthy individuals. In fact, that  $\mathcal{R}_o = 0.0344 (< 1)$ , shows that it is possible to eradicate the disease from the population, only if medical practitioners and researchers understand the role of weakened-immune individuals in the spread of chickenpox in our population.

Figure 2 illustrates, under the SEIWRV model, a contour plot which shows the sensitivity analysis of the basic reproduction number by varying the parameter values,  $\beta_W$  and  $\beta_S$ . It is observed that as the contact rate with a weakened immune individual increases, the spread of chickenpox infection increases sharply, and increases slightly as the contact rate with infected individual without weakened immunity. Hence, this plot will help us to understand and forecast the possible basic reproduction number, when there are changes in the contact rate of chickenpox infection by a weakened and non-weakened immune individuals, in relation to SEIWRV model, in a population.

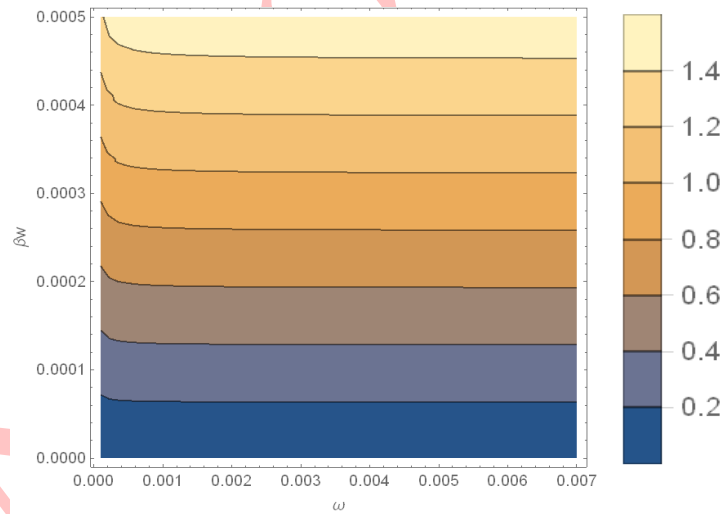
Figure 3 shows, under the SEIWRV model, a contour plot that shows the sensitivity analysis of the basic reproduction number by varying the parameter values,  $\beta_W$  and  $\omega$ . It is observed that as the contact rate with a weakened immune individual and chickenpox exposure rate increases, the spread of chickenpox infection increases tremendously. This tells us that a continuous increase in  $\beta_W$  and  $\omega$ , may lead to chickenpox endemic state in a population.

Figure 4 illustrates, under the SEIWRV model, a contour plot that demonstrates the sensitivity analysis of the basic reproduction number by varying the parameter values,  $\beta_W$  and  $\sigma$ . It is observed that as the contact rate with a weakened immune individual and rate at which infected individuals move to the weakened-immune class increases, the spread of chickenpox infection increases very sharply. This simply tells us that a continuous increase in  $\beta_W$  and  $\sigma$ , may as well lead to chickenpox endemic state in a population.

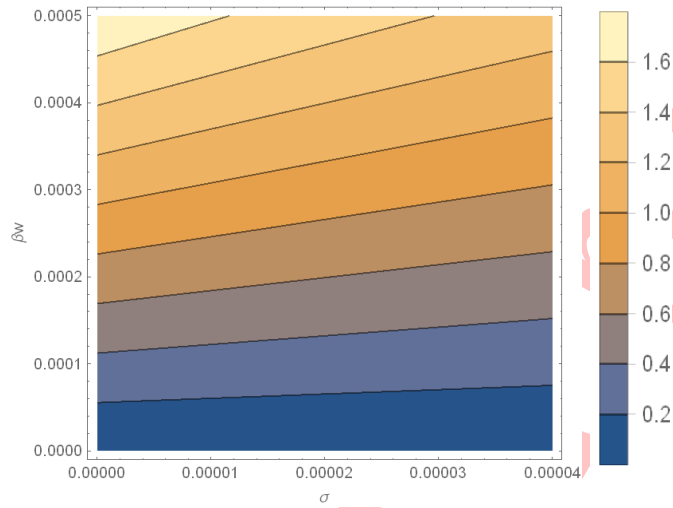
Figure 5 shows, under the SEIWRV model, a contour plot that gives the sensitivity analysis of the



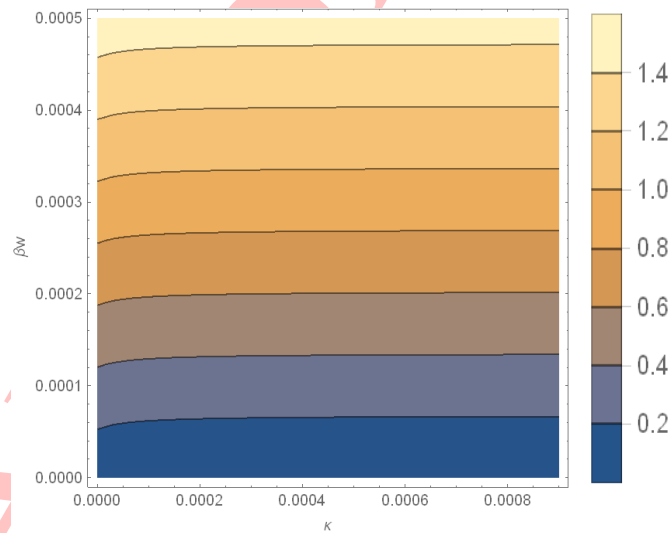
**Figure 2:** The behavior of reproduction numbers  $\mathcal{R}_0$  with respect to disease transmission of class S ( $\beta_s = \beta_s$ ) and disease transmission of class W ( $\beta_w = \beta_w$ )



**Figure 3:** The behavior of reproduction numbers  $\mathcal{R}_0$  with respect to disease transmission of class W ( $\beta_w$ ) and rate at which exposed individuals moves to the infected class,  $\omega$

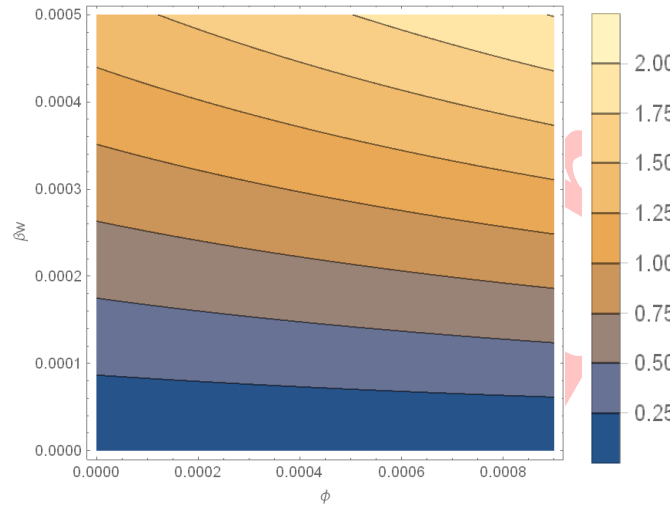


**Figure 4:** The behavior of reproduction numbers  $\mathcal{R}_o$  with respect to disease transmission of class  $W$  ( $\beta_W$ ) and weakened-immune transmission rate,  $\sigma$



**Figure 5:** The behavior of reproduction numbers  $\mathcal{R}_o$  with respect to disease transmission of class  $W$  ( $\beta_W$ ) and vaccination rate,  $\kappa$

basic reproduction number for vary  $\beta_W$  and  $\kappa$ . It is found that as the contact rate with a weakened immune individual and vaccination rate increases, the spread of chickenpox infection increases sharply. This again, tells us that a continuous increase in  $\beta_W$  and  $\kappa$ , may again lead to chickenpox endemic state in a population.



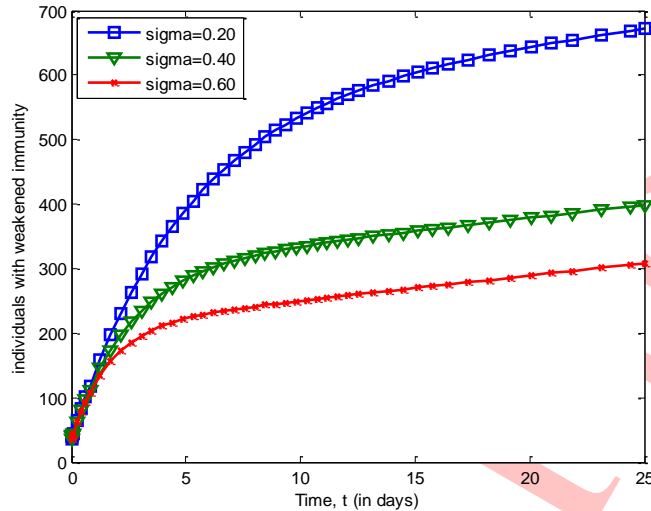
**Figure 6:** The behavior of reproduction numbers  $\mathcal{R}_0$  with respect to disease transmission of class  $W$  ( $\beta_W$ ) and rate at which vaccinated individuals moves to weakened-immune class,  $\phi$

Figure 5 shows, under the SEIWRV model, a contour plots that illustrate the sensitivity analysis of the basic reproduction number by varying the parameter values,  $\beta_W$  and  $\phi$ . It is observed that as the contact rate with a weakened immune individual and rate at which vaccinated individuals moves to weakened-immune class increases, the spread of chickenpox infection will experience monumental increases. This again, tells us that a continuous increase in  $\beta_W$  and  $\phi$ , will lead to chickenpox endemic state in a population.

Figure 7 - Figure 11 show the simulation results of the number of individuals with weakened immunity in the population for varies value of  $\sigma$  and over a period of 25 days, the number of exposed individuals in the population for varies value of  $\sigma$  and over a period of 100 days, the number of individuals with weakened immunity in the population for varies value of  $\beta_W$  and over a period of 25 days, and the number of individuals with weakened immunity in the population for varies value of  $\beta_W$  and over a period of 1000 days, respectively.

In Figure 7, which represents the simulation result of individuals with weakened immunity in the population for varies value of  $\sigma$  and for a period of 25 days, shows that as the rate at which infected individuals move to the weakened-immune class increases, the number of individuals with weakened immunity will decrease over time, and vice versa, for a period of 25 days. This shows that the rate  $\sigma$ , has an inverse effect on the growth of the weakened-immune individuals in the population for a period of 25 days, and direct effect on weakened-immune individuals in the exposed class over time.

In Figure 8, it shows the simulation result of the number of exposed individuals in the population for varies value of  $\sigma$  and over a period of 25 days. It is observed that as  $\sigma$  increases, the number of exposed individuals will decrease over time, for a period of 25 years.



**Figure 7:** The simulation result showing the number of individuals with weakened immunity in the population for varies value of  $\sigma$  and over a period of 25 days

Figure 9 which represents the simulation result of individuals with weakened immunity in the population for varies value of  $\sigma$  and for a period of 100 days, shows that as the rate at which infected individuals move to the weakened-immune class increases, the number of individuals with weakened immunity will continue to decrease over time, and vice versa, for a period of 100 days.

In Figure 10, which represents the simulation result that shows the number of individuals with weakened immunity in the population for varies value of  $\beta_W$ , for a period of 25 days. It is observed that as the contact rate between a weakened-immune individual and infected individual increases, the number of individuals with weakened immunity decreases for a period of 25 days. In

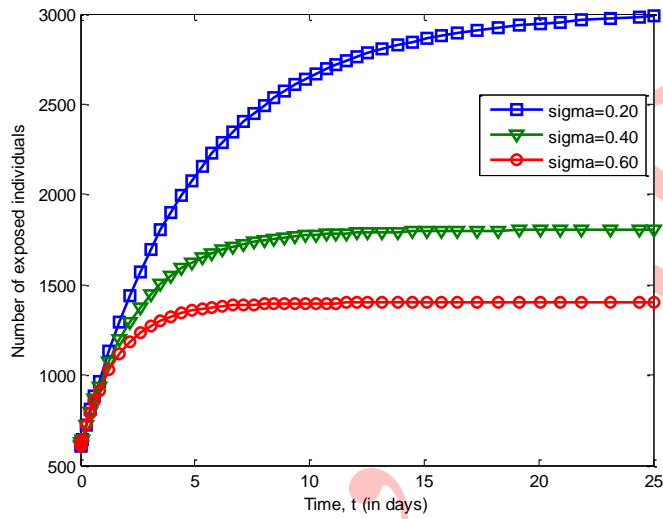
Figure 11, it is observed that for  $\beta_W = 0.00001$ , there is a shape increase in the number of individuals with weakened immunity up to about 100 days, and there is a shape decline thereafter, then remain stationary after 1000 days. When  $\beta_W = 0.0001$ , there is a shape increase in the number of weakened-immune individuals up to about 40 days, and start to decline thereafter up to about 300 days, then remain stationary thereafter. Similar interpretation goes to when  $\beta_W = 0.001$ .

## 7 Conclusion and Recommendations

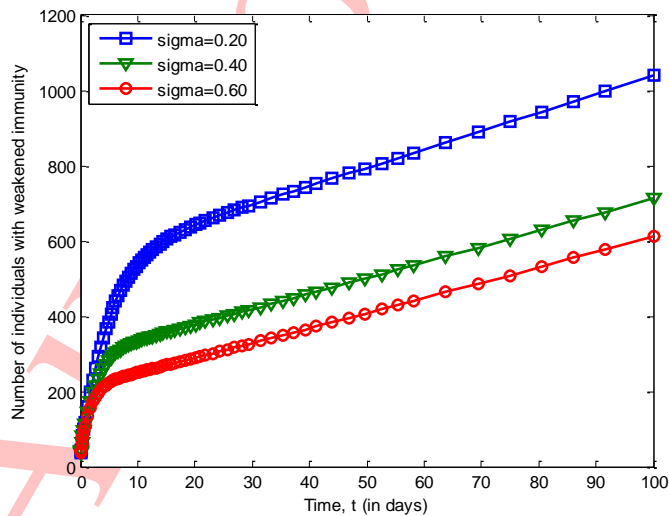
In this section, we present the concluding part of the paper as well as some clinical recommendations.

### 7.1 Concluding part of the paper

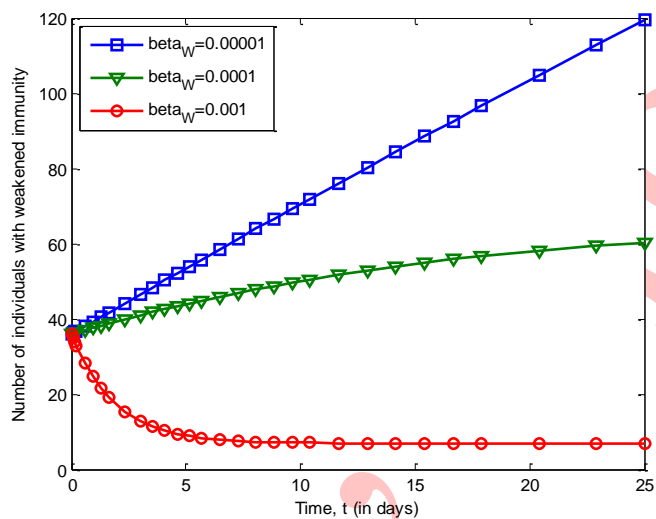
This paper studies SEIWRV dynamics of chickenpox models with vaccination, treatment and weakened-immune individuals. We carried out mathematical and stability analysis of our chickenpox disease model. The basic reproduction number of our chickenpox model was obtained. Also, the global stability of the disease-free equilibrium and the local stability of the endemic equilibrium in the feasible region of the chickenpox model were discussed, in this paper. We adopted a geometric approach to determine the



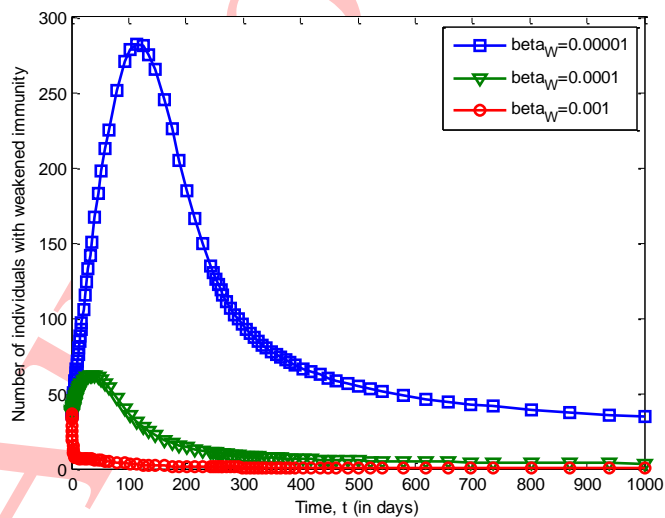
**Figure 8:** The simulation result showing the number of exposed individuals in the population for varies value of  $\sigma$  and over a period of 25 days



**Figure 9:** The simulation result showing the number of weakened-immune individuals in the population for varies value of  $\sigma$  and over a period of 100 days



**Figure 10:** The simulation result showing the number of weakened-immune individuals in the population for varies value of  $\beta_W$  and over a period of 25 days



**Figure 11:** The simulation result showing the number of weakened-immune individuals in the population for varies value of  $\beta_W$  and over a period of 1000 days

global stability of our chickenpox endemic equilibrium in a defined positive invariant set. We found from our theoretical results that  $\mathcal{R}_o$  was made up of two parts,  $\mathcal{R}_{o1}$  and  $\mathcal{R}_{o2}$ , where  $\mathcal{R}_{o1}$  is the expected number of secondary infections produced in compartment  $E$  by an infected individual who was originally in compartment  $E$ , and  $\mathcal{R}_{o2}$  is the expected number of secondary infections produced in compartment  $E$  by an infected individual who was originally in compartment  $W$ . It was observed that chickenpox will continue to have additional reproduction rate  $\mathcal{R}_{o2}$ , as long as the weakened-immune individuals remain in the population, and unattended to.

Using the real data for chickenpox outbreak in Phuket Province, Thailand as presented in Jose *et.al* (2023), the following study results were found:

- The basic reproduction number produced by a weakened-immune individual tremendously increase the spread of chickenpox in Phuket Province, Thailand. We found that as the contact rate of a weakened-immune person and an infected person increases, the basic reproduction number increases sharply. It therefore implies that, to reduce the spread of chickenpox in Phuket Province, Thailand, contact rate of an infected person and a weakened-immune person should be reduced or possibly, prevented.
- It was found that a weakened-immune individual has a direct relationship with the basic reproduction number. Therefore, an increase in the number of weakened-immune individuals will lead to increase in the spread of chickenpox in Phuket Province, Thailand.
- It was also observed that vaccination against chickenpox infections has a direct relationship with the basic reproduction number. Hence, an increase in the number of chickenpox vaccinations in Phuket Province, will lead to decrease in the number of infection of persons in the Province.
- It was also found that the rate at which vaccinated individuals developed weakened-immune system will go a long way in increasing the spread of chickenpox in Phuket Province.

By studying the mathematical model of the spread of chickenpox in the presence of weakened-immune individuals, the model helps the medical practitioners and researchers to have thorough understanding of the factors that have resulted to the continuous outbreak of this disease, even in the presence of vaccines, as well as the factor that has made the disease to persist in our population for such a long time. Therefore, this mathematical model and the results arising therefrom, will give the stakeholders a new way to look at the control and management of the outbreak of this disease, by reducing the numbers of weakened-immune individuals in the population.

Many control and management strategies, such as vaccination, treatment, quarantine, isolation, e.t.c, have been adopted in trying to reduce, even eradicate chickenpox from the society, still the disease persist. This paper has identified the major factor that perhaps, made the disease to persist for such a long time.

The results show that chickenpox will continue to persist in our population as long as individuals with weakened-immune system are still in the population, and if no actions are taken.

## 7.2 Recommendations

To reduce or possibly eradicate chickenpox in our society, we therefore recommend clinically, the following:

- Individuals with weakened-immunity should be identified in the population;
- Their immune should be boosted periodically to ensure that they have stronger immunity to keep the virus inactive in their body tissues;
- Since it is possible for an individual who has a strong immune system or who has been vaccinated, to later develops weakened immunity, medical practitioners should ensure that more attention is paid to people's immune system at all time, and require medications are recommended for anyone with weakened immunity.

## Conflicts of interest

The authors declare that there are no conflicts of interest.

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