Flinders University

College of Science and Engineering

A mathematical model for seasonal patterns of Dengue fever in Laos

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Declaration

I certify that this thesis does not incorporate without acknowledgment any material previously submitted for a degree or diploma in any university; and that to the best of my knowledge and belief it does not contain any material previously published or written by another person except where due reference is made in the text.

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Abstract

Dengue fever has been a health problem for many decades especially in tropical and sub-tropical countries. Laos is a one of the countries where a dengue epidemic has been declared twice in the current decade; in 2010 and 2013. This paper states the concerns whether the seasonal pattern influence the disease transmission in this country. A mathematical dynamic methodology is used in this paper to indicate how the spreading of dengue fever is influenced by seasonal patterns. This thesis will be based on the model of research study from (Ding et al., 2012) only is considered because as it has similar focusing area as this paper aims to present. However, the study of (Ding et al., 2012) considered multiple groups, no explicit formula for equilibria were given and the basic reproduction number was not computed. In this thesis, only one group is considered, but explicit formulae for equilibria are found as the basic reproduction number R_0. Numerical simulations demonstrate the seasonal influence

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Chapter 1

Introduction

1.1 Research Problem

Dengue fever is a well-established disease and found in many countries especially in tropical and subtropical regions. According to the World Health Organisation (WHO, 2018b), there are 4 types of dengue fever (DEN1-4); all types are transmitted to humans by a bite from the Aedes mosquitos (female mosquitos). Over 40 percent of the worlds population or more than 2.5 billion people who are currently living in Africa, Central and South America, the Caribbean, the Eastern Mediterranean, South and Southeast Asia, and Oceania are at the highest risk of this vector-born-disease. It is estimated that each year, there are 50 to 100 million cases worldwide of dengue infection. Over 500 thousand of these cases develop into the severe dengue type known as haemorrhagic fever, resulting in up to 12,000 deaths annually worldwide. Laos is one of the countries where dengue fever is a common public health problem (Sayavong, 23, January 2018). As illustrated in Figure 1.1., the number of people infected with dengue in Laos capital city (Vientiane) varies seasonally and between years, the latest epidemic outbreak occuring in the year 2013 (Sayavong, 23, January 2018).

According to (WPRO, 2018), in 2010 there were 22,890 reported cases of dengue fever in Laos and in 2013, there were 44,171 reported cases (approximately 10,471 cases in Vientiane), showing a general increasing trend over the years (Sayavong, 23, January 2018).This presents an alerting situation for the Lao PDR representative, Dr Juliet Fleischl, who expresses concern of another epidemic outbreak and advises there is crucial need for prevention and monitoring methods (WHO, 2018a).

Mathematical models are useful for short to medium length prediction

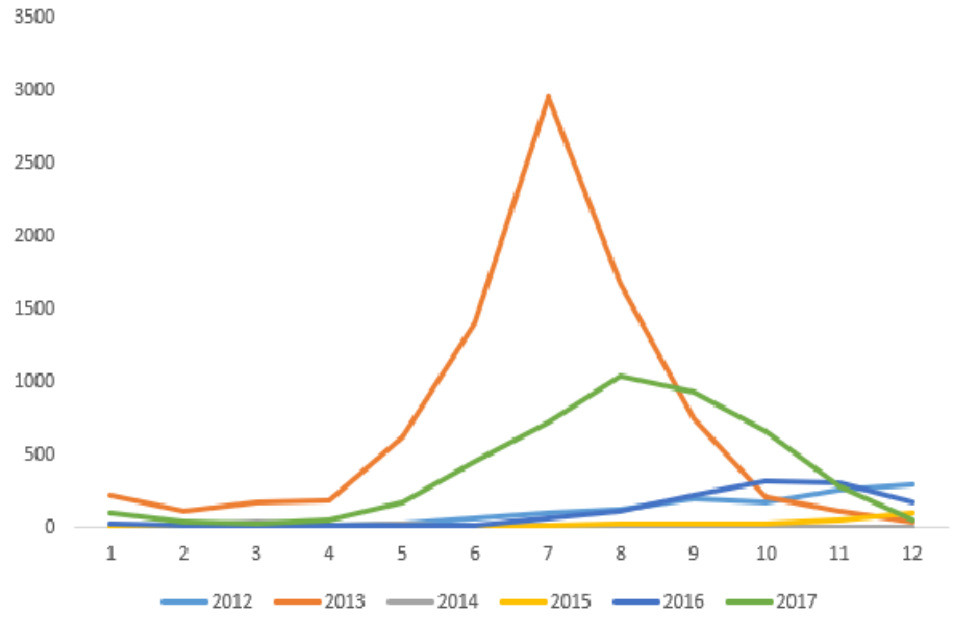

Figure 1.1: The number of patients from 2012 to 2017 in Vientiane

of the number of new infections. The model in this project is used to predict how the seasonal pattern will influence the spreading of dengue fever and the number of patients, focusing in Vientiane. This research will be useful for planning strategies to manage and possibly reduce the severity of outbreaks.

1.2 Lao Context

Laos is located in South East Asia and is the only landlocked country in this region. Bordering countries include Myanmar (Burma) and China to the northwest, Vietnam to the east, Cambodia to the southwest, and Thailand to the west and southwest. Laos contains 17 provinces and one prefecture (Vientiane, the capital city) and has a population of approximately 6,900,000 persons (UNFPA, 2018). The climate is tropical and influenced by the monsoon pattern, and there are two seasons; a rainy season from May to November and a dry season from December to April. Temperature depends on altitude, and Laos encompasses a variety of colder climate mountainous

and upland plateaux terrain, as well as flat plains (such as in Vientiane) where temperatures are usually between 22 to 29 degrees Celsius throughout the year. Mosquito larvae require rainfall to develop, with temperature being a dependent variable, hence according to (Valdez et al., 2018), climate influences mosquito population growth. We can imply that Vientiane is a suitable location for mosquito survival as its temperature is not too cold and not too hot with abundant rainfall throughout most of the year (Figure 1.2).

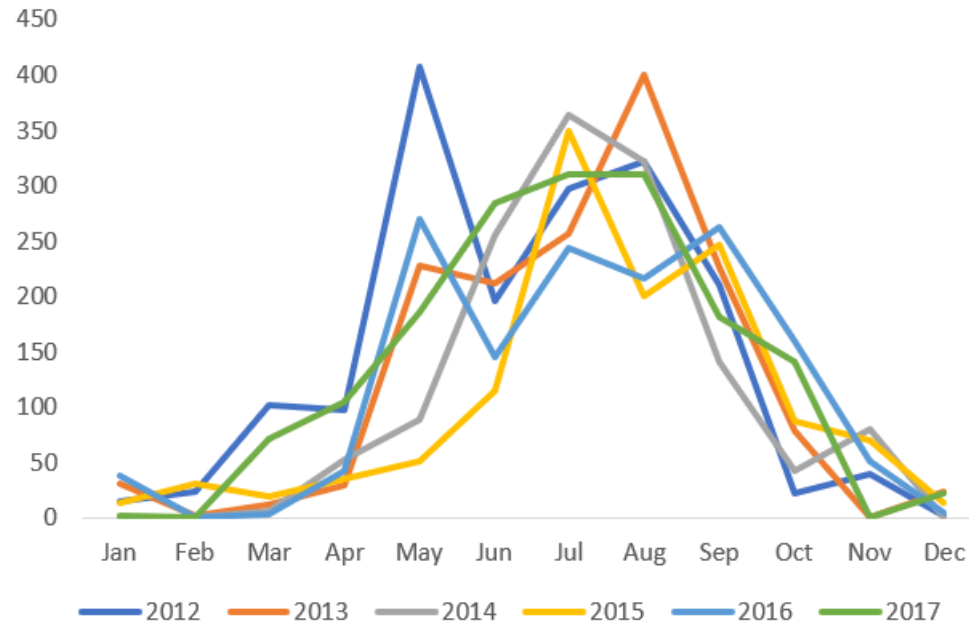

Figure 1.2: The amount of rainfall in mm from 2012 to 2017 in Vientiane (Meteorology/Hydrology, March, 2018)

1.3 Research aim and question

The thesis aims to formulate the equilibria points, the basic reproduction number R_0) and also to investigate whether a mathematical model for seasonal pattern can predict the spread of dengue in Vientiane, Laos. To achieve these aims, one question is formulated: can the mathematical model for seasonal pattern predict the spread of dengue in Vientiane? This research will focus on Vientiane, where the number of cases of dengue fever is higher than in the province. Emphasising the use of Vientiane is also necessary due to

the lack of available data from other Laos provinces required to test the feasibility of the mathematical model.

1.4 Literature review

There are many mathematicians or mathematics related authors who have done research regarding vector-borne diseases (Abdelrazec et al., 2016), (Cai et al., 2009), (Ding et al., 2012), (Esteva and Vargas, 1998). These diseases are infections transmitted by the bite of infected arthropod species such as mosquitoes as in the case of dengue fever. The analysis of a dengue disease transmission model involves finding the conditions that the number of humans and vectors are stable. In relation to this, the basic reproduction number (R_0) can be considered, where the expected average number (not rate) of secondary cases that result from a primary case in a fully susceptible population is tested to generally characterise control of the disease (Rodrigues et al., 2016). The study of (Esteva and Vargas, 1998) found that if the basic reproduction number is less than one, the disease-free state is asymptotically stable and if the basic production number is greater than one, the disease endemic state is globally asymptotically stable. Similarly, using a graph theory method to demonstrate the dengue disease transmission, a study conducted by (Ding et al., 2012) points out that the global stability of a multigroup dengue disease transmission model also supports the result of the previous study. However, it can only be applied to groups of populations. This means that if the basic reproduction number is less than one, the disease dies out in all groups and if the basic reproduction number is greater than one, the same endemic state is reached in all groups.

Furthermore, the global dynamics of dengue epidemic mathematical model also has similar investigation and set human and vectors to be constant. (Cai et al., 2009) used an SIR model for dynamics of dengue disease with saturation and bilinear incidence to determine the basic reproduction. In addition to that, (Abdelrazec et al., 2016) set a model of the spreading and controlling of dengue fever with limited public health resources where the main results of the paper were to indicate the existence of multiple endemic equilibria and show the phenomenon of backward bifurcation.

Another study from Thailand attempts to investigate how entomological and biological variables influence dengue by setting mathematical models regarding to seasonal varying parameters, predicting the prevalence of infection, and then comparing it to observe the seasonal patterns of the disease.

The papers listed above use different methods to investigate the basic reproductive disease (R_0) and an equilibria. This thesis will be based on the research study from (Ding et al., 2012) and use mathematical dynamic method to analyse the model for seasonal patterns of dengue fever in Laos because the study of (Ding et al., 2012) considered multiple groups, no explicit formula for equilibria were given and the basic reproduction number was not computed. In this thesis, only one group is considered, but explicit formulae for equilibria are found as is the basic reproduction number R_0 This thesis mainly focuses on how the system in this model work and show the disease-free equilibrium and the endemic equilibrium points then making a comparation with data from Laos with the numerical result.

This research paper is divided into four chapters. In Chapter 1, the introduction part provides some background of the research problem regarding to Dengue fever, brief discussing Lao context, and then the problem interests regarding to this researchs concerns and literature reviews. In chapter 2, introducing a mathematical model and providing the mathematical terminology in terms of biological denotation such as state variables. In further section, a mathematical model analysis using dynamic methodology to obtain the result whether the system in the model has a disease-free equilibrium point and an endemic equilibrium. In the chapter 3, the result discussion from the model analysis in chapter 2 and compare with data from Laos to see whether the model is adequate to the situation in Laos. The final chapter, chapter 4 provides some better approaches to Laos community, some limitations and future research suggestions.

Chapter 2

A new model for spreading dengue fever

2.1 Methodology

The dynamic method will be used to model dengue fever in Laos and it will be a sequence of autonomous models. This will allow us to compute equilibrium, determine stability and determine the formula for the basic reproduction number.

2.2 Setting the model

The model is a deterministic compartmental model and there are five state variables:

S the number of susceptible humans per unit area.
I the number of infected humans per unit area.
R the number of recovered humans per unit area.
V the number of susceptible vectors per unit area.
G the number of infected vectors per unit area.

In this paper, the essential elements of a mathematical model for the transmission of an infectious disease is represented as a SIRVG model. There are two groups including humans and vectors. The human population is divided into three compartments: Susceptible humans are recruited at a rate of P persons per unit time (per unit area), where recruitment may involve birth or immigration from other areas. The number of susceptible humans that become infectious through contact with an infected vector per unit time is αSG

and the number of natural deaths is δS. Infected humans are not modelled as having an increased mortality rate. Although some people do die of dengue, this is a small number and denoted as $(\delta + \beta)I$. The number of recovered humans is βI and to the number of deaths of recovered humans is denoted as δR. Further, the vector group is set into two compartments: susceptible vectors are recruited at a rate of Q vectors per unit time (per unit area). The number of susceptible vectors that become infectious through contact with an infected persons per unit time is αVI and the number of natural deaths is θV. θG is represented as the mortality of infected vectors. This leads to the following system of differential equations.
The system of differential equations:

$$\begin{aligned} S' &= P - \alpha SG - \delta S && (2.1)\\ I' &= \alpha SG - (\delta + \beta)I && (2.2)\\ R' &= \beta I - \delta R && (2.3)\\ V' &= Q - \gamma VI - \theta V && (2.4)\\ G' &= \gamma VI - \theta G && (2.5) \end{aligned}$$

Where, P is referred to the recruitment rate of humans, the mortality rate for humans denoted as δ, β is the recovery rate for humans, the infection rate of human from vectors denoted as α, γ is the infection rate of vectors from humans, θ is denoted as the mortality rate of vectors, Q is referred to the recruitment rate of vector. Hence, P, Q , δ and θ are bigger than 0 and the rests of all values are supposed to be nonnegative.

2.3 Equilibria

From Equations 2.1 - 2.5, points of equilibrium must satisfy the following equations.

$$\begin{aligned} 0 &= P - \alpha SG - \delta S && (2.6)\\ 0 &= \alpha SG - (\delta + \beta)I && (2.7)\\ 0 &= \beta I - \delta R && (2.8)\\ 0 &= Q - \gamma VI - \theta V && (2.9)\\ 0 &= \gamma VI - \theta G && (2.10) \end{aligned}$$

2.3.1 The disease-free equilibrium

A system is said to have a disease-free equilibrium if there is a point of equilibrium in the state space with no infected individuals, other words, with $I = 0$. Therefore, by Equations (2.6) and (2.9), neither S nor V can be zero since P and Q are fixed positive constants.

If $I = 0$, then by (2.8), $R = 0$ and by (2.10), $G = 0$. Then by (2.6), $S = P/\delta$ and by (2.9), $V = Q/\theta$.

Thus there is a disease free equilibrium at

$$\left(\frac{P}{\delta}, 0, 0, \frac{Q}{\theta}, 0\right). \tag{2.11}$$

If $G = 0$, then since $V \neq 0$, Equation (2.10) gives that $I = 0$ and so the disease free equilibrium in (2.11) results.

If $R = 0$, then by (2.8), $I = 0$ and so the disease free equilibrium is found again.

Thus the only equilibria other than the disease free equilibrium in (2.11) must have all postive values for the state variables.

2.3.2 The disease endemic equilibrium

A system is said to have a disease endemic equilibrium if there is a point of equilibrium in the state space, where all state variables have positive values. Therefore, in order to obtain all positive state variables, from equations (2.8) and (2.10) give that

$$I = \frac{\delta}{\beta}R \qquad \text{and} \qquad G = \frac{\gamma}{\theta}IV = \frac{\gamma\delta}{\theta\beta}RV. \tag{2.12}$$

The three remaining equations (2.6, 2.7 and 2.9) become

$$0 = P - \frac{\alpha\gamma\delta}{\theta\beta}SRV - \delta S \tag{2.13}$$

$$0 = \frac{\alpha\gamma\delta}{\theta\beta}SRV - \frac{(\delta+\beta)\delta}{\beta}R \tag{2.14}$$

$$0 = Q - \frac{\gamma\delta}{\beta}RV - \theta V \tag{2.15}$$

Solving Equation (2.15) for R gives

$$R = \frac{\beta}{\gamma\delta}\left(\frac{Q}{V} - \theta\right). \tag{2.16}$$

With this, formula for R, Equation (2.13)becomes

$$0 = P - \frac{\alpha S}{\theta}(Q - \theta V) - \delta S. \tag{2.17}$$

Equation (2.14) may be rewritten as

$$V = \frac{\theta(\delta + \beta)}{\alpha\gamma S}. \tag{2.18}$$

Substituting (2.18) into (2.17) gives

$$0 = P - \frac{\alpha}{\theta} S \left(Q - \frac{\theta^2(\delta + \beta)}{\alpha\gamma S} \right) - \delta S$$

and so

$$0 = P - \frac{\alpha}{\theta} SQ + \frac{\theta(\delta + \beta)}{\gamma} - \delta S.$$

Solving this last expression for S gives

$$S = \frac{\theta}{\gamma} \left(\frac{P\gamma + \theta(\delta + \beta)}{\alpha Q + \delta\theta} \right).$$

Now using (2.18), (2.16) and the formulas in (2.12), formulas for all the state variables may be found. The disease endemic equilibrium is at

$$S = \frac{\theta}{\gamma} \left(\frac{P\gamma + \theta(\delta + \beta)}{\alpha Q + \delta\theta} \right) \tag{2.19}$$

$$V = \frac{(\delta + \beta)}{\alpha} \left(\frac{\alpha Q + \delta\theta}{P\gamma + \theta(\delta + \beta)} \right) \tag{2.20}$$

$$R = \frac{\beta}{\gamma\delta} \left(\frac{Q\alpha(P\gamma + \theta(\delta + \beta)}{(\delta + \beta)(\alpha Q + \delta\theta)} - \theta \right) \tag{2.21}$$

$$I = \frac{1}{\gamma} \left(\frac{Q\alpha(P\gamma + \theta(\delta + \beta)}{(\delta + \beta)(\alpha Q + \delta\theta)} - \theta \right) \tag{2.22}$$

$$G = \frac{1}{\theta} \left(\frac{Q\alpha(P\gamma + \theta(\delta + \beta)}{(\delta + \beta)(\alpha Q + \delta\theta)} - \theta \right) \tag{2.23}$$

2.4 The Jacobian and the characteristic polynomial

Equilibria are defined as points in the state space where Equations 2.1 - 2.5 are zero. The stability of the equilibria are determined by the signs of the eigenvalues (λ) of the matrix $J - \lambda I_d$, where J is the Jacobian matrix and I_d is the identity matrix.
The general n dimensional Jacobian is

$$J(x_1, x_2, \ldots, x_n) = \begin{pmatrix} \frac{\partial f_1}{\partial x_1} & \frac{\partial f_1}{\partial x_2} & \cdots & \frac{\partial f_1}{\partial x_n} \\ \frac{\partial f_2}{\partial x_1} & \frac{\partial f_2}{\partial x_2} & \cdots & \frac{\partial f_2}{\partial x_n} \\ \vdots & \vdots & & \vdots \\ \frac{\partial f_n}{\partial x_1} & \frac{\partial f_n}{\partial x_2} & \cdots & \frac{\partial f_n}{\partial x_n} \end{pmatrix}.$$

For the model from equations 2.1 to 2.5, the Jacobian is

$$J(S, I, R, V, G) = \begin{pmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial R} & \frac{\partial f_1}{\partial V} & \frac{\partial f_1}{\partial G} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial R} & \frac{\partial f_2}{\partial V} & \frac{\partial f_2}{\partial G} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial R} & \frac{\partial f_3}{\partial V} & \frac{\partial f_3}{\partial G} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial R} & \frac{\partial f_4}{\partial V} & \frac{\partial f_4}{\partial G} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial R} & \frac{\partial f_5}{\partial V} & \frac{\partial f_5}{\partial G} \end{pmatrix}.$$

Hence,

$$J - \lambda I_d = \begin{pmatrix} -(\alpha G + \delta) - \lambda & 0 & 0 & 0 & -\alpha S \\ \alpha G & -(\delta + \beta) - \lambda & 0 & 0 & \alpha S \\ 0 & \beta & -\delta - \lambda & 0 & 0 \\ 0 & -\gamma V & 0 & -(\gamma I + \theta) - \lambda & 0 \\ 0 & \gamma V & 0 & \gamma I & -\theta - \lambda \end{pmatrix}. \tag{2.24}$$

2.4.1 Stability of the disease free equilibrium

At a disease free equilibrium (2.11)and setting $I = 0$, $R = 0$ and $G = 0$ and plug into equation (2.24) gives

$$J - \lambda I_d = \begin{pmatrix} -\delta - \lambda & 0 & 0 & 0 & -\alpha S \\ 0 & -(\delta + \beta) - \lambda & 0 & 0 & \alpha S \\ 0 & \beta & -\delta - \lambda & 0 & 0 \\ 0 & -\gamma V & 0 & -\theta - \lambda & 0 \\ 0 & \gamma V & 0 & 0 & -\theta - \lambda \end{pmatrix}. \tag{2.25}$$

In this case,

$$\begin{aligned}
\det(J-\lambda I_d) &= (-\delta-\lambda)\det\begin{pmatrix} -(\delta+\beta)-\lambda & 0 & 0 & \alpha S \\ \beta & -\delta-\lambda & 0 & 0 \\ -\gamma V & 0 & -\theta-\lambda & 0 \\ \gamma V & 0 & 0 & -\theta-\lambda \end{pmatrix}. \\
&= (-\delta-\lambda)\left[(-(\delta+\beta)-\lambda)\det\begin{pmatrix} -\delta-\lambda & 0 & 0 \\ 0 & -\theta-\lambda & 0 \\ 0 & 0 & -\theta-\lambda \end{pmatrix}\right. \\
&\quad \left. -\alpha S\ \det\begin{pmatrix} \beta & -\delta-\lambda & 0 \\ -\gamma V & 0 & -\theta-\lambda \\ \gamma V & 0 & 0 \end{pmatrix}\right] \\
&= (-\delta-\lambda)\left[(-\delta-\beta-\lambda)(-\delta-\lambda)(-\theta-\lambda)(-\theta-\lambda)\right. \\
&\quad \left. -\alpha S(\gamma V(-\theta-\lambda)(-\delta-\lambda)\right]. \\
&= (-\delta-\lambda)^2(-\theta-\lambda)\left[(-\delta-\beta-\lambda)(-\theta-\lambda)-\alpha\gamma SV\right] \\
&= (-\delta-\lambda)^2(-\theta-\lambda)\left(\lambda^2+(\delta+\beta+\theta)\lambda+\theta(\delta+\beta)-\frac{\alpha\gamma PQ}{\delta\theta}\right)
\end{aligned}$$

Thus $\lambda_1=-\delta$, $\lambda_2=-\delta$, $\lambda_3=-\theta$ and λ_4 and λ_5 are given by

$$\frac{1}{2}\left[-(\delta+\beta+\theta)\pm\sqrt{(\delta+\beta+\theta)^2-4\left(\theta(\delta+\beta)-\frac{\alpha\gamma PQ}{\delta\theta}\right)}\right]. \qquad (2.26)$$

Thus all the eigenvalues have negative real parts if and only if

$$\theta(\delta+\beta)-\frac{\alpha\gamma PQ}{\delta\theta}>0.$$

Thus the disease free equilibrium is stable if and only if

$$\frac{\alpha\gamma PQ}{\delta\theta^2(\delta+\beta)}<1.$$

This indicates that the basic reproduction number is

$$R_0=\frac{\alpha\gamma PQ}{\delta\theta^2(\delta+\beta)}.$$

2.4.2 Stability of the disease endemic equilibrium

At the disease endemic equilibrium point, we get the result by using numerical analysis as the steps below.

Define the following quantities.

$$A = \alpha G + \delta, \qquad C = \alpha S, \qquad D = \alpha G, \qquad E = \delta + \beta,$$

$$F = \gamma V, \qquad H = \gamma I + \theta, \qquad K = \gamma I.$$

Then

$$J - \lambda I_d = \begin{pmatrix} -A-\lambda & 0 & 0 & 0 & -C \\ D & -E-\lambda & 0 & 0 & C \\ 0 & \beta & -\delta-\lambda & 0 & 0 \\ 0 & -F & 0 & -H-\lambda & 0 \\ 0 & F & 0 & K & -\theta-\lambda \end{pmatrix}. \tag{2.27}$$

The last column will be used to decompose the determinant of $J - \lambda I_d$ into minors.

$$\begin{aligned} \det(J-\lambda I_d) &= -C\det\begin{pmatrix} D & -E-\lambda & 0 & 0 \\ 0 & \beta & -\delta-\lambda & 0 \\ 0 & -F & 0 & -H-\lambda \\ 0 & F & 0 & K \end{pmatrix} \\ &\quad -C\det\begin{pmatrix} -A-\lambda & 0 & 0 & 0 \\ 0 & \beta & -\delta-\lambda & 0 \\ 0 & -F & 0 & -H-\lambda \\ 0 & F & 0 & K \end{pmatrix} \\ &\quad -(\theta+\lambda)\det\begin{pmatrix} -A-\lambda & 0 & 0 & 0 \\ D & -E-\lambda & 0 & 0 \\ 0 & \beta & -\delta-\lambda & 0 \\ 0 & -F & 0 & -H-\lambda \end{pmatrix} \end{aligned} \tag{2.28}$$

Expanding each of these 4×4 determinants,

$$\begin{aligned} det(J-\lambda I_d) &= -CD\det\begin{pmatrix} \beta & -\delta-\lambda & 0 \\ -F & 0 & -H-\lambda \\ F & 0 & K \end{pmatrix} \\ &\quad +C(A+\lambda)\det\begin{pmatrix} \beta & -\delta-\lambda & 0 \\ -F & 0 & -H-\lambda \\ F & 0 & K \end{pmatrix} \\ &\quad -(\theta+\lambda)(A+\lambda)(E+\lambda)(\delta+\lambda)(H+\lambda). \end{aligned} \tag{2.29}$$

The two matrices in Equation (2.29) are identical and so

$$\begin{aligned}\det(J-\lambda I_d) &= C(A-D+\lambda)\det\begin{pmatrix}\beta & -\delta-\lambda & 0\\ -F & 0 & -H-\lambda\\ F & 0 & K\end{pmatrix} \\ &\quad -(\theta+\lambda)(A+\lambda)(E+\lambda)(\delta+\lambda)(H+\lambda)\end{aligned} \tag{2.30}$$

and so

$$\begin{aligned}\det(J-\lambda I_d) &= C(A-D+\lambda)\left[F(H+\lambda)(\delta+\lambda)-FK(\delta+\lambda)\right] \\ &\quad -(\theta+\lambda)(A+\lambda)(E+\lambda)(\delta+\lambda)(H+\lambda). \end{aligned} \tag{2.31}$$

Thus

$$\begin{aligned}\det(J-\lambda I_d) &= CF(A-D+\lambda)(\delta+\lambda)(H-K+\lambda) \\ &\quad -(\theta+\lambda)(A+\lambda)(E+\lambda)(\delta+\lambda)(H+\lambda). \end{aligned} \tag{2.32}$$

This shows that $\det(J-\lambda I_d) = (\delta+\lambda)(W-Z)$, where W and Z are the polynomials

$$\begin{aligned} W &= CF(A-D+\lambda)(H-K+\lambda) \\ &= CF\left[\lambda^2+(A-D+H-K)\lambda+(AH-AK-DH+DK)\right] \qquad (2.33)\\ Z &= (\theta+\lambda)(A+\lambda)(E+\lambda)(H+\lambda). \qquad (2.34)\end{aligned}$$

The polynomial Z may be expanded as

$$Z = \lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0, \tag{2.35}$$

where

$$\begin{aligned} a_4 &= 1 \qquad (2.36)\\ a_3 &= A+E+H+\theta \\ a_2 &= AE+AH+A\theta+EH+E\theta+H\theta \\ a_1 &= AEH+AE\theta+AH\theta+EH\theta \\ a_0 &= AEH\theta \end{aligned}$$

With these formulas for W and Z it is possible to write $\det(J-\lambda I_d)$ in the form

$$\det(J-\lambda I_d) = (\delta+\lambda)(W-Z) = b_5\lambda^5+b_4\lambda^4+b_3\lambda^3+b_2\lambda^2+b_1\lambda+b_o. \tag{2.37}$$

Where, there are explicit formulas for b_i

$$
\begin{aligned}
b_5 &= 1 \qquad (2.38)\\
b_4 &= A+E+H+\theta+\delta\\
b_3 &= (AE+AH+A\theta+EH+E\theta+H\theta-CF)+(A+E+H+\theta)\delta\\
b_2 &= (AEH+AE\theta+AH\theta+EH\theta-CFA+CFD-CFK+CFH)\\
&\quad +(AE+AH+A\theta+EH+E\theta+H\theta-CF)\delta\\
b_1 &= AEH\theta+CFAK-CFAH-CFDK+CFDH+((AEH+AE\theta+AH\theta\\
&\quad +EH\theta-CFA+CFD-CFK+CFH)\delta\\
b_0 &= (AEH\theta+CFAK-CFAH-CFDK+CFDH)\delta \qquad (2.39)
\end{aligned}
$$

The advantage of this form of the characteristic polynomial is that Routh Hurwitz method can be applied to find the number of eigenvalues with negative real parts.

Chapter 3

Experiments

3.1 Numerical implementation

Numerical implementation requires knowledge of values of the parameters in the model (equations 2.1 - 2.5). The ranges of the parameter values used here appear in Table 3.1.

Table 3.1: A set of parameters

Parameter	Description	Value	Reference
P	recruitment rate for humans	0.0001	Assumed
Q	recruitment rate for vectors	0.1–0.5	Assumed
δ	mortality rate for humans	0.0001	(Abdelrazec et al., 2016)
β	recovery rate for humans	0.01–0.10	(Abdelrazec et al., 2016)
α	infection rate: humans from vectors	0.01–0.03	(Abdelrazec et al., 2016)
γ	infection rate: vectors from humans	0.01–0.03	(Abdelrazec et al., 2016)
θ	mortality rate for vectors	0.02–0.07	(Abdelrazec et al., 2016)

Matlab was used to compute 10,000 numerical solutions to the system in equations 2.1 - 2.5 with parameter values selected randomly from the ranges in table 3.1 The code appears in the appendix A. To analyse the model in this paper, some parameters such as the recruitment rate for of humans, rate of vectors are not well-known. Thus, ranges for those parameters are estimated respectively with the values of 0.0001, 0.1-0.5 (assumed values). For the parameters of recovery rate, mortality rate for humans, mortality rate for vectors, infection rate of human from vectors, and infection rate of vectors from humans are 0.01-0.10, 0.0001, 0.02-0.07, 0.01-0.03, and 0.01-0.03 respectively (Abdelrazec et al., 2016).

A Matlab program called **Equilibria.m** was written to compute numerical values for the disease free and the disease endemic equilibria using the formulas for these equilibria in Section 2.3. The characteristic polynomial for the Jacobian of the system is then calculated using the formulas in Section2.4 The coefficients of the characteristic polynomial are then passed to a program called **RouthTable.m** to decide if the disease endemic equilibrium is stable or not. A program called **ModelExperiment.m** was written to input various variations of the input parameters to see if the stability of the equilibria is robust to the parameters or not.

3.2 Results

The thesis research formulates the basic reproduction R_0 and also found the formulae for the disease-free equilibrium as in the section 2.3.1 and the endemic equilibrium in the section 2.3.2 and this thesis also obtains the aim that how the seasonal patterns influence the dengue. Therefore, numerical simulations as in the figure 3.1 and 3.2 are invented to see if the same model could explain the dengue fever in Laos influenced by the seasonal patterns. This can be demonstrated by setting some parameters values as in table 3.2 and the recruitment rates of vectors (mosquitos), denoted Q. The recruitment rate as $Q = 0.1$ and $Q = 0.5$ refers to dry and rainy seasons respectively. The computation results correspond to the data collected from Laos as in figure 1.1 and figure 1.2.

Table 3.2: A set of fixed parameters

Parameter	Description	Value
P	recruitment rate for humans	0.0001
Q	recruitment rate for vectors	0.1–0.5
δ	mortality rate for humans	0.0001
β	recovery rate for humans	0.05
α	infection rate: humans from vectors	0.02
γ	infection rate: vectors from humans	0.02
θ	mortality rate for vectors	0.045

This thesis model can be just used as a guide model in order to obtain the best fit for the influence of seasonal patterns to dengue fever. Therefore, with the result of computation at figure 3.1, this is an example of how dengue fever spreads over 200 days by using R program to compute to see the scenario as in the appendix B, because some parameters rates must be current each year

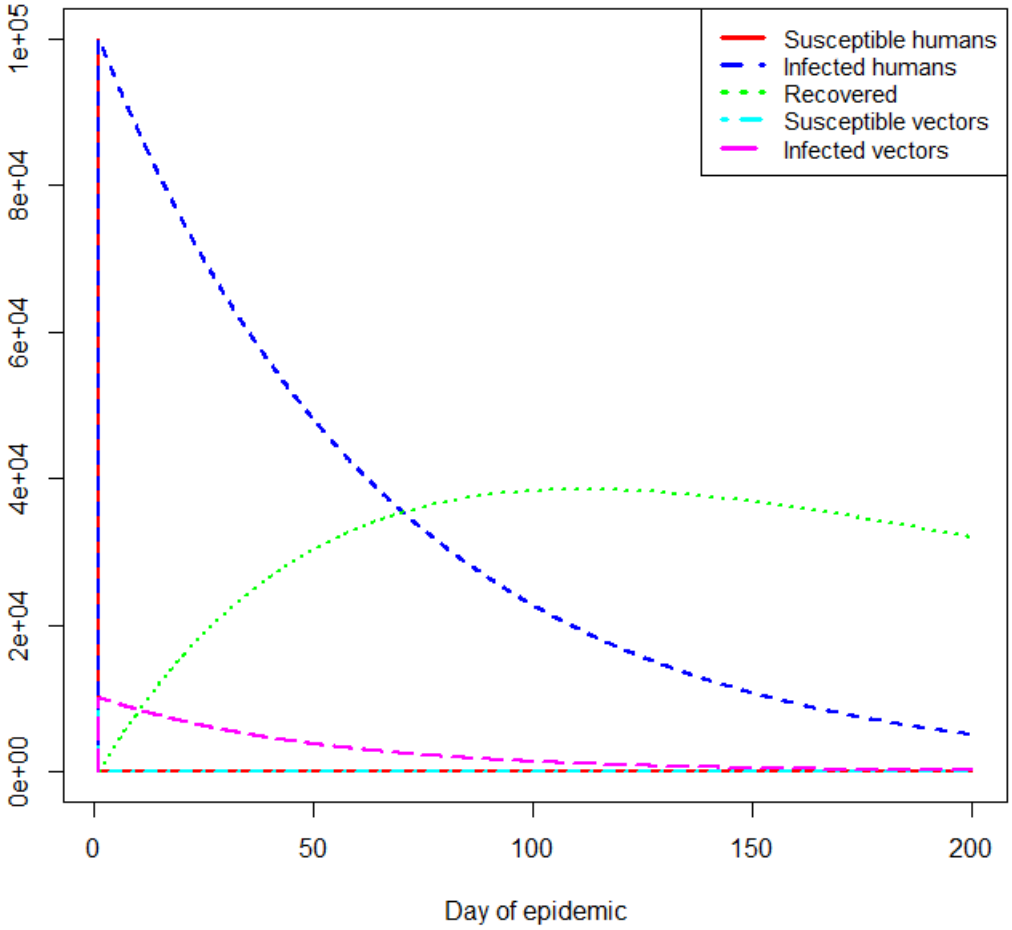

Figure 3.1: An example of state variables value over time, the result with $Q = 0.5$ and the initial values of $S_0 = 100,000, I_0 = 10, R_0 = 0, V_0 = 1,000, G_0 = 100$ for the state variables during high rainfall for 200 days.

and each particular area. Similarly, at figure 3.3, this might not accurately predict how dengue fever spreads during each season over ten years by setting the fixed parameters and using recruitment rates represented as $Q = 0.1$ for dry season and rainy season with $Q = 0.5$. Hence, the recruitment rate is depended on the amount of rainfall and temperature conditions and each year might need to use a particular the recruitment rate to compute to see the equilibria as well as the basic reproduction number.

Disease free equilibria:

Number of stable equilibria = 4607
Number of unstable equilibria = 5393
Number failed trials = 0

	S	V
min	0.670335	1.4708
max	1.48398	15.0316
mean	0.992937	5.0943
std	0.163666	2.23583

Disease endemic equilibria:

Number of stable equilibria = 5393
Number of unstable equilibria = 4607
Number failed trials = 0

	S	I	R	V	G
min	0.0159377	6.03782e-07	0.000497879	1.88156	2.57169e-07
max	1.36488	0.0109684	1.38323	24.7295	0.0131249
mean	0.478253	0.0019146	0.551764	9.52468	0.00119882
std	0.277916	0.0019167	0.293171	4.25286	0.00141841

Figure 3.2: Ten thousand random combinations of parameter values within the ranges in Table 3.1 were used to compute points of equilibria. The top half of the figure shows the number of stable and unstable disease-free equilibria for these combinations of parameter values. The category "failed trails" was included in order to track errors were detected during the computations but none were found. Statistics for the values of the state variables S and V for stable disease-free equilibria are reported. The lower half of the figure shows analogous results for the disease endemic equilibria.

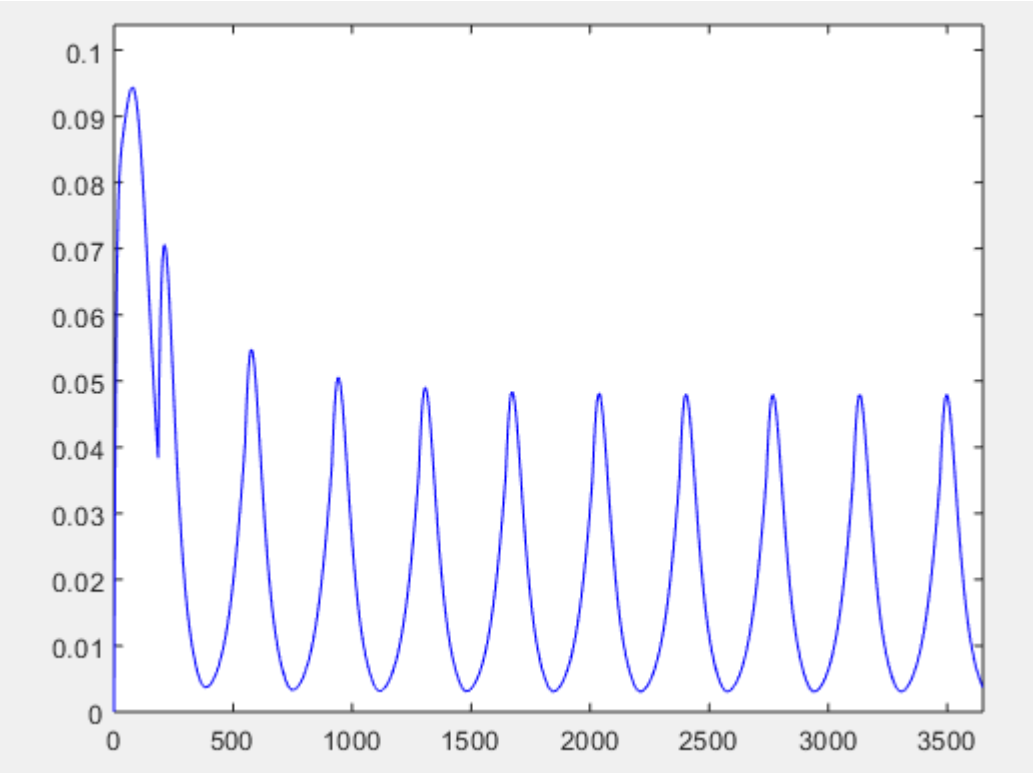

Figure 3.3: Proportions of infected humans, I, over ten-year periods with values of parameters as in table 3.2 and with Q set to 0.1 and 0.5 for alternating sequences of 180-day periods. The initial values were set at $S_0 = 1.0, I_0 = 0, R_0 = 0, V_0 = 0.5, G_0 = 0.5$.

Chapter 4

Discussion

4.1 Limitation of the current thesis

Some parameters such as recruitment rate of both humans and vectors are not necessarily accurate due to the various regions in terms of geographical condition such as rainfall and temperature where the previous studies were taken. This paper discusses only general basic assumption regarding to dengue and do not focus on any type of dengue fever (DEN1-4). Thus, this finding cannot be generalized for all type of dengue fever. This research only focuses on one specific area of Laos namely Vientiane where the largest number of dengue infected patients occurs. Therefore, this research might not be transferrable into another context. The study mainly discusses the aspect of seasonal pattern as the factors causing dengue.

4.2 Further research and future to extend the work

There are needs to consider other aspects such as environment conditions, density of population when discussing the factors causing dengue. As a time limitation for this thesis research, there are some further research that may need to be done.

1. Further research might need to consider other contexts of Laos.

2. One may consider to put 4 types of dengue fever into the model because finding ways to get rid of dengue fever is quite hard since each person

can have 4 chances to infect with this disease. This is also emphasized by (Rodrigues et al., 2016) that dengue fever has four different types and one has a likelihood getting infection again from other types because after recovering from a particular type can only has an immunity for a particular one.

3. On call optimise the fit between the data and the parameter value in the ranges given in table 3.1 to find the best fit between the data and the model.

4. One could introduce a continuous time varying value for Q such as $Q(t) = a + b\sin(s(t-d))$.

5. One could model the link between Q and rainfall $Q(t) = a + bR(t-d)$, where $R(t)$ is the rainfall at time t.

4.3 Summary

This research formulates explicit formulae for equilibria found as is the basic reproduction number R_0. According to the result of this research, there are still some aspects needed to be added into the dengue fever model to analyse the best fit for finding the influences of the dengue fever for further research. The Matlab codes as in the appendix A are invented to compute the equilibria as in the figure 3.2 as well as using numerical simulations to demonstrate the seasonal pattern influence the dengue fever over ten years as in the figure 3.3 and using R program to compute the code as in the appendix B, to see the scenario how dengue fever spreads over 200 days. This thesis did not specifically discuss any particular type of dengue fever and also did not consider the aspects with various regions' conditions in terms of temperature and rainfall.

Appendix A

Source code: MATLAB

```
function OutVec = Equilibria(ParamVec)

% May 24 2018
%
% This program implements the model for the spread of dengue fever in the
% thesis by PinkamThanavanh.
%
% The input variable ParamVec 1 x 7 is the vector of parameter inputs:
%    ParamVec = [P Q delta beta alpha gamma theta]
%
% The program computes the coordinates for the disease free equilibrium and
% the disease endemic equilibrium. The program RouthTable.m is used to
% decide if the equilibria are stable or not.
%
% The output variable is of size 1 x 9. The components are as follows:
% 1. Zdf   equilibria index for the disease free equilibrium
% 2. Sdf   S value for the disease free equilibrium
% 3. Vdf   V value for the disease free equilibrium
% 4. Zend  equilibria index for the disease endemic equilibrium
% 5. Send  S value for the disease endemic equilibrium
```

```
21 % 6. Iend  I value for the disease endemic
       equilibrium
22 % 7. Rend  R value for the disease endemic
       equilibrium
23 % 8. Vend  V value for the disease endemic
       equilibrium
24 % 9. Gend  G value for the disease endemic
       equilibrium
25 %
26 % The equilibriium index has three possible values
27 % Z = 0 stable
28 % Z = 1 unstable
29 % Z = 2 test failed (the program RouthTable.m does
       not cover all possible
30 %       scenarios.
31 %
32 % The notation and terminology follows the notes by
       Pin.
33
34 % %%%%% Extract the parameters from the input data.
35 P = ParamVec(1);
36 Q = ParamVec(2);
37 d = ParamVec(3);  % d = delta in the notes
38 b = ParamVec(4);  % b = beta  in the notes
39 a = ParamVec(5);  % a = alpha in the notes
40 g = ParamVec(6);  % g = gamma in the notes
41 t = ParamVec(7);  % t = theta in the notes
42
43 % %%%%% Compute and display the disease free
       equilibrium
44 S = P/d;    % S is the S value for the disease free
       equilibrium
45 I = 0;      % I is the I value for the disease free
       equilibrium
46 R = 0;      % R is the R value for the disease free
       equilibrium
47 V = Q/t;    % V is the V value for the disease free
       equilibrium
48 G = 0;      % G is the G value for the disease free
       equilibrium
49
```

```
50 %disp(' ')
51 %disp(['The disease free equilibrium is at'])
52 %disp([S I R V G])
53
54 % %%%%% Find the characteristic polynomial for the
      disease free
55 % %%%%% quilibrium.
56
57 A = a*G+d;
58 C = a*S;
59 D = a*G;
60 E = d + b;
61 F = g*V;
62 H = g*I + t;
63 K = g*I;
64
65 W2 = C*F;
66 W1 = C*F*(A - D + K- H);
67 W0 = C*F*(A*H + D*K - A*K - D*H);
68
69 Z4 = 1;
70 Z3 = A + E + H + t;
71 Z2 = A*E + A*H + A*t + E*H + E*t + H*t;
72 Z1 = A*E*H + A*E*t + A*H*t + E*H *t;
73 Z0 = A*E*H*t;
74
75 Cvec = [Z4 Z3 (Z2-W2) (Z1-W1) (Z0-W0)];
76
77 Zdf = RouthTable(Cvec);
78 Sdf = S;
79 Vdf = V;
80
81
82
83 % %%%%% Compute and display the disease endemic
      equilibrium
84 Stop = t*(P*g + t*(d + b));
85 Sbot = g*(a*Q + d*t);
86 S = Stop/Sbot;      % S value for the disease endemic
      equilibrium.
87
```

```
88  Vtop = (d + b)*(a*Q + d*t);
89  Vbot = a*(P*g + t*(d + b));
90  V = Vtop/Vbot;      % V value for the disease endemic
        equilibrium.
91
92  Top = Q*a*(P*g + t*(d+b));
93  Bot = (d+b)*(Q*a + d*t);
94  X = (Top/Bot) - t;
95
96  R = b*X/(g*d);      % R value for the disease endemic
        equilibrium.
97  I = X/g;            % I value for the disease endemic
        equilibrium.
98  G = X/t;            % G value for the disease endemic
        equilibrium.
99
100
101 % %%%%% Find the characteristic polynomial for the
        disease endemic
102 % %%%%% quilibrium.
103
104 A = a*G+d;
105 C = a*S;
106 D = a*G;
107 E = d + b;
108 F = g*V;
109 H = g*I + t;
110 K = g*I;
111
112 W2 = C*F;
113 W1 = C*F*(A - D + K- H);
114 W0 = C*F*(A*H + D*K - A*K - D*H);
115
116 Z4 = 1;
117 Z3 = A + E + H + t;
118 Z2 = A*E + A*H + A*t + E*H + E*t + H*t;
119 Z1 = A*E*H + A*E*t + A*H*t + E*H *t;
120 Z0 = A*E*H*t;
121
122 Cvec = [Z4 Z3 (Z2-W2) (Z1-W1) (Z0-W0)];
123
```

```
124 Zend = RouthTable(Cvec);
125
126 OutVec = [Zdf Sdf Vdf Zend S I R V G];
```

```
1  function ModelExperiment
2
3  % May 27 2018
4  %
5  % This is a shell program for checking the stability
       of equilibria for the
6  % dengue fever model using the program Equilibria.m.
       The program
7  % Equilibria.m outputs a vector of length 9 the
     components of which are as
8  % follows:
9  % 1. Zdf    equilibria index for the disease free
     equilibrium
10 % 2. Sdf    S value for the disease free equilibrium
11 % 3. Vdf    V value for the disease free equilibrium
12 % 4. Zend   equilibria index for the disease endemic
     equilibrium
13 % 5. Send   S value for the disease endemic
     equilibrium
14 % 6. Iend   I value for the disease endemic
     equilibrium
15 % 7. Rend   R value for the disease endemic
     equilibrium
16 % 8. Vend   V value for the disease endemic
     equilibrium
17 % 9. Gend   G value for the disease endemic
     equilibrium
18 %
19 % The equilibriium index has three possible values
20 % Z = 0 stable
21 % Z = 1 unstable
22 % Z = 2 test failed (the program RouthTable.m does
     not cover all possible
23 %       scenarios.
24
25
26 % Paramter ranges
```

```
27 Pmin = 0.00008; % minimum recruitment rate for
      humans
28 Pmax = 0.00012; % maximum recruitment rate for
      humans
29 Qmin = 0.1;     % minimum recruitment rate for
      mosquitos
30 Qmax = 0.5;     % maximum recruitment rate for
      mosquitos
31 dmin = 0.00008; % minimum mortality rate for humans
                  (delta)
32 dmax = 0.00012; % maximum mortality rate for humans
                  (delta)
33 bmin = 0.01;    % minimum recovery rate for humans
                   (beta)
34 bmax = 0.10;    % maximum recovery rate for humans
                   (beta)
35 amin = 0.01;    % minimum infection rate: humans
      from mosquitos (alpha)
36 amax = 0.03;    % maximum infection rate: humans
      from mosquitos (alpha)
37 gmin = 0.01;    % minimum infection rate: mosquitos
      from humans (gamma)
38 gmax = 0.03;    % maximum infection rate: mosquitos
      from humans (gamma)
39 tmin = 0.02;    % minimum mortality rate for
      mosquitos          (theta)
40 tmax = 0.07;    % maximum mortality rate for
      mosquitos          (theta)
41
42 % Set the number of random combinations of parameter
       values
43 N = 10000;
44
45 % Initialise the array for storing results for each
      trial.
46 OutMat = zeros(N,9);
47
48 % Loop to computed equilibria and stability for each
       trial.
49 for k = 1:N
50     % Randomly choose a value for each parameter
```

```
        according to a uniform
51      % over distribution over its range.
52      P = (Pmax-Pmin)*rand + Pmin;
53      Q = (Qmax-Qmin)*rand + Qmin;
54      d = (dmax-dmin)*rand + dmin;
55      b = (bmax-bmin)*rand + bmin;
56      a = (amax-amin)*rand + amin;
57      g = (gmax-gmin)*rand + gmin;
58      t = (tmax-tmin)*rand + tmin;
59      ParamVec = [P Q d b a g t];
60
61      % Call the program Equilibria.m to find the
           disease free and the
62      % disease endemic equilibria and report the
           stability for each.
63      OutVec = Equilibria(ParamVec);
64
65      % Record the output of the current trial in the
           array OutMat.
66      OutMat(k,:) = OutVec;
67  end
68
69  % Extract the columns from the output array OutMat
70  ZdfVec = OutMat(:,1);
71  %SdfVec = OutMat(:,2);
72  %VdfVec = OutMat(:,3);
73  ZenVec = OutMat(:,4);
74  %SenVec = OutMat(:,5);
75  %IenVec = OutMat(:,6);
76  %RenVec = OutMat(:,7);
77  %VenVec = OutMat(:,8);
78  %GenVec = OutMat(:,9);
79
80  % Tabulate the stability results for the disease
       free equilibrium
81  dfVec0 = find(ZdfVec == 0);
82  Ndf0 = length(dfVec0);
83  dfVec1 = find(ZdfVec == 1);
84  Ndf1 = length(dfVec1);
85  dfVec2 = find(ZdfVec == 2);
86  Ndf2 = length(dfVec2);
```

```
87
88  disp(' ')
89  disp('Disease free equilibria:')
90  disp(' ')
91  disp(['Number of stable equilibria =    ' int2str(
        Ndf0)])
92  disp(['Number of unstable equilibria =  ' int2str(
        Ndf1)])
93  disp(['Number failed trials =           ' int2str(
        Ndf2)])
94
95  DFarray = [
96      min(OutMat(:,[2 3]));
97      max(OutMat(:,[2 3]));
98      mean(OutMat(:,[2 3]));
99      std(OutMat(:,[2 3]))];
100 TextArray = [
101     'min   ';
102     'max   ';
103     'mean  ';
104     'std   '];
105 DFtot = [TextArray num2str(DFarray)];
106 disp(' ')
107 disp(['          S           V'])
108 disp(DFtot)
109
110 % Tabulate the stability results for the disease
        endemic equilibrium
111 deVec0 = find(ZenVec == 0);
112 Nde0 = length(deVec0);
113 deVec1 = find(ZenVec == 1);
114 Nde1 = length(deVec1);
115 deVec2 = find(ZenVec == 2);
116 Nde2 = length(deVec2);
117
118 disp(' ')
119 disp('Disease endemic equilibria:')
120 disp(' ')
121 disp(['Number of stable equilibria =    ' int2str(
        Nde0)])
122 disp(['Number of unstable equilibria =  ' int2str(
```

```
    Nde1)])
123 disp(['Number failed trials =            ' int2str(
    Nde2)])
124
125 DEarray = [
126     min(OutMat(:,[5 6 7 8 9]));
127     max(OutMat(:,[5 6 7 8 9]));
128     mean(OutMat(:,[5 6 7 8 9]));
129     std(OutMat(:,[5 6 7 8 9]))];
130 TextArray = [
131     'min   ';
132     'max   ';
133     'mean  ';
134     'std   '];
135 DEtot = [TextArray num2str(DEarray)];
136 disp(' ')
137 disp(['          S           I               R
                V           G'])
138 disp(DEtot)
```

```
1 function Z = RouthTable(A)
2
3 % 24 May 2018
4 %
5 % Construct the Routh table for a polynomial. If all
    the element are the
6 % same sign, and the polynomial is the
    characteristic polynomial for the
7 % Jacobian of a system evaluated at a point of
    equilibrium, then all the
8 % eigenvalues of the system have negative real parts
    and so the equilibrium
9 % is stable.
10 %
11 % A is vetor of coefficients of a polynomial. Thus
    if
12 %
13 % P(x) = a_nx^n + a_n-1x^n-1 + ... + a_2x^2 + a_1x +
    a_0
14 %
15 % Then the vector A = [a_n a_n-1 ... a_2 a_1 a_0].
```

```
16 %
17 % The output parameter Z has three possible values:
18 % Z = 0:  stable
19 % Z = 1:  unstable
20 % Z = 2;  no result (either an entire row was
      prematurely zero or a zero
21 %         appeared in the first column
22 %
23 % The user has the option of displaying the Routh
      array by setting the
24 % display flag dispflag = 1.
25
26 % The user can choose to display the Routh table by
      setting the flag = 1.
27 dispflag = 0;
28
29 % Construct the first two rows of the table. The
30 n = length(A);
31 if rem(n,2) == 1
32     A = [A 0];
33     n = n+1;
34 end
35 n2 = n/2;
36 T12 = reshape(A,2,n2);
37
38 % Start constructiong the Routh Table. Pad by zeros
      to allow computations
39 % of table entries involving coefficient outside the
       initial range.
40 m = 2*n;
41 n21 = n2 + 1;
42 RT = zeros(m,n21);
43
44 % Insert the first two rows
45 RT(1:2,1:n2) = T12;
46
47 % Initialise the row counter for the main iteration.
48 RowNum = 2;
49
50 % If the second row is all zeros, set Z = 2 (test
      failed) and terminate.
```

```
51 curvec = RT(RowNum,:);
52 posvec = find(abs(curvec) > 0);
53 L = length(posvec);  % L is the number of non-zero
      terms in the second row.
54 if L == 0
55     Z = 2;           % Assign the output parameter Z
           = 2 (test failed).
56     if dispflag == 1
57         disp(' ')
58         disp(RT(1:RowNum,1:n2))   % Display the
              Routh array (aborted).
59         disp(' ')
60         disp('Test failed')       % Display the
              conclusion.
61         disp(' ')
62     end
63     return                        % Terminate the
          computation.
64 end
65
66 while L > 0          % As long as rows are not all
      zeros, iterate.
67     RowNum = RowNum + 1;              % Update the
          current row number.
68     a = RT(RowNum-1,1);               % Extract a
69     if a == 0                         % If a = 0,
          the test fails.
70         Z = 2;                        % Assign Z =
              2, test failed.
71         if dispflag == 1
72             disp(' ')
73             disp(RT(1:RowNum,1:n2))   % Display
                  aborted Routh table.
74             disp(' ')
75             disp('Test failed')       % Display the
                  conclusion.
76             disp(' ')
77         end
78         return                        % Terminate
              the compuation
79     end
```

```
80      c = RT(RowNum-2,1);                 % Extract c
81      for k = 1:n2                        % For every
           column, extract b and d
82          b = RT(RowNum-1,k+1);
83          d = RT(RowNum-2,k+1);
84          RT(RowNum,k) = (a*d - b*c)/a; % Compute the
               current table entry
85                                          % as (ad-bc/a.
86      end
87      curvec = RT(RowNum,:);
88      posvec = find(abs(curvec) > 0);     % Find the
           number of postive terms,
89      L = length(posvec);                 % L, in the
           new row.
90  end
91
92  C1 = RT(:,1);                           % Extract the
       first column of the
93                                          % Routh table.
94  Cpos = find(C1 > 0);
95  Cneg = find(C1 < 0);                    % Lpos is the
       number of positive
96  Lpos = length(Cpos);                    % terms and
       Lneg is the number of
97  Lneg = length(Cneg);                    % negative
       terms in the first column.
98  Lmin = min(Lpos,Lneg);                  % If both are
       postive, the system is
99  if Lmin > 0                             % unstable. If
        one is zero, the
100     Z = 1;                              % system is
           stable.
101 else
102     Z = 0;
103 end
104
105 if dispflag == 1                        % Display
       output if dispflag = 1.
106     disp(' ')
107     disp(RT(1:RowNum,1:n2))             % Display the
           full Routh table
```

```
    disp(' ')
    if Z == 0
        disp('The system is stable')
    elseif Z == 1
        disp('The system is unstable')
    elseif Z == 2
        disp('There is no conclusion')
    end
    disp(' ')
end
```

```
function Dengue

% October 2 2018
%
% Compute the model in Pin's thesis for different
   values of Q

% Set the parameters other than Q
global P alpha beta delta gamma theta
P = 0.0001;
alpha = 0.02;
beta = 0.05;
delta = 0.0001;
gamma = 0.02;
theta = 0.045;

% Set the duration of the two seasons
t1 = 180;  % time interval for dry season
t2 = 185;  % time interval for wet season

% Set the vector recruitment values for the two
   seasons.
Q1 = 0.1;  % vector recruitment during the dry
   season
Q2 = 0.5;  % vector recruitment during the wet
   season

% Compute R0 for the two values of Q
R01 = alpha*gamma*P*Q1/(delta*(theta^2)*(delta+beta)
   );
```

```
26 R02 = alpha*gamma*P*Q2/(delta*(theta^2)*(delta+beta)
   );
27 disp(['With Q1 = ' num2str(Q1) ', R0 = ' num2str(R01
   )])
28 disp(['With Q2 = ' num2str(Q2) ', R0 = ' num2str(R02
   )])
29
30 % initial conditions at the start of the wet season
31 S0 = 1;
32 I0 = 0;
33 R0 = 0;
34 V0 = .5;
35 G0 = .5;
36
37 Tinit = 0;
38 TotIvec = [ ];
39 TotTvec = [ ];
40
41 Nyears = 10; % number of years
42 for k = 1:Nyears
43
44     % Compute the system for the wet season
45     u0 = [S0 I0 R0 V0 G0];
46     Tfinal = Tinit + t2;
47     tint = [Tinit Tfinal];
48     Tinit = Tfinal;
49
50     [tvec u] = ode45('DengueFun2',tint,u0);
51
52     Ivec = u(:,2);
53     TotIvec = [TotIvec ; Ivec];
54     TotTvec = [TotTvec ; tvec];
55
56     % Compute the system for the dry season
57     [a,b] = size(u);
58     %lengthtvec = length(tvec)
59     s0 = u(a,1);
60     I0 = u(a,2);
61     R0 = u(a,3);
62     V0 = u(a,4);
63     G0 = u(a,5);
```

```
64      u0 = [S0 I0 R0 V0 G0];
65      Tfinal = Tinit + t1;
66      tint = [Tinit Tfinal];
67      Tinit = Tfinal;
68
69      [tvec u] = ode45('DengueFun1',tint,u0);
70
71      Ivec = u(:,2);
72      TotIvec = [TotIvec ; Ivec];
73      TotTvec = [TotTvec ; tvec];
74
75      [a,b] = size(u);
76      %lengthtvec = length(tvec)
77      s0 = u(a,1);
78      I0 = u(a,2);
79      R0 = u(a,3);
80      V0 = u(a,4);
81      G0 = u(a,5);
82
83
84  end
85
86  plot(TotTvec,TotIvec,'b')
87  T = (t1+t2)*Nyears;
88  axis([0 T 0 1.1*max(TotIvec)])
```

```
1   function f = DengueFun1(tvec,u)
2
3   global P alpha beta delta gamma theta
4   S = u(1);
5   I = u(2);
6   R = u(3);
7   V = u(4);
8   G = u(5);
9
10  Q = 0.1;
11  fS = P - alpha*S*G - delta*S;
12  fI = alpha*S*G - (delta+beta)*I;
13  fR = beta*I - delta*R;
14  fV = Q - gamma*V*I - theta*V;
15  fG = gamma*V*I - theta*G;
```

```
16
17 f = [fS ; fI ; fR ; fV ; fG];
```

```
1  function f = DengueFun2(tvec,u)
2
3  global P alpha beta delta gamma theta
4  S = u(1);
5  I = u(2);
6  R = u(3);
7  V = u(4);
8  G = u(5);
9
10 Q = 0.5;
11 fS = P - alpha*S*G - delta*S;
12 fI = alpha*S*G - (delta+beta)*I;
13 fR = beta*I - delta*R;
14 fV = Q - gamma*V*I - theta*V;
15 fG = gamma*V*I - theta*G;
16
17 f = [fS ; fI ; fR ; fV ; fG];
```

Appendix B

Source code: R program

```
library(deSolve)
library(scatterplot3d)

SIRVGmodel<-function(time,state, parameters){
with(as.list(c(state, parameters)),{

dS<- P-alpha*S*G-delta*S
dI<- alpha*S*G-(delta+beta)*I
dR<- beta*I-delta*R
dV<- Q-gamma*V*I-theta*V
dG<- gamma*V*I-theta*G

return(list(c(dS,dI,dR,dV, dG)))
})
}
SIRVGpars<-c(P=.0001, Q=.1, delta=.005, beta=.01,
   alpha=.03, gamma=.03, theta=.02)

init<- c(S=100000, I=10, R=0, V=1000, G=100)
times<- seq(1,200.1)

out <- ode(y=init, times=times, func=SIRVGmodel,
   parms=SIRVGpars, method="ode23")
out <- as.data.frame(out)

print(head(out))
plot(out$time, out$S, type='l', col="red", lwd=2,
```

```
    xlab="Day of epidemic", ylab="", main="SIRVG
    model")
lines(out$time, out$I, lty=2, col="blue", lwd=2)
lines(out$time, out$R, lty=3, col="green", lwd=2)
lines(out$time, out$V, lty=4, col="cyan", lwd=2)
lines(out$time, out$G, lty=5, col="magenta", lwd=2)
legend("topright", legend=c("Susceptible humans", "
    Infected humans", "Recovered", "Susceptible
    vectors", "Infected vectors"),
col=c("red", "blue", "green", "cyan", "magenta"),
    lty=1:5, lwd=rep(3,5), cex=0.9)
```

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