MATHEMATICAL MODEL IN EPIDEMIOLOGY

by

SAMUEL OBARA

(Under the Direction of Malcolm R. Adams)

ABSTRACT

The spread of disease in humans and animals can be modeled by systems of differential equations. The total population is subdivided into three categories: Susceptible *(S)*, infective *(I)* and Recovered *(R)* i.e. $N = S(t) + I(t) + R(t)$. R_0 is a parameter that measures the initial growth rate. A system with $R_0 < 1$ is considered not to result in an epidemic whereas when $R_0 > 1$, the system will result into an epidemic. We will consider three models. The first and simplest ignores vital dynamics like births and deaths; it is appropriate for studying "short term" diseases. The second model takes account of those vital dynamics needed for an effective analysis of diseases taking a longer time to sweep through the population. More refined dynamics allowed in the third model take the transmission process of a disease into account.

INDEX WORDS: Epidemiology, Epidemic, Population, Model, Jacobian Matrix, Bifurcation

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DEDICATION

To my wife, who encourages me daily, and to my kids, Esther and Zachariah and to my beloved parents Zachariah Obara and Esther Nyaberi.

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CHAPTER 1

INTRODUCTION

From time immemorial, outbreaks of epidemics have been a main source of human suffering and misery (Mollison, 1994). The threat of death from epidemics has been minimized in developed countries, but not in developing countries. In some cases, epidemics have led to population decline. This is not only the case for human populations but it is also evidenced in several animal species in the world, such as African carnivores that are susceptible to pathogens found in domestic animals (Fiorello, 2004; Van Heerden *et al.*, 1989).

The earliest epidemic ever recorded in history occurred in Athens between 430 – 426 B.C. during the Peloponnesian war fought between Sparta and Athens. Athens was a city with a strong navy and weak army whereas Sparta had a strong army with a weak navy. The leader of Athens, Pericles, decided to bring his people from around Athens to the fortified city to avoid any attack by land. Due to overcrowding and limited sanitary facilities this led to the outbreak of highly contagious diseases. It is believed that about 30% – 60% of the population succumbed to this plague. When the epidemic ended, Pericles ordered his mighty navy to capture Sparta but the navy was struck by the plague while en-route and Pericles called off the attack to return to Athens. Athens was again attacked by the plague in 428 and 426 A.D. Pericles was among those who succumbed to this outbreak. The war lasted for many years and since the epidemic in Athens weakened their navy and army, Sparta was finally able to defeat Athens (Smith, 1996).

Another reported case of epidemic was in the fourteenth century when bubonic plague killed about 25% of the population in Europe. “Bubonic plague, the most common form, is characterized by very high fever, chills, prostration, delirium, hemorrhaging of the small capillaries under the skin, and enlarged, painful lymph nodes (buboes), which suppurate and may discharge” ("Plague", 2005). In 1520 a smallpox epidemic caused half of the population of the Aztec to perish. In 1919 there was a widespread outbreak of influenza worldwide that claimed around 20 million people (Collins & Lehman, 1953). Epidemics have been reported in various parts of the world in recent time. The main cases that are currently being reported are due to outbreaks of Malaria and HIV AIDS.

In the seventeenth century, a number of people started investigating the incidence of epidemics. John Graunt (1620-1674) gathered data of incidences and locations of epidemics (Champion, 1993). In 1760, Daniel Bernoulli came up with a mathematical model that was to be used to study the population dynamics of infectious diseases (Daley & Gani, 1999). In his model, Bernoulli developed differential equations which he solved with the aim of analyzing the risks that come with preventive inoculation. The modern theory of epidemics came into being by the work of William Hammer and Sir Ronald Ross early in the twentieth century (Haggett, 1994).

Epidemic models play a very crucial role in life today. With high occurrences of epidemics in recent times, epidemic models are highly in demand. One of the reasons for formulating epidemic models that adequately describe communicable disease data is that the model provides a convenient summary of the data. Another more important reason is that such models can help to provide insight into the

biological and sociological mechanisms underlying the process of disease spread (Becker, 1979, p. 298).

Epidemics such as AIDS pose a serious challenge to develop an appropriate model. “The problem in modeling this epidemic is that it is nonlinear and no simple mathematical formula predicts how the number of cases will increase”(Kolata, 1987, p. 1464).

Epidemics in A Closed Population

In reality, demographic dynamics model old individuals that die out and are replaced with newborn. The time required for this demographic process to occur is much longer than the time it takes for an infectious disease to sweep through the entire population. For short term diseases this factor can be safely ignored. In such a situation, we consider the population to be closed and assume that it is free from the disease we are concerned about (a “virgin” population). Assume that for some reason or another, the disease is introduced to at least one host. In such a scenario, the following questions may be asked:

- Does this cause an epidemic?
- If so, with what rate does the number of infective hosts increase during the rise of the epidemic?
- What proportion of the population will ultimately have experienced infection? (Diekmann, 2000, p.3).

At the initial stages of the introduction of the disease-causing organisms that may lead to epidemics, there are only few infective in a large susceptible population. But it should be noted that there exists a period after which the newly infective becomes infectious (latency period). Denoting the length of this latency period by T_1, and the length

infectious period by $T_2 - T_1$, we are faced with the question: What happens at the end of T_1 and at the end of T_2? As Diekmann, (2000, p.4) states:

> In order to distinguish between an avalanche-like growth and an almost-immediate extinction, we introduce the basic reproduction ratio:
>
> R_0 : = expected number of secondary cases per primary case in a 'virgin' population.
>
> In other words, R_0 is the initial growth rate (more accurately: multiplication factor; note that R_0 is dimensionless) when we consider the population on a generation basis (with 'infecting another host' likened to 'begetting a child'). Consequently, R_0 has threshold value 1, in the sense that an epidemic will result from the introduction of the infective agent when $R_0 > 1$, while the number of infectives is expected to decline (on a generation basis) right after the introduction when $R_0 < 1$. The advantage of measuring growth on a generation basis is that for many models one has an explicit expression for R_0 in terms of the parameters.

This thesis is presented in five parts. The first introduces the SIR model, which is simplest and in particular ignores vital dynamics. It provides a foundation for subsequent study in more specific situations. The second part is devoted to the SIR model with vital dynamics, which more effectively represents longer-term diseases. The third part focuses on a model, which allows for a more complicated transmission dynamics. Due to its complexity, we will not analyze the third model completely. Instead we will focus on certain illuminating examples. In my analysis of the three models, I will make extensive use of the MAPLE© software package for solving the systems of differential equations and also plotting their solutions. The fourth part lists references. I use the APA format to

organize my reference lists. Finally, in the appendix, I provide the reader with all the MAPLE codes used in the body of the paper.

Epidemiology has been extensively studied for a long time as is documented in the literature today. My contribution to this important topic is as follows. While most of the literature is written for experts, my aim is to digest it for more general readers and hope to present a relatively self-contained account. Secondly, I will provide MAPLE codes at the end of the document and illustrate their uses; these are not normally presented in other publications. Finally we will discuss and derive the results for a model that has been newly developed for application to animal diseases in South America; we will discuss a unique kind of birfurcation, Hopf birfurcation. The Hopf bifurcation (or Poincare-Andronov-Hopf) occurs when a pair of complex eigenvalues crosses the imaginary axis as a parameter is moved (and, in dimensions, bigger than two, the remaining eigenvalues have negative real part), provided that some additional technical conditions hold. This process usually causes the formation of a limit cycle. Using the third model, I will provide evidence of the existence of a Hopf burfurcation and also provide a proof of it. So in general, I will provide the readers with a cohesive and comprehensive document as a gift to this important area of study.

CHAPTER 2

MODEL I: THE SIR MODEL WITHOUT VITAL DYNAMICS

In the simplest epidemic model a constant population size, N, is assumed. That is to say, there are no deaths or movement of population to affect the population size. Since many epidemics take a relatively short time as compared to the life span of the individuals in the population, the assumption of constant population size is reasonable. That is to say, "Since an epidemic occurs relatively quickly, the model does not include births and deaths (vital dynamics). Epidemics are common for diseases such as influenza, measles, rubella and chickenpox" (Levin *et al.*, 1989, p. 128). The next assumption in this simple model is that the population is subdivided into three mutually exclusive sets:

1. The *susceptibles* are those individuals of the population that do not have the disease at a certain time, t ,but may have it at a later date.
2. The *infectives* are those individuals of the population that have already been infective by the disease at a given time, t ,and have the potential of transmitting it to others
3. The *recovered* are those individuals in the population that have recovered from the disease and are no longer infectious at time t.

I will denote susceptible, infective and Recovereds at time t by *S(t), I(t)* and R(t) in that order. Using the above assumption we can note that:

$$S(t) + I(t) + R(t) = N \qquad (1.1)$$

for all time *t*.

Next, we assume that the rate of decrease of *S(t)* is proportional to the product of the number of susceptibles and the number of infectives which means that the number of susceptibles and infective are homogeneously mixed. Therefore:

$$\frac{dS}{dt} = -\beta S(t)I(t) \qquad (1.2)$$

for all time. (Note that this assumption holds for animals populations but different interaction terms are often used for human population, e.g. in descriptions of HIV AIDS propagation)

Where:

- β is the constant of proportionality which is referred to as infection rate.
- The product S(t)I(t) represents the rate of contact between the susceptibles and infectives.
- βS(t)I(t) represents the proportion of contacts which results in the infection of susceptibles .

We can again assume that the rate of change of the recovered is proportional to the number of infectives. Therefore:

$$\frac{dR}{dt} = rI(t) \qquad (1.3)$$

for all t.

Here r is called the removal rate.

By solving equation (1.1) for I, i.e. I = N – S – R and taking the derivatives on (1.1), we get

$$\frac{dI}{dt} = -\frac{dS}{dt} - \frac{dR}{dt}$$

Using equation (1.2) and (1.3), we get

$$\frac{dI}{dt} = \beta S(t)I(t) - rI(t) = (\beta S(t) - r)I(t) \qquad (1.4)$$

Therefore the system that models the epidemics discussed above is

$$\frac{dS}{dt} = -\beta SI \qquad (1.5a)$$

$$\frac{dI}{dt} = (\beta S - r)I \qquad (1.5b)$$

$$\frac{dR}{dt} = rI \qquad (1.5c)$$

Where β and r are positive constants. The initial conditions for this model are the initial number of susceptibles $S(0) > 0$, the initial number of infectives $I(0) > 0$, and the initial number of recovered $R(0) = 0$. The expected number, R_0, of secondary cases per primary case in a 'virgin' population (introduced above) of this model, will play a key role in the following discussion. To determine R_0 in this situation, first note that the infective individuals are contagious for a period of approximately $\frac{1}{r}$ since, if we consider I to be nearly constant initially, then (1.5c) gives $R(t) \approx rI(0)t$ so when t = $\frac{1}{r}$ the original number of infective, $I(0)$, will have recovered. Next, we rewrite equation (1.5b) to give

$$\frac{dI}{dt} = r\left(\frac{S\beta}{r} - 1\right)I$$

and so at t = 0, when S(t) is approximately N, we have

$$\left.\frac{dI}{dt}\right|_{t=0} \approx r\left(\frac{\beta N}{r} - 1\right)I$$

Again, if we think of $I(t)$ as being nearly constant during the first generation of the disease, we find that the number of infectives at $t = \frac{1}{r}$ is approximately

$$I\left(\frac{1}{r}\right) \approx \frac{\beta N}{r} I(0) - I(0),$$

Thus $\frac{\beta N}{r}$ gives the expected number of secondary case per primary case in the "virgin" population, i.e. $R_0 = \frac{\beta N}{r}$.

WARNING: This multiplication factor R_0 has nothing to do with *R(0),* the initial condition for the recovered. For this reason, we carefully distinguish between the parameter R_0 and the initial condition *R(0);*

To analyze the model in equation (1.5), let us investigate the general behavior of this model. Given $\beta > 0$, $S(t) \geq 0$ and I(t) ≥ 0, $\frac{dS}{dt} = -\beta SI \leq 0$ for all t. As a matter of fact, $\frac{dS}{dt} < 0$ unless $S = 0$ *or* $I = 0$. When $\frac{dS}{dt} < 0$, the susceptibles S(t), is a strictly decreasing function. Furthermore since $r > 0$ and $I(t) \geq 0$, $\frac{dR}{dt} = rI \geq 0$, then $\frac{dR}{dt} > 0$ unless $I = 0$, which means that the number of Recovered is astrictly increasing function of time, *t*. Since I(t) ≥ 0, and $\frac{dI}{dt} = (\beta S - r)I$, the change in the number of infective depends on the nature of $(\beta S - r)$ when $I \neq 0$. For $(\beta S - r) > 0$ i.e. S $> \frac{r}{\beta}$, $\frac{dI}{dt} > 0$, which means the number of infective increases. Whereas when $(\beta S - r) < 0$, i.e. $S < \frac{r}{\beta}$, $\frac{dI}{dt} < 0$, the number of infective decreases. The ratio $\frac{r}{\beta}$ is the relative removal rate. Since $S(t)$ is a

strictly decreasing function, $0 \leq S(t) \leq S(0)$where S(0) is the initial number of susceptibles. If *S(0)* is less than $\frac{r}{\beta}$, no epidemic occurs since $\frac{dI}{dt} = (\beta S - r)I \leq (\beta S(0) - r)I < 0$ which means that *I(t*) is a strictly decreasing function. That is, if *S(0*) < $\frac{r}{\beta}$, the number of infectives decreases monotonically to zero from the initial value of *I(0).* On the other hand if *S(0)*> $\frac{r}{\beta}$ (condition for an epidemic), the number of infectives increases from the initial value of *I(0)* to a maximum value which occurs when the number of susceptibles has decreased to the value $\frac{r}{\beta}$ at some value *t** when *t* > *t*, S (t)* < $\frac{r}{\beta}$ and the number of infectives decreases. This result is what epidemiologist calls the threshold phenomenon. That is, there is a critical value which the number of initial susceptibles must exceed before an epidemic can occur (Brauer & Castillo-Châavez, 2001; Daley & Gani, 1999). The threshold theorem which was proved by W. O. Kermack and A. G. McKendrick in 1927 for equation (1.5) states that:

- If *S(0)* < $\frac{r}{\beta}$, then *I(t)* decreases monotonically to zero.

- If *S(0)* > $\frac{r}{\beta}$, *I(t)* increases monotonically to a maximum value and then decreases monotonically to zero. The $\lim_{t \to \infty} S(t)$, exists and is the unique solution, *x*, of

$S(0)e^{-\beta(N-x)/r} = x$

We can determine these limiting values by manipulating equation (1.5).

Using the chain rule and equation (1.5) $\frac{dS}{dR} = \frac{dS/dt}{dR/dt} = \frac{-\beta SI}{rI} = \frac{-\beta}{r}S$. Integrating,

$\int \frac{dS}{S} = \int \frac{-\beta}{r} dR$ yields $\log_e S = \frac{-\beta}{r}R + \log_e S(0)$, since R(0) = 0. Therefore

$$S = e^{(\frac{-\beta R}{r} + \log_e S(0))} = S(0)e^{(\frac{-\beta R}{r})} \geq S(0)e^{(\frac{-\beta N}{r})} > 0$$

"Thus $S(\infty) > 0$, or there will always be susceptibles remaining in the population. Thus some individuals will escape the disease altogether, and, in particular, the spread of the disease does not stop for lack of a susceptible population" (Waltman, 1974, p. 4). On the other hand, the system can further be examined by using the S-I plane. Also using the chain rule,

$$\frac{dI}{dS} = \frac{dI/dt}{dS/dt} = \frac{\beta S - r}{-\beta S} = \frac{\beta S - r}{-\beta S} = -1 + \frac{r}{\beta}\frac{1}{S} \qquad (1.6)$$

Thus $\int^{I(t)} d\xi = \int^{S(t)} (-1 + \frac{r}{\beta}\frac{1}{\vartheta}) d\vartheta$, which implies

$I(t) = -S(t) + \frac{r}{\beta}\log_e S(t) + I(0) + S(0) - \frac{r}{\beta}\log_e(S(0))$, where $I(0) + S(0) - \frac{r}{\beta}\log_e(S(0)) =$

C_0 is a constant. We can write this as

$$I(t) = -S(t) + \frac{r}{\beta}\log_e S(t) + C_0 \qquad (1.7)$$

The phase portrait representing equation (1.5) above is illustrated in Fig 1.1

below. The value of S in which the epidemic reaches it's peak is when I is maximal.

Since $\frac{dI}{dt} = (\beta S - r)I = 0$ for I maximal, and $I \neq 0$, then $S = \frac{\beta}{r}$. Also note that $\lim_{t\to\infty} I(t) = 0$ since each solution curve eventually hits the S axis. Taking the limit in (1.7), and letting x denote $\lim_{t\to\infty} S(t)$, we see that $0 = -x + \frac{r}{\beta}\log_e x + C_0$ or $e^{\frac{\beta(x-C_0)}{r}} = x$. Noting that $C_0 = I(0) + S(0) - \frac{r}{\beta}\log_e S(0) = N - \frac{r}{\beta}\log_e S(0)$ we see that $x = S(0)e^{\frac{-\beta(N-x)}{r}}$ as promised.

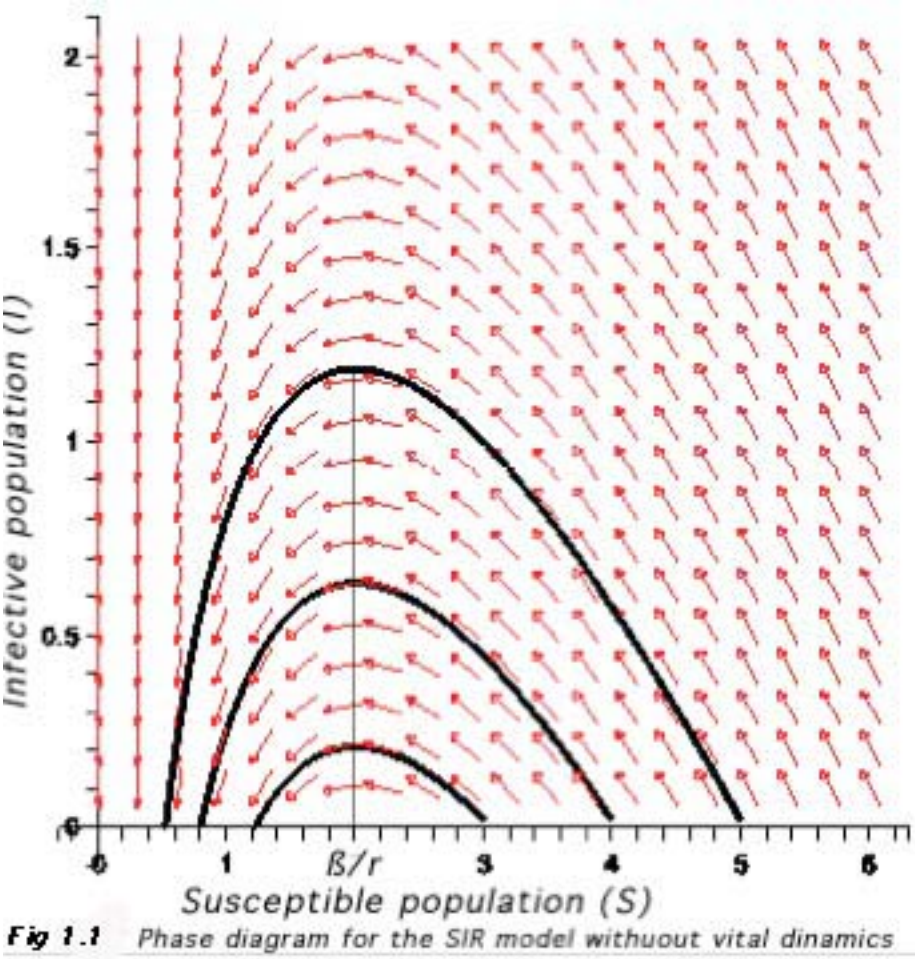

Fig 1.1 Phase diagram for the SIR model withuout vital dinamics

See appendix 1.1 for the maple file that created Fig 1.1

The epidemic curve *I(t)* shown in figure 1.2 below is a bell shaped.

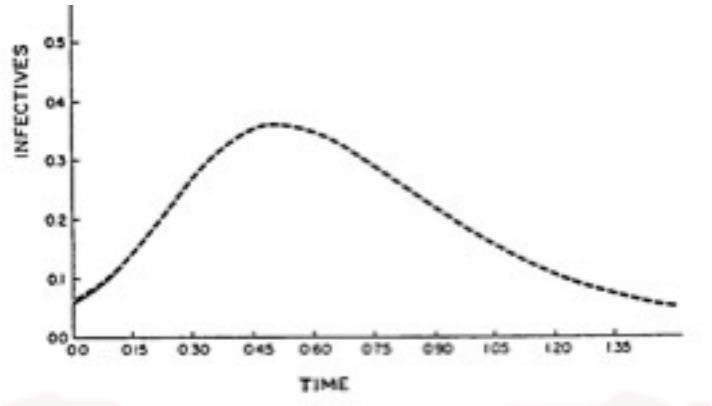

Fig 1.2 An epidemic curve starting at $I_0 = 0.06$ with $\beta = 10$ and $r = 3$ so that $\beta/r = 10/3$

Equilibrium and stability

The equilibrium found algebraically then stability shown by perturbing about equilibrium points (eq. 1.5). From the maple output (appendix 1.2a), the Jacobian matrix of the equation is

$$jc := \begin{bmatrix} -\beta\, In & -\beta\, S \\ \beta\, In & \beta\, S - r \end{bmatrix}$$

Here we use In to denote the infective population, since maple has reserved the symbol I for $\sqrt{-1}$.

The equilibrium states in this model are at the points where $I = 0$, and $S = S_*$ where $S_* = N$ is any positive constant (hence the equilibrium points are non-isolated). From the Maple output

(see appendix 1.2a), there are two eigenvalues, 0 and $\beta S_* - r$.

> "If the condition for an epidemic is satisfied, this second eigenvalue is positive and the equilibrium is unstable. If the second eigenvalue is negative, there is no conclusion about the stability for the linearization because of the zero eigenvalue. However, it is obvious that the equation is not strictly stable even in that case, because any perturbation with non-zero *I* will lead to a situation in which *S* decreases and therefore does not return to
>
> S_*" (Clark, 2002a).

Since $S_* = N$, the second eigenvalue is positive precisely when $R_0 = \frac{\beta N}{r} > 1$. Thus an epidemic occurs when, and only when, each of the initially infective individuals infects more than one other individual.

As a final example for this model, we use Maple to integrate the following hypothetical epidemic model below (see appendix 1.2b).

$$\frac{dS}{dt} = -.0067SI \qquad (1.7a)$$

$$\frac{dI}{dt} = .0067SI - .9I \qquad (1.7b)$$

$$\frac{dR}{dt} = .9I \qquad (1.7c)$$

on the interval [0, 10] and initial conditions: *S(0)* = 400*, I(0)* =7*, R(0) = 0*.

Since $R_0 = \frac{\beta N}{r} = \frac{0.0067 * 407}{0.9} = 3.0298$, there will be an epidemic.

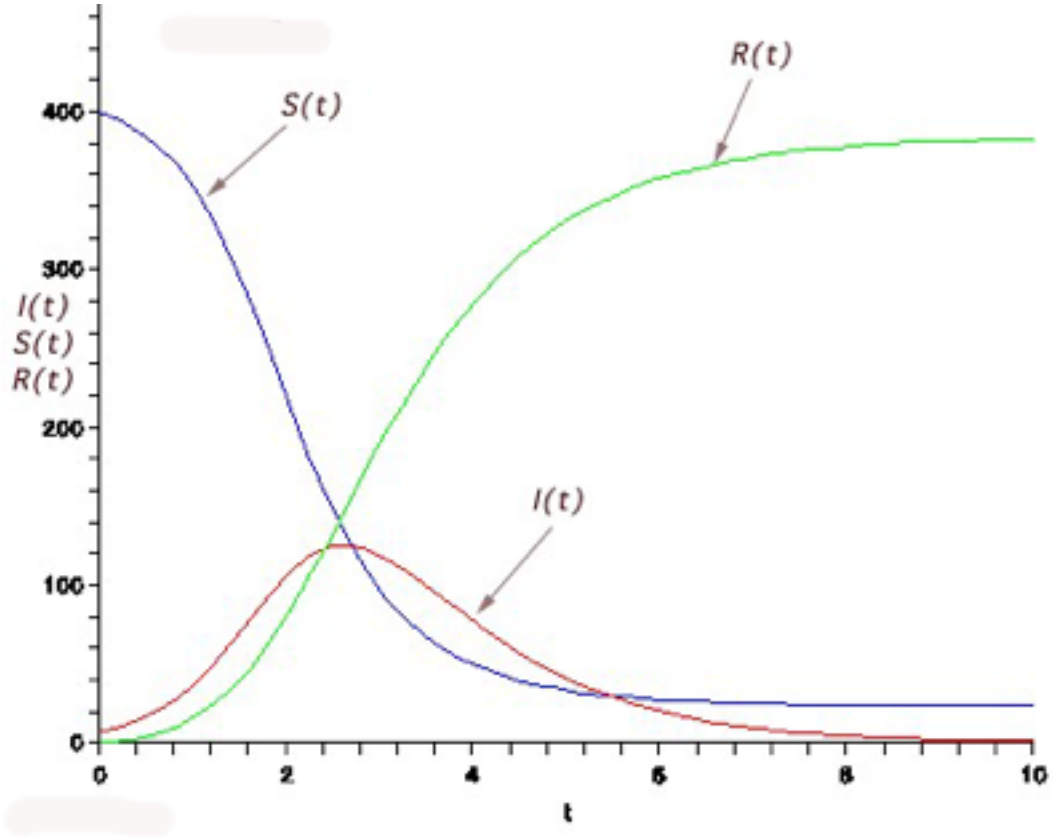

Fig 1.3 A Graph of *S(t), R(t), I(t)*

The epidemic model (1.10) is represented in the graph (Fig 1.3) above where $0 \leq t \leq 10$ and $0 \leq S,I,R \leq 400$. In this model, *S* decreases monotonically from 400 to around 14 whereas *R* increases monotonically from 0 to around 400. *I(t)* has a very different shape as compared with the other two. *I(t)* increases monotonically from 4 and attains its maximum value of about 125 at approximately $t = 2.25$ and then starts decreasing monotonically to zero. The graph for *I(t)* is the most significant. In this model, an epidemic occurs in the period when *I(t)* increases and then decreases.

CHAPTER 3

MODEL II: THE SIR MODEL WITH VITAL DYNAMICS

The model with vital dynamics is very similar to (1.5) except that the inflow and outflow of deaths and births are added in such a way that deaths and births balance to maintain the population size at a constant size N (Hethcote, 2000). This model allows us to investigate events at a longer duration. “A disease is called endemic if it is present in a population for more than 10 or 20 years. Because of the longer time period involved, a model for an endemic disease must include birth as a source of new susceptibles and natural deaths in each class” (Levin et al., 1989, p. 132). As in the case of *S-I-R* model discussed above, this model is also divided into three groups: the susceptibles S, the infective I, and the recovered, R. The total population is $S(t) + I(t) + R(t) = N$ which still remains a constant. Just as Clark (2002b, p. 1) explains that:

> The susceptibles are those who are not infective and not immune, the infectives are those who are infective and can transmit the disease, and the recovered are those who have been infective, have recovereds and are permanently immune. We will include in the model the natural birth and death rates, although with simplifying assumptions. We assume that all births are into the susceptibles. We assume that the death rate is equal for members of all three classes, and we assume that the births and death rates are equal so that the total population is stationary. Finally we assume that this is a non-lethal disease so that the recovered are truly recovered and not dead”.

The differential equations for the SIR model with vital dynamics is

$$\frac{dS}{dt} = -\beta SI + \mu N - \mu S \qquad (2.1a)$$

$$\frac{dI}{dt} = \beta SI - \mu I - rI \qquad (2.1b)$$

$$\frac{dR}{dt} = -\mu R + rI \qquad (2.1c)$$

where μ is the birth and death rate.

Note that (2.1a) with (2.1b) forms a closed system, i.e. *R* does not appear in (2.1a) and (2.1b), thus (2.1c) can be disregarded in the analysis of the system. To analyze the system, let us consider what happens before the infection (infection-free state) i.e. *(S(0), I(0)) = (N, 0).* After that, we will look at what happens when the system is at an endemic steady state. In this case, R_0, the expected number of secondary cases per primary case becomes $\frac{\beta N}{r+\mu}$ since the main difference between this model and the previous one is that the initial infectious period is $\frac{1}{r+\mu}$, see discussion on page 11.

Case 1- infection-free state

Let us find the steady state. To find the steady we set the right hand side of (2.1a) and (2.1b) equal to 0. i.e.

$$-\beta SI + \mu N - \mu S = 0 \qquad (2.2a)$$

$$(\beta S - \mu - r)I = 0 \qquad (2.2b)$$

Equation 2.2b implies either $I = 0$ or $\beta S - \mu - r = 0$. If $I = 0$, 2.2a implies S = N. This steady state $(\bar{S},\bar{I})$ = (N, 0) is called the *infection free* steady state. To analyze this state further we linearise 2.1a and 2.1b around the state steady state $(\bar{S},\bar{I})$ = (N, 0); we have

$$\frac{\partial}{\partial S}(-\beta SI + \mu N - \mu S) = -\beta I - \mu$$

$$\frac{\partial}{\partial I}(-\beta SI + \mu N - \mu S) = -\beta S$$

$$\frac{\partial}{\partial S}(\beta SI - \mu I - rI) = \beta I$$

$$\frac{\partial}{\partial I}(\beta SI - \mu I - rI) = \beta S - \mu - r$$

yielding the jacobian matrix

$$\begin{pmatrix} -\mu & -\beta N \\ 0 & \beta N - \mu - r \end{pmatrix} \qquad (2.2)$$

when *S = N and I = 0.*

Since this is upper triangular, the eigenvalues are $\lambda_1 = -\mu$ and $\lambda_2 = \beta N - \mu - r$. The infection free equilibrium is asymptotically stable when both eigenvalues have negative real parts (Conrad, 2003). Since $\lambda_1 = -\mu$ is always negative then the condition for stability is that $\lambda_2 = \beta N - \mu - r < 0$. If $\lambda_2 > 0$ then the linearized system is a saddle point and so the equilibrium is unstable. Therefore, the infection-free equilibrium is stable if and only if

$\beta N - \mu - r < 0$ (equivalently: $R_0 < 1$) (2.3)

and unstable if

$\beta N - \mu - r > 0$ (equivalently: $R_0 > 1$) (2.4)

> It is worth noting that although we can have a disease-free state with *S* and *R* both positive and *I* = 0, it is not strictly speaking an equilibrium, because the recovereds gradually die out and are not replaced. Only when all of the recovereds are gone can the population distribution be stationary (Clark, 2002b)

Case 2 – endemic steady state (I ≠0)

The endemic steady state is a state in the system "in which the inflow of new susceptibles is balanced by the incidence (and by death)"(Diekmann & Heesterbeek, 2000, p. 42).

That is to say that endemic steady state $(\bar{S},\bar{I})$ has $\bar{I} > 0$. If $I \neq 0$, there can be another steady state $(\bar{S},\bar{I})$, 2.2b implies that $\beta\bar{S} - \mu - r = 0$ i.e. $\bar{S} = \frac{\mu + r}{\beta}$ and 2.2a then gives $\bar{I} = \frac{\mu N - \mu\bar{S}}{\beta\bar{S}} = \frac{\mu}{\beta}\left(\frac{N}{\bar{S}} - 1\right)$. $\bar{I}$ is positive only when $\frac{N}{\bar{S}} = \frac{NB}{\mu + r} = R_0 > 1$. In this case we have a physically realistic steady state called the endemic steady state.

Note that if $R_0 < 1$, then the endemic state does not exist in the first quadrant and the infection free state is an attractor. Thus there will be no epidemic in this case and the infective population diminishes to zero.

To analyze the state further we will linearize (2.1a) and (2.1b) around the steady state $(\bar{S},\bar{I})$ with $\bar{I} > 0$, we have

$$\frac{\partial}{\partial S}(-\beta SI + \mu N - \mu S) = -\beta I - \mu = -\beta \bar{I} - \mu,$$

$$\frac{\partial}{\partial I}(-\beta SI + \mu N - \mu S) = -\beta S = -\beta \bar{S},$$

$$\frac{\partial}{\partial S}(\beta SI - \mu I - rI) = \beta I = \beta \bar{I}$$

$$\frac{\partial}{\partial I}(\beta SI - \mu I - rI) = \beta \bar{S} - \mu - r = 0$$

yielding the jacobian matrix

$$\begin{pmatrix} -\beta \bar{I} - \mu & -\beta \bar{S} \\ \beta \bar{I} & 0 \end{pmatrix} \qquad (2.5)$$

Therefore (2.5) has trace T = $-\beta \bar{I} - \mu < 0$ and determinant D = $\beta^2 \bar{S} \bar{I} > 0$. Using Fig 2.1 below depicting the trace-determinant plane, we can conclude that since $T = -\beta \bar{I} - \mu < 0$ and $D = \beta^2 \bar{S} \bar{I} > 0$, the endemic state is (locally asymptotically) stable because the point (T,D) lies in the second quandrant of the trace-determinant plane (Blanchard et al., 2002). In other words, if we consider (2.5), the characteristic equation

$$\lambda^2 - T\lambda + D = 0 \qquad (2.6)$$

$$\lambda^2 + (\beta \bar{I} + \mu)\lambda + (\beta^2 \bar{S} \bar{I}) = 0 \qquad (2.7)$$

yields

$$\lambda = \frac{-(\beta \bar{I} + \mu) \pm \sqrt{(\beta \bar{I} + \mu)^2 - 4\beta^2 \bar{S} \bar{I}}}{2} \qquad (2.8)$$

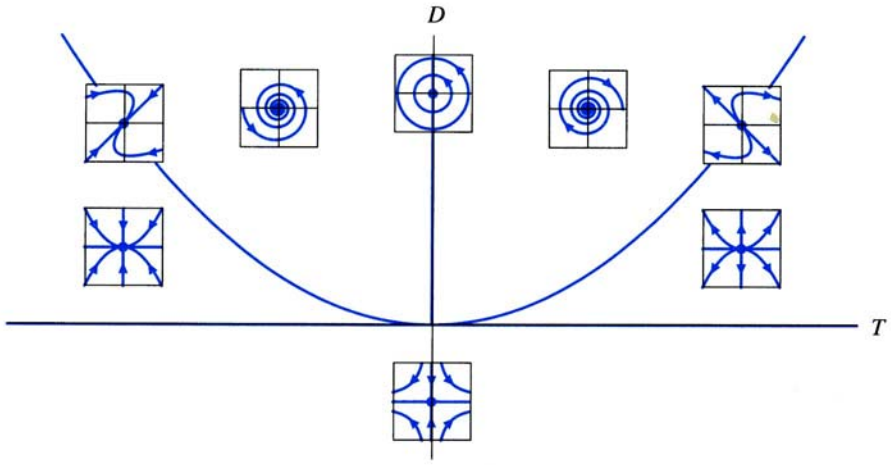

Fig 2.1 The trace-determinant plane (Blanchard, 2002, p. 337)

the eigenvalues of (2.5) have negative real part as shown in (2.8) . This means that the endemic steady state is stable. Note that the endemic state is present if the values of $\bar{S}$ and $\bar{I}$ are positive and also (2.4) is satisfied. This means the endemic state can only exist when the infection-free state is unstable (Clark, 2002b).

Note from (2.8), the radical $\pm\sqrt{(\beta\bar{I}+\mu)^2-4\beta^2\bar{S}\bar{I}}$ may be real or imaginary, thus the phase plane may have either a spiral sink or a sink depending on the value of the parameters. But research done in this area only focuses on the spiral sink, in that the term inside the radical is negative. This is so because it makes sense from a practical point of view, when the recovery rate is much larger than the death rate. In this case, after an epidemic runs its course (forcing the number of susceptible below the epidemic threshold) new susceptibles are born so that eventually a new epidemic cycle can begin. Diekmann, (2000) explained this point as follows:

Consider the characteristic equation (2.7)

$\lambda^2 + (\beta\bar{I} + \mu)\lambda + (\beta^2\bar{S}\bar{I}) = 0$, where

(a) $\bar{S} = \frac{\mu + r}{\beta}$.

(b) $\bar{I} = \frac{\mu}{\beta}(R_0 - 1)$

In the characteristic equation $\lambda^2 + (\beta\bar{I} + \mu)\lambda + (\beta^2\bar{S}\bar{I}) = 0$, dividing by μ^2 and using (a) and (b), we can rewrite this as

$$\left(\frac{\lambda}{\mu}\right)^2 + (R_0 - 1 + 1)\frac{\lambda}{\mu} + (R_0 - 1)\frac{\beta}{\mu}\frac{N}{R_0} = 0,$$

i.e.

$$\left(\frac{\lambda}{\mu}\right)^2 + R_0\frac{\lambda}{\mu} + \frac{r + \mu}{\mu}(R_0 - 1) = 0.$$

When $\frac{1}{\mu} >> \frac{1}{r}$, we have $\frac{r}{\mu} >> 1$, and consequently we approximate the last term by $\frac{r}{\mu}(R_0 - 1)$. The equation

$$y^2 + R_0 y + \frac{r}{\mu}(R_0 - 1) = 0$$

has roots

$$y = \frac{R_0 \pm \sqrt{R_0{}^2 - 4\frac{r}{\mu}(R_0 - 1)}}{2}$$

Using once more that $\frac{r}{\mu} >> 1$, we see that the expression under the square root is negative

(we are considering the endemic steady state, so implicitly we have assumed that R_0>1) and that the roots are, to first approximation,

$$\frac{\lambda}{\mu} = y = -\frac{R_0}{2} \pm i\sqrt{\frac{r}{\mu}(R_0 - 1)}.$$

So the relaxation time $\frac{1}{|\operatorname{Re}\lambda|}$ equals $\frac{2}{\mu R_0}$, while the frequency equals $\sqrt{\frac{r}{\mu}(R_0 - 1)}$, both to first approximation with respect to the small parameter $\frac{\mu}{r}$. For $\mu << r$, the relaxation time is of the order of $\frac{1}{\mu}$ but the period is of the order of $\frac{1}{\sqrt{\mu}}$ so the ratio between the two goes to infinity for $\mu \downarrow 0$. This means that we shall see many oscillations while the deviation from steady state is damping out (p. 205 – 206).

The model that we have discussed (S-I-R), brings forth two possibilities i.e. $R_0 < 1$ and when $R_0 > 1$. This is so because "the behavior is almost completely dependent on the threshold quantity R_0, which determines not only when the local stability of the disease-free equilibrium switches, but also when the endemic equilibrium enters the feasible region with a positive infective fraction" (Hethcote, 2000, p. 609). For this model, there is a bifurcation at

R_0 = 1. The following examples will illustrate this point in greater depth.

Case 1 $R_0 = 0.5 < 1$

$$\frac{dS}{dt} = -0.0175SI + 5000 - 5S \qquad (2.9a)$$

$$\frac{dI}{dt} = 0.0175SI - 5I - 30I \qquad (2.9b)$$

$$R_0 = \frac{\beta N}{(\mu + r)} = \frac{17.5}{35} = 0.5$$

In this case, the number of infectives will immediately decline after introduction of an infective agent when $R_0 < 1$, in this case, $R_0 = 0.5 < 1$. Using the maple output (see appendix 2.2), the jacobian matrix is:

$$jc := \begin{bmatrix} -0.0175\,In - 5 & -0.0175\,S \\ 0.0175\,In & 0.0175\,S - 35 \end{bmatrix}$$

and the equilibrium points; sp = {$In = I = 0.$, S = 1000.}

Therefore, $p1$: = eval (jc (S = 1000, I = 0))

$$p1 := \begin{bmatrix} -5. & -17.5000 \\ 0. & -17.5000 \end{bmatrix}$$

The eigenvalues of ($p1$) = $\lambda_1 = -5, \lambda_2 = -17.5$ (see appendix 2.2). Since $\lambda_1 < \lambda_2 < 0$, then the equilibrium point is a sink, which lie in the 2nd quadrant of the trace determinant plane (see Fig 2.1). The phase plane portrait for this system is shown in fig (2.2) below

(see appendix 2.3).

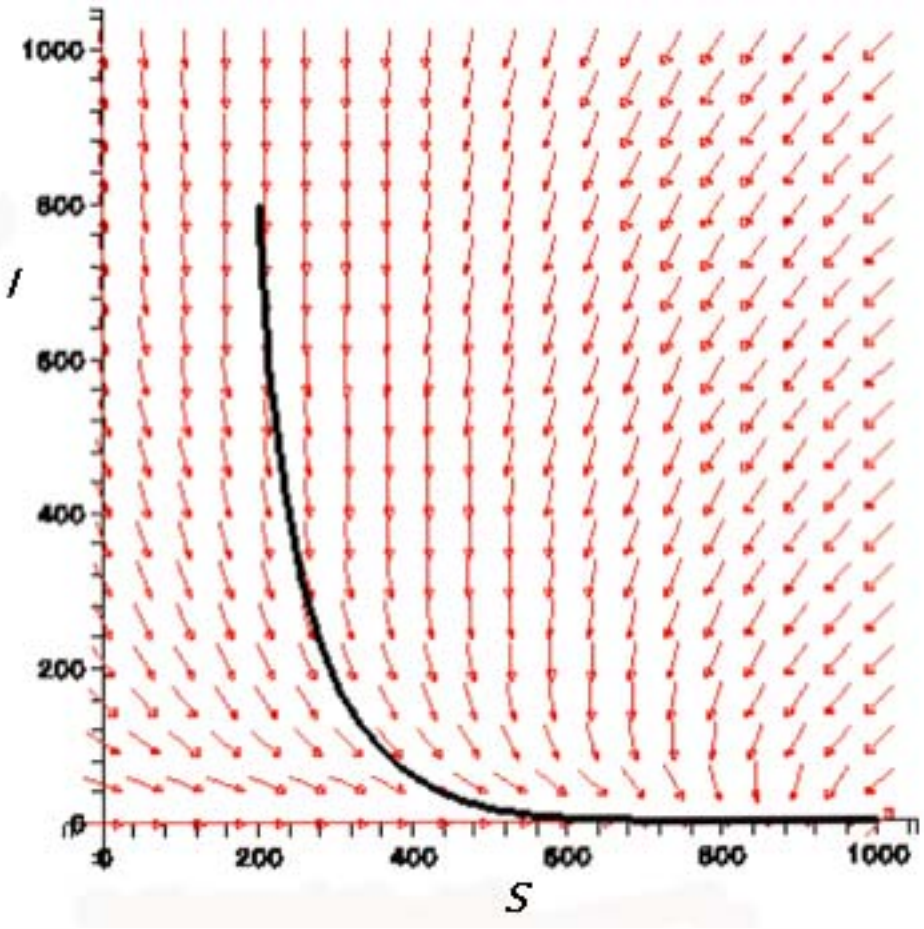

Fig 2.2 The phase plane portrait $R_0 = 0.5$

The *S(t)* and the *I(t)* plots are shown in fig 2.3 below was created by maple9.5 (see appendix 2.4).

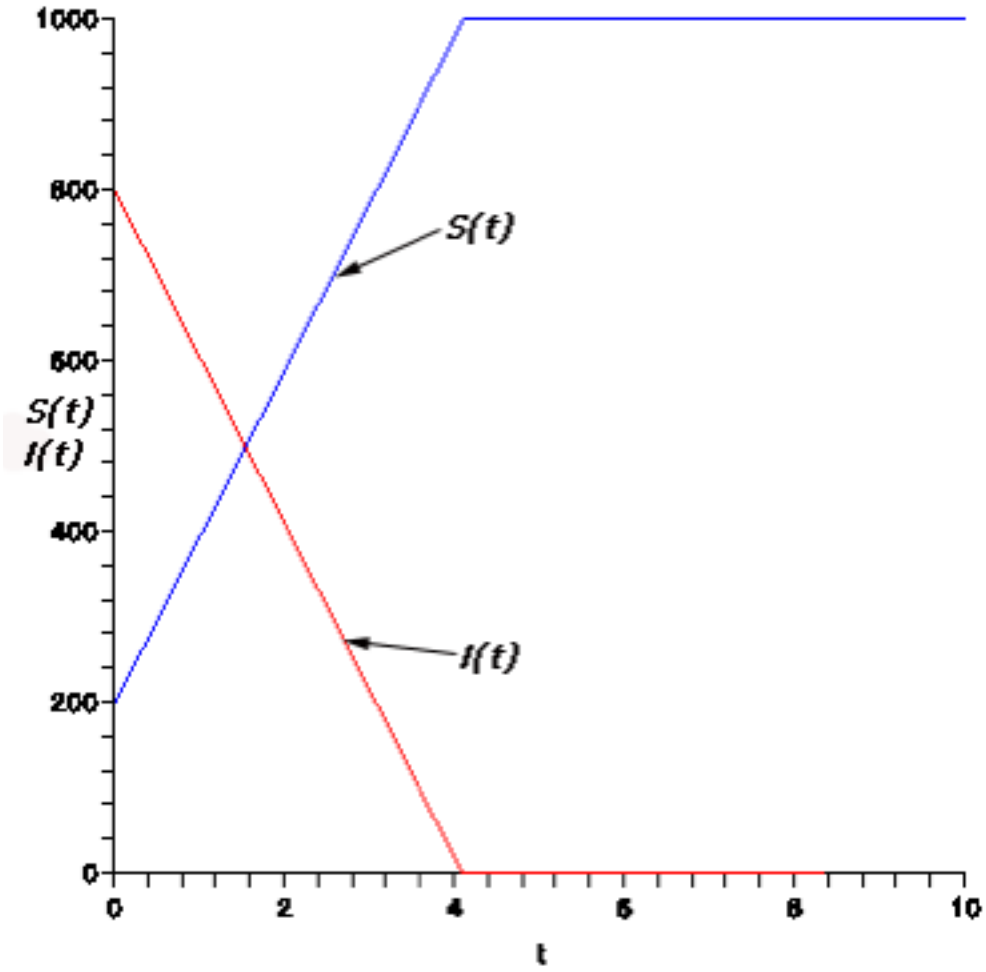

Fig 2.3 A graph of *S(t), I(t)* $R_0 = 0.5$

From the phase portrait (fig 2.2) and *S(t)* and *I(t)* graphs (fig 2.3), it can be noted that if $R_0 = 0.5 < 1$, then the infectives decrease to zero (Hethcote, 2000). For this model, the infective *(I)* are 800 whereas the suceptibles *(S),* are 200. In less then 5 years, all the initial infective have recovered from the 800 to zero whereas the initial susceptible have rapidly increased from 200 to 1000. Therefore it should be noted that if $R_0 < 1$ the solution path approaches the disease free equilibrium *(I, S) = (I, N) = (0, N).*

Case 2, $R_0 > 1$

This is the situation where an epidemic results when an infective agent is introduced into the susceptible population. This is a different scenario than what occurred when $R_0 < 1$. As Hethcote, (2000) explains that:

> if $R_0 = \sigma > 1$ (see appendix 2.5), I_0 is small and, and S_0 is large with $\sigma S_0 > 1$, then S(t) decreases and *I(t)* increases up to a peak and then decreases, just as it would for an epidemic. However after the infective has decreased to a low level, the slow process of the deaths of recovered people and the births of new susceptibles gradually (over 10 or 20 years) increase the susceptibles until $\sigma S(t)$ is large enough that another smaller epidemic σS_0 occurs. .. For this SIR model there is a transcritical (stability exchange) bifurcation at $\sigma = R_0 > 1$ (p. 608).

The following examples, will illustrate more what happens when $R_0 > 1$.

Example 1

$$\frac{dS}{dt} = -0.003SI + 50 - 0.05S \qquad (2.10a)$$

$$\frac{dI}{dt} = 0.003SI - I - 0.05I \qquad (2.10b)$$

$$R_0 = \frac{\beta N}{(\mu + r)} = \frac{(.003)(1000)}{(0.05 + 1)} = \frac{20}{7} = 2.857 > 1$$

Using the maple output (see appendix 2.6), the jacobian matrix is:

$$jc := \begin{bmatrix} -0.003\ In - 0.05 & -0.003\ S \\ 0.003\ In & 0.003\ S - 1.05 \end{bmatrix}$$

and the equilibrium points; sp = { S = 350., I = 30.95238095}

Therefore *pI*: = eval (jc(S = 350, I = 30.95238095) (see appendix 2.6)

$$p1 := \begin{bmatrix} -0.1428571428 & -1.050 \\ 0.09285714285 & 0. \end{bmatrix}$$

Hence the eigenvalues $\lambda = -0.07142857 \pm 0.30397I$

Since -0.07142857 < 0, then the equilibrium point is a spiral sink. The phase portrait for this system is shown in fig 2.4 below which was created by maple with initial conditions S(0) = 990, I(0) = 10 (see appendix 2.7)

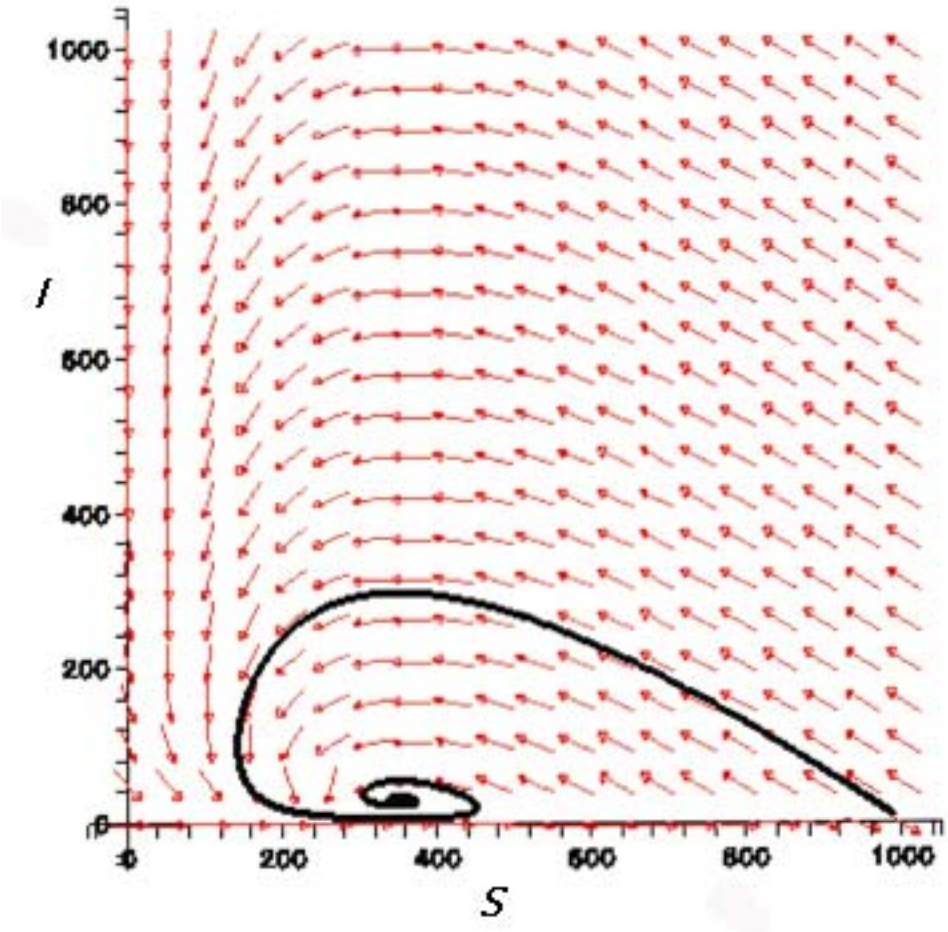

Fig 2.4 The phase plane portrait R_0 = 2.857

Fig 2.4 shows a speedy development of the epidemic, which then slows in a spiral form to the endemic state. According to maple output (see appendix 2.7) of fig 2.4, the

endemic state is *(S, I)* = (348.098, 31.532). This means that the number that recovered in this population is (1000 – 348 – 31) = 621.

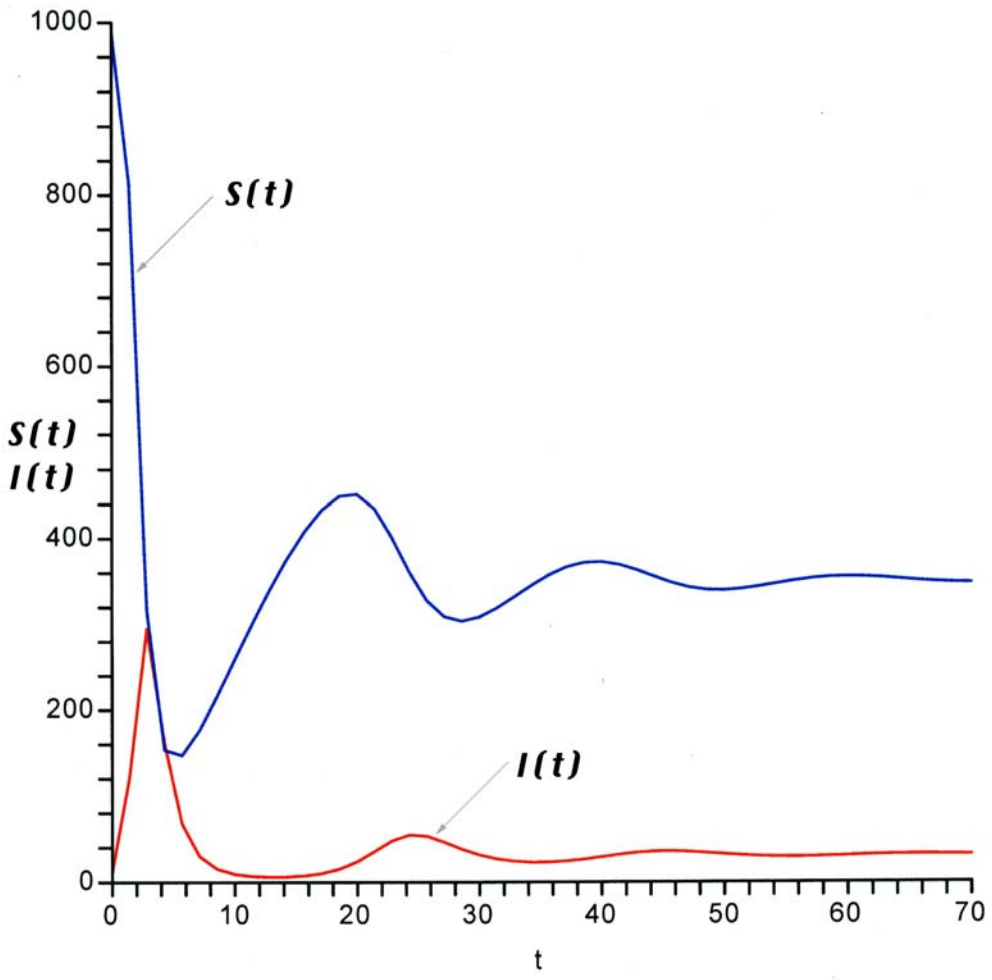

Fig 2.5 A Graph of *S(t) I(t)* R_0 = 2.857 (see appendix 2.8)

On the same note, the time plot, *S (t)* and *I (t)* (fig 2.5) shows a speedy development of the epidemic, which is then followed by damped oscillations leading to the endemic state (348.098, 31.532). In the endemic state, any perturbations will be damped oscillations. Since the eigenvalues of this system

are $\lambda = -0.07142857 \pm 0.30397I$, then the oscillation period is $\frac{2\pi}{.30397} = 20.67$, with frequency of $\frac{.30397}{2\pi} = 0.0483$

Example 2 (Clark, 2002b).

$$\frac{dS}{dt} = -0.05SI + 14.29 - 0.01429S \qquad (2.11a)$$

$$\frac{dI}{dt} = 0.05SI - 10.01429I \qquad (2.11b)$$

$$R_0 = \frac{\beta N}{(\mu + r)} = \frac{(0.05)(1000)}{(0.01429 + 10)} = \frac{50}{10.01429} = 4.992865196 > 1$$

(see appendix 2.8), the jacobian matrix is:

$$jc := \begin{bmatrix} -0.05\ In - 0.01429 & -0.05\ S \\ 0.05\ In & 0.05\ S - 10.01429 \end{bmatrix}$$

and equilibrium points: sp = {S = 200.2858, I = 1.141161).

Therefore pI: = eval (jc(S = 200.2858, I = 1.141161) (see appendix 2.8)

$$pI := \begin{bmatrix} -0.07134804365 & -10.01429000 \\ 0.05705804365 & 0. \end{bmatrix}$$

Hence the eigenvalues

$$eI := \begin{bmatrix} -0.03567402182499999922 + 0.7550650038974047 18\ I \\ -0.03567402182499999922 - 0.7550650038974047 18\ I \end{bmatrix}$$

Since -0.0356740..<0, the equilibrium point is a spiral sink. The phase portrait for this system is shown in fig 2.6 below created by maple with initial conditions S(0) = 998, I(0) = 2 (see appendix 2.9).

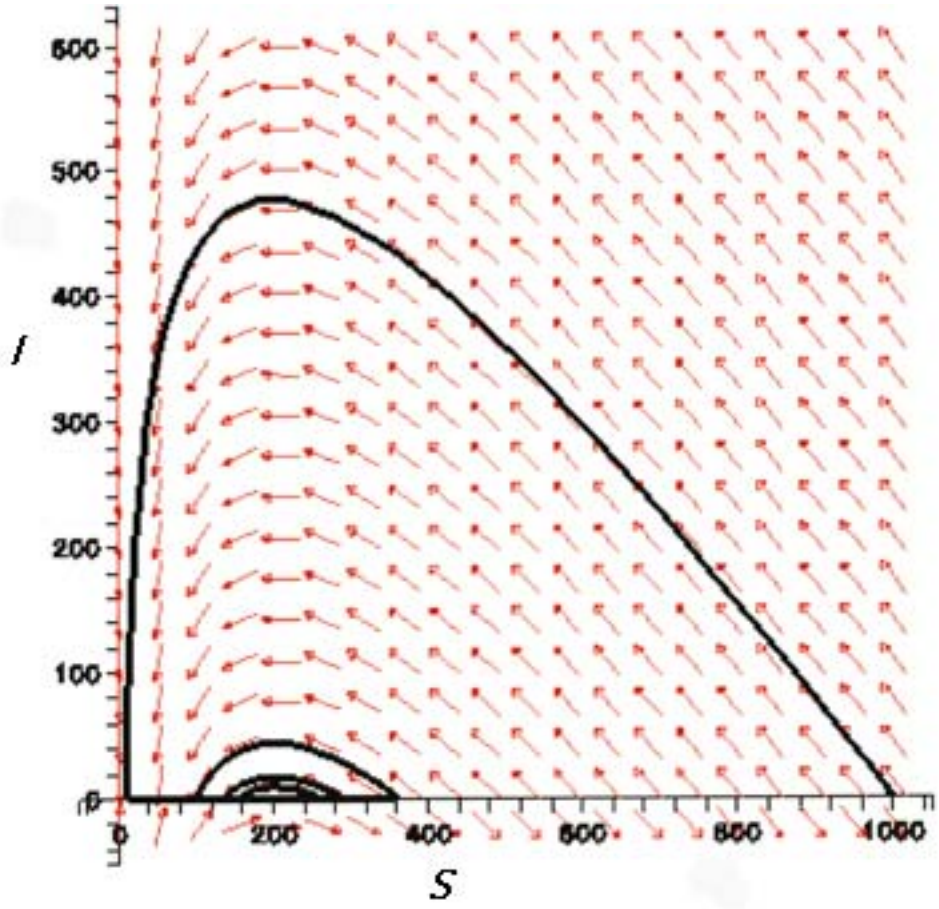

Fig 2.6 The phase plane portrait $R_0 = 4.99$

Interesting! What is going on in figure 2.6? Let's investigate this system by using the graphs *S(t)* and *I(t)* for system (2.11) (see appendix 2.10) in figure 2.7 below.

This rather striking curve can be described as a succession of epidemics, of ever-diminishing amplitude. Eventually, those oscillations disappear and the system settles in to the stable endemic equilibrium. The system requires 200 years to settle into the endemic state! The most characteristic feature of this system is the existence of two different time scales – the short time scale of a single epidemic, and the long generational time scale on which susceptibles are replenished (Clark, 2002b).

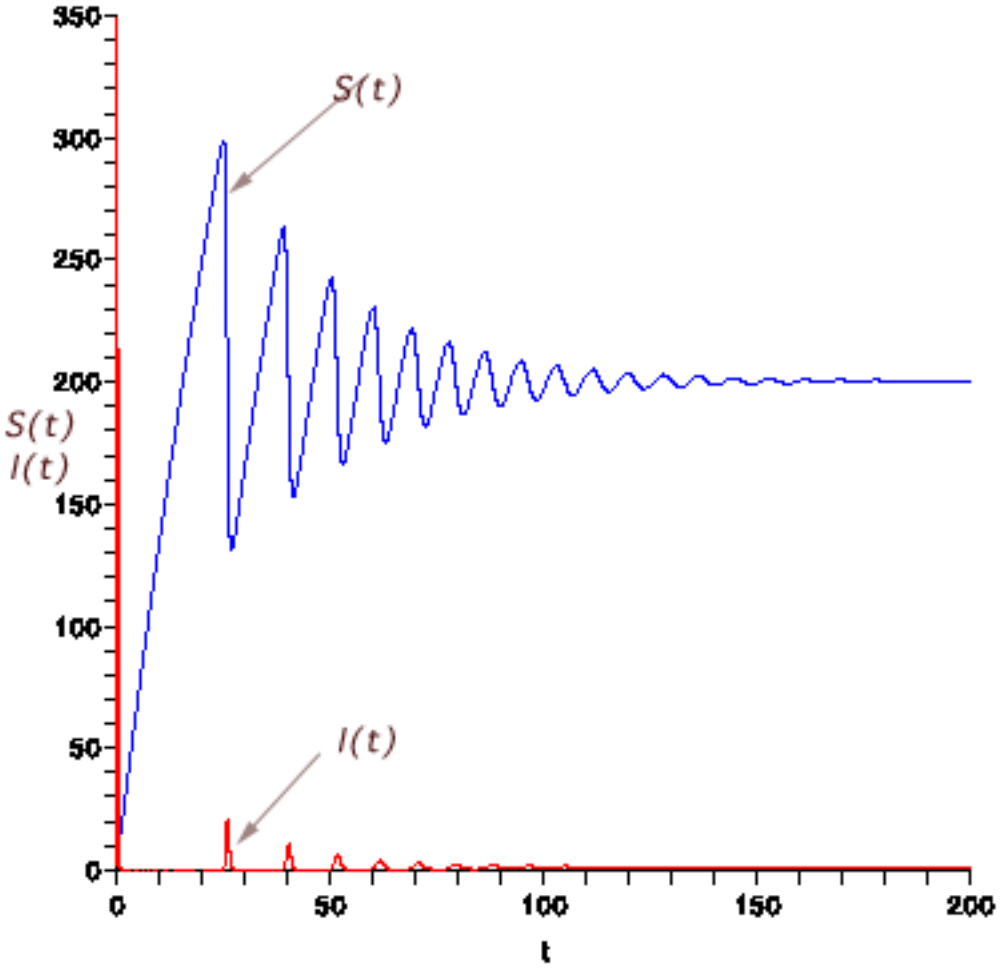

Fig 2.7 A Graph of *S(t), I(t)* R_0 = 4.99

Since the scale for *I(t)* needs to be big, figure 2.8 and 2.9 below will illustrate what is going on in figure 2.7 above.

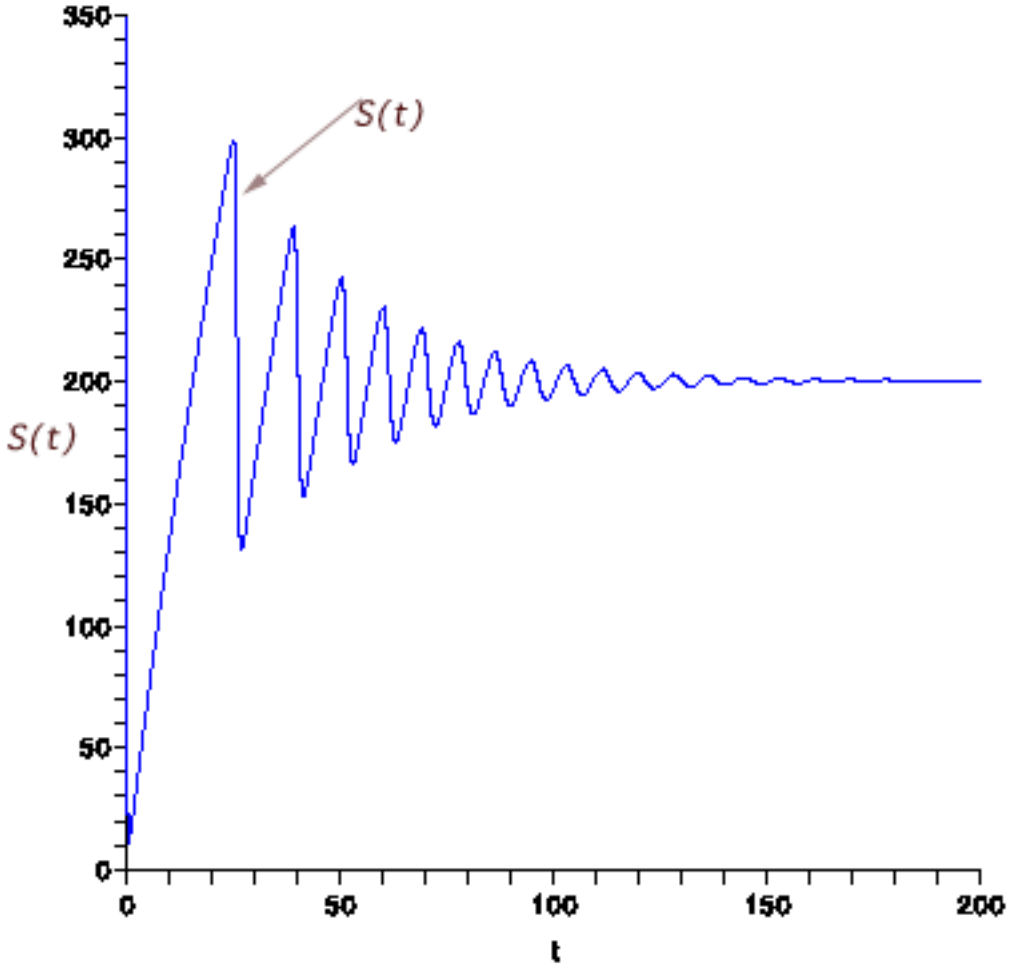

Fig 2.8 A Graph of $S(t)$ $R_0 = 4.99$

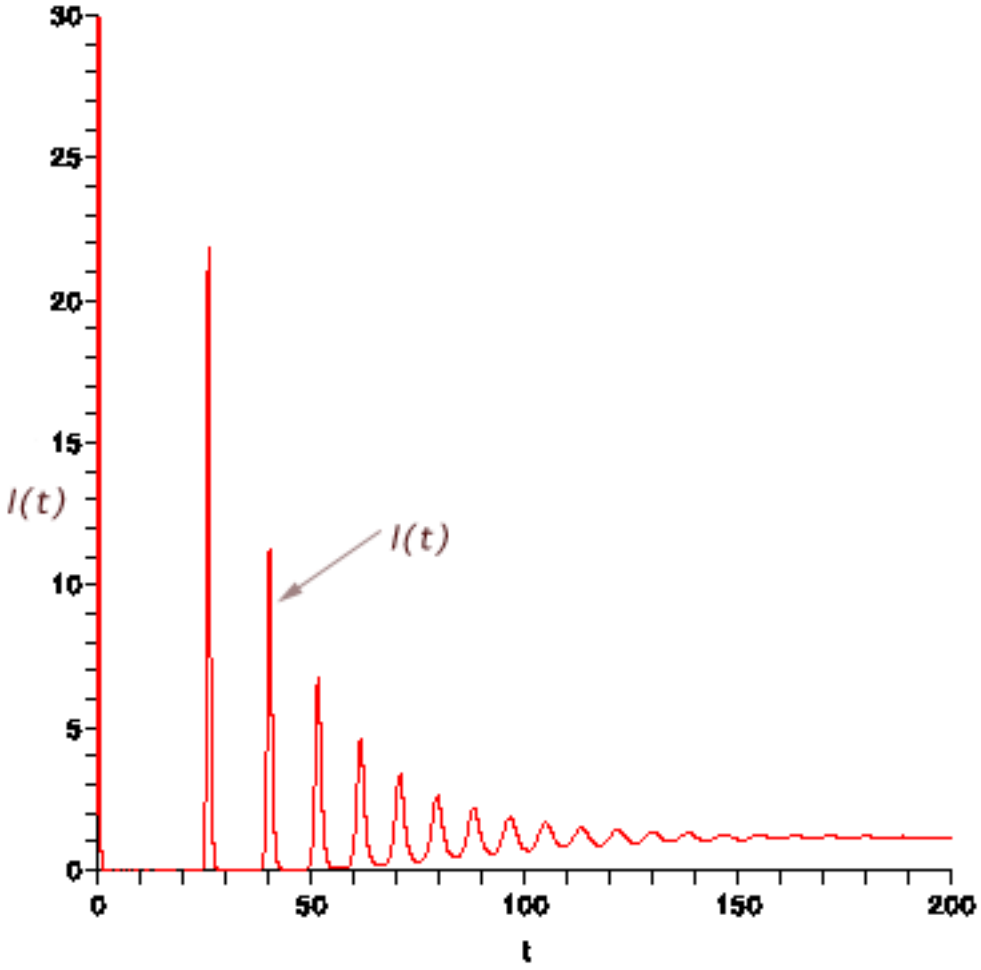

Fig 2.9 A Graph of $I(t)$ $R_0 = 4.99$

CHAPTER 4

MODEL III: PARVOVIRUS STRAINS IN DOGS AND WILD CARNIVORES

The S-I-R model (1.5) discussed above is a simple case of the model:

$$\frac{dS}{dt} = bN(1-\frac{N}{K}) - \beta S(I+F) - dS \qquad (3.1a)$$

$$\frac{dI}{dt} = \beta S(I+F) - I(r+d_i+d) \qquad (3.1b)$$

$$\frac{dR}{dt} = rI - dR \qquad (3.1c)$$

$$\frac{dF}{dt} = \delta I - \alpha F \qquad (3.1d)$$

Where:

β is the infection rate.

N = S(t) + I(t) + R(t)

r is the recovery rate

b is the birth rate

d is the death rate due to natural caurses

d_i is the death rate of the infectives

K is the carrying capacity

F, α and δ will be discussed below.

This model was discussed in Fiorello, (2004) in which she discussed canine parvovirus (CPV) that affect domestic dogs in Isoso region of the Bolivian Chaco. She was particularly interested in the transmission of disease to wild animals. Three conditions

(present in Bolivian Chaco) that are vital for the disease to be passed over from domestic animals to wild animals are: (1) the domestic animal must have the pathogen that cause the disease. (2) The pathogen must be able to cause disease in the wild animals (3) there should be a medium that allows the transmission of the disease.

Condition (3) needs extra attention and careful consideration. There are two ways in which disease can spillover from domestic animals to the wild animals. One, pathogen can spillover when the infective come into contact with the susceptible. But this condition can be tricky especially, when the animals involved avoid each other. Secondly, the pathogens can be transmitted through feces. As Fiorello noted:

> …pathogens that survive in the environment for a period of time may be transmitted even if individuals from the two populations never meet. Because carnivores often use urine and feces to mark territories and communicate with conspecifics, they are motivated to investigate such materials and therefore may be exposed to infectious agents shed in excreta (p. 153).

For that matter, equation (3.1d), has the parameters F, α and δ in which F is the fecal density per km^2, δ is the fecal accumulation and α is the fecal decay. Just as Fiorello (2004) notes "In the equation describing fecal accumulation and decay, we assume that accumulation is linearly proportional to the number of infectious animals with fecal production rate δ and that decay is exponential, with decay constant, α" (p. 163). In this model, it is considered that nearly all domestic dogs are seropositive and are never vaccinated. The locals use of dogs for hunting makes the two conditions present in the Bolivian Chaco.

*Assumptions and parameter values (*Fiorello, 2004)

- Dogs were exposed to CPV
- Once Dogs recover from the infection, they recover for life

$b = \frac{3}{365}$

$d = \frac{1}{8 * 365}$

$\beta = 0.01$

$d_i = 0.01$

$r = 0.09$

$\alpha = \frac{1}{90}$

$\delta = 1$

In this model, the behavior of solutions was studied as a function of the bifurcation parameter, K.

While investigating model 3.1 above, a typo occurred in (3.1a) by which the first N was replaced with S as shown below. This model although it does not represent parvovirus, we discovered shows interesting behavior, limit cycles, that were not present in the original model. This model may be applicable to some diseases. The assumption made in this model is that once dogs recover, they cannot reproduce.

$$\frac{dS}{dt} = bS(1-\frac{N}{K}) - \beta S(I+F) - dS - v$$

$$\frac{dI}{dt} = \beta S(I+F) - I(r + d_i + d)$$

$$\frac{dR}{dt} = rI - dR$$

$$\frac{dF}{dt} = \delta I - \alpha F$$

We now use maple to show the behavior of this system with the above values for the parameters, and various choices for K.

Case 1, K = 0.9

This yields

$$\frac{dS}{dt} = \frac{3}{365}S(1-\frac{N}{0.9}) - 0.01S(I+F) - \frac{1}{8*365}S \qquad (3.2a)$$

$$\frac{dI}{dt} = 0.01S(I+F) - I(0.09 + 0.01 + \frac{1}{8*365}) \qquad (3.2b)$$

$$\frac{dR}{dt} = 0.09I - \frac{1}{8*365}R \qquad (3.2c)$$

$$\frac{dF}{dt} = I - \frac{1}{90}F \qquad (3.2d)$$

Using the maple output, (See appendix 3.1), the jacobian matrix is

$$jc := \begin{bmatrix} -0.01826\,S - 0.01913\,In - 0.009132\,R + 0.007876 - 0.01\,F & -0.01913\,S & -0.009132\,S & -0.01\,S \\ 0.01\,In + 0.01\,F & -0.1003424658 + 0.01\,S & 0 & 0.01\,S \\ 0 & 0.09 & \frac{-1}{2920} & 0 \\ 0 & 1 & 0 & \frac{-1}{90} \end{bmatrix}$$

and the equilibrium points; sp3 = { I = 0.002069731470, S = 0.1102664459, F = 0.1862758323, R = 0.5439254303}

Therefore, P3 = eval (jc(I = 0.002069731470, S = 0.1102664459, F = 0.1862758323, R = 0.5439254303)) (see appendix 3.1) , therefore,

$$p3 := \begin{bmatrix} -0.001006999505 & -0.002109663965 & -0.001006999506 & -0.001102664459 \\ 0.001883455638 & -0.09923980134 & 0 & 0.001102664459 \\ 0 & 0.09 & \frac{-1}{2920} & 0 \\ 0 & 1 & 0 & \frac{-1}{90} \end{bmatrix}$$

The eigenvalues e3 are:

$$e3 := \begin{bmatrix} -0.1105026454649221 11 + 0.\ I \\ -0.0000286785135819749916 + 0.0045622616844040 1151\ I \\ -0.0000286785135819749916 - 0.0045622616844040 1151\ I \\ -0.0011403752174498 5988 + 0.\ I \end{bmatrix}$$

Since the real parts of all of the eigenvalues are negative, the equilibrium point is a spiral sink. This can be shown by the graph *S(t), I(t), F(t) and R(t)* of figure 3.3 below which was created by maple (see appendix 3.2)

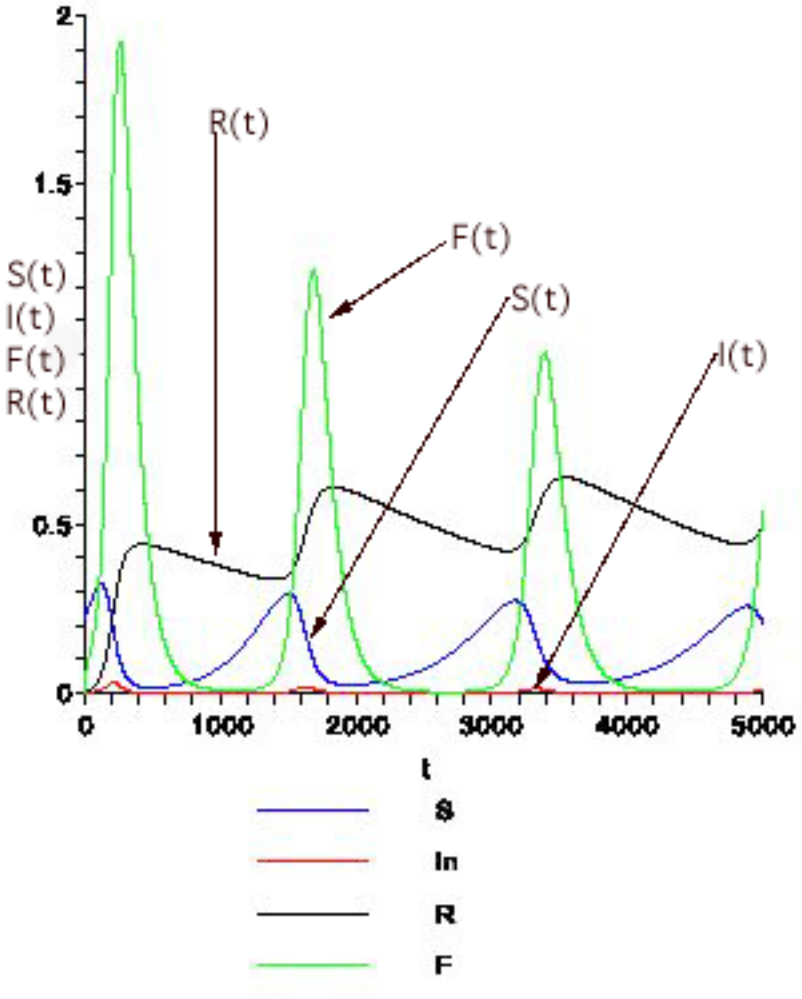

Fig 3.1 A Graph of *R(t), F(t), S(t), I(t),* K = 0.9

Since we cannot see what is going on with *I(t),* let us choose a different scale below:

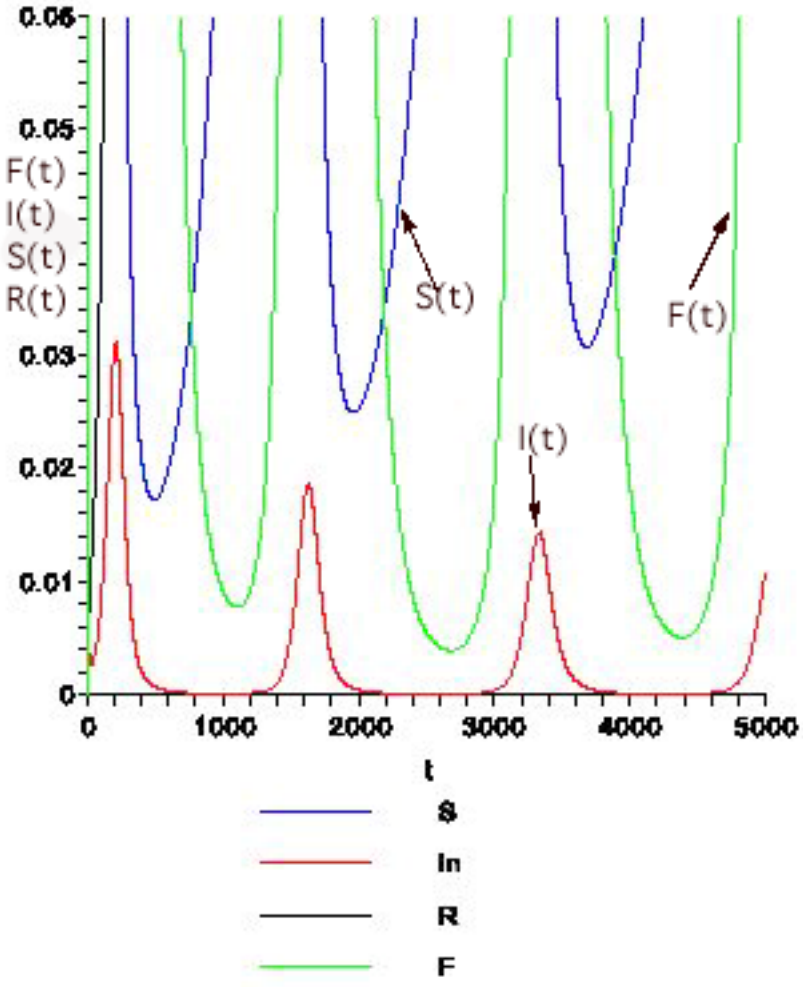

Fig 3.2 A Graph of *R(t), F(t), S(t), I(t)* K = 0.9

The following diagram (Fig 3.3 & 3.4) shows the damping oscillation of the system at late times.

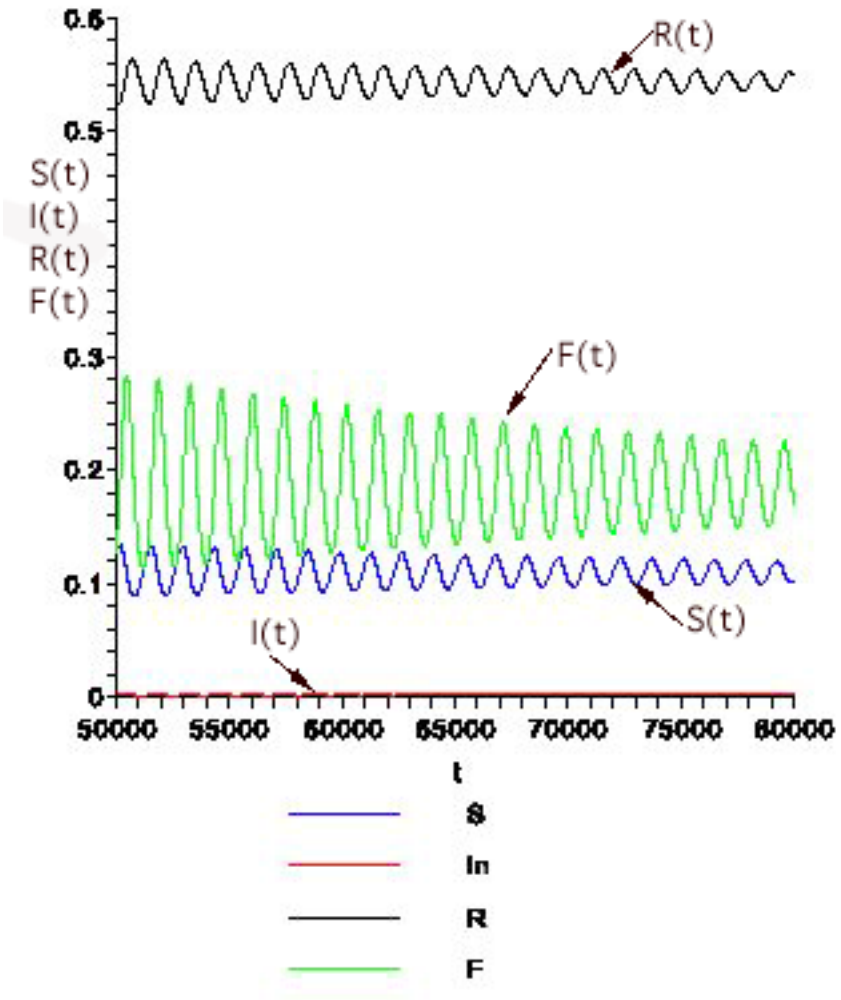

Fig 3.3 A Graph of *R(t), F(t), S(t), I(t),* K = 0.9

It should be remarked that although these oscillations are damped, many cycles occur before the amplitude diminish considerably. Thus, in the short term, the oscillatory nature is far more important than the damping.

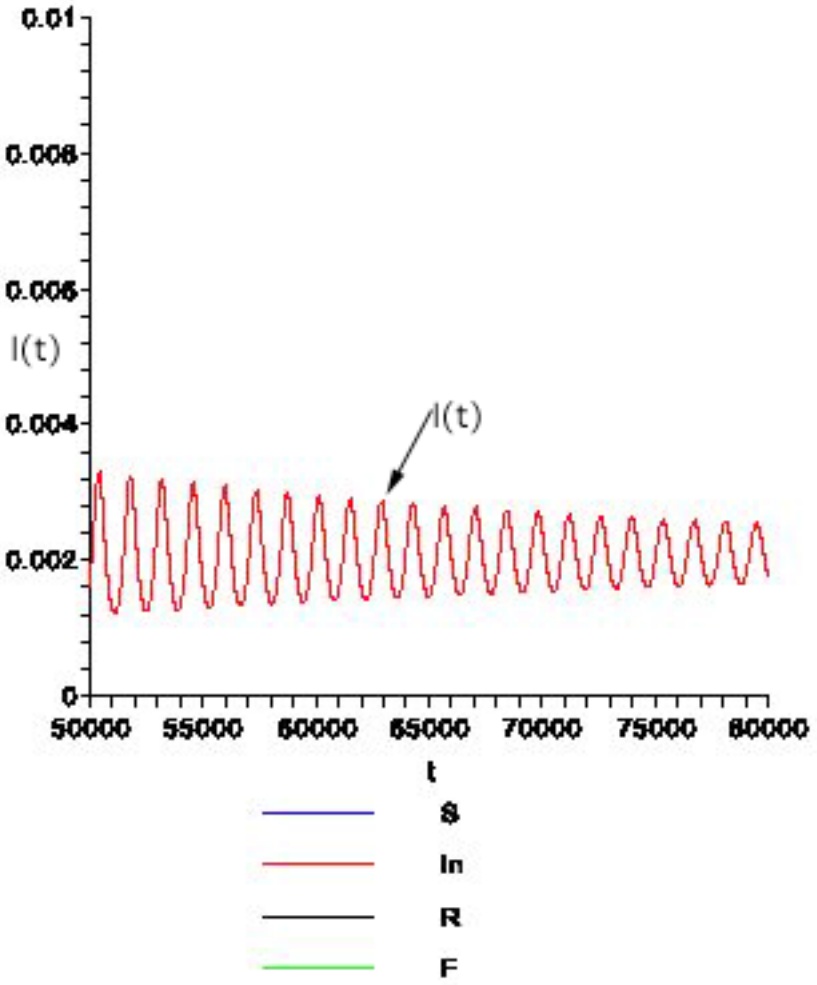

Fig 3.4 A Graph of *I(t)* K = 0.9

Case 2 K = 1.5

$$\frac{dS}{dt} = \frac{3}{365} S(1 - \frac{N}{1.5}) - 0.01S(I + F) - \frac{1}{8 * 365} S \qquad (3.3a)$$

$$\frac{dI}{dt} = 0.01S(I + F) - I(0.09 + 0.01 + \frac{1}{8 * 365}) \qquad (3.3b)$$

$$\frac{dR}{dt} = 0.09I - \frac{1}{8 * 365} R \qquad (3.3c)$$

$$\frac{dF}{dt} = I - \frac{1}{90} F \qquad (3.3d)$$

Using the maple output, (see appendix 3.3), the jacobian matrix is

$$jc := \begin{bmatrix} -0.008\,S - 0.02\,In - 0.006\,R + 0.008 - 0.01\,F & -0.02\,S & -0.006\,S & -0.01\,S \\ 0.01\,In + 0.01\,F & -0.1003424658 + 0.01\,S & 0 & 0.01\,S \\ 0 & 0.09 & \frac{-1}{2920} & 0 \\ 0 & 1 & 0 & \frac{-1}{90} \end{bmatrix}$$

The equilibrium points; sp3 =

$$\{F = 0.2778738467,\ In = 0.003087487186,\ S = 0.1102664459,\ R = 0.8113916324\}$$

Which means that

$$p3 := \begin{bmatrix} -0.000604199703 & -0.001706864163 & -0.0006041997036 & -0.001102664459 \\ 0.002809613339 & -0.09923980134 & 0 & 0.001102664459 \\ 0 & 0.09 & \frac{-1}{2920} & 0 \\ 0 & 1 & 0 & \frac{-1}{90} \end{bmatrix}$$

and the eigenvalues e3 are:

$$e3 := \begin{bmatrix} -0.1105777847875264 31 + 0.\ I \\ 0.0000589992817215470938 + 0.0054745037991044 1754\ I \\ 0.0000589992817215470938 - 0.0054745037991044 1754\ I \\ -0.0008377916834524 36087 + 0.\ I \end{bmatrix}$$

As indicated, the real parts of the two complex eigenvlaues are positive which means that the equilibrium point is a spiral source in a two dimensional attracting surface. This can be shown in fig 3.5 and 3.6 below

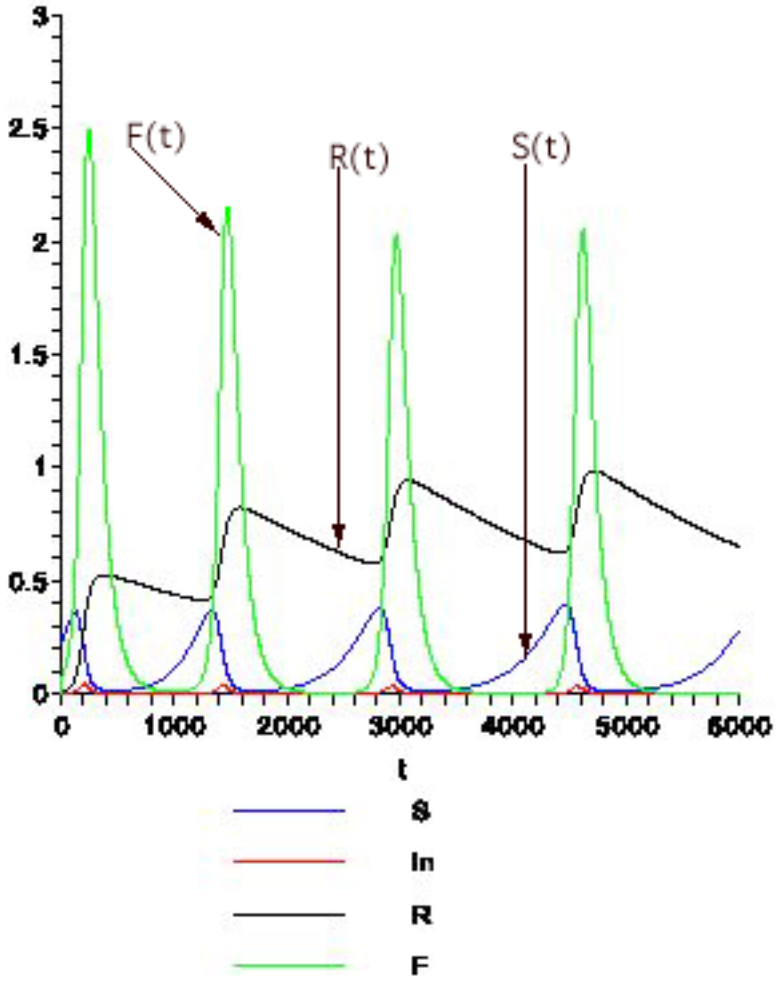

Fig 3.5 A Graph of *R(t), F(t), S(t), I(t)*, K = 1.5

Since the scale for Fig 3.5 does not show us what happens with *I(t),* I modified the scale as show in Fig 3.6 below.

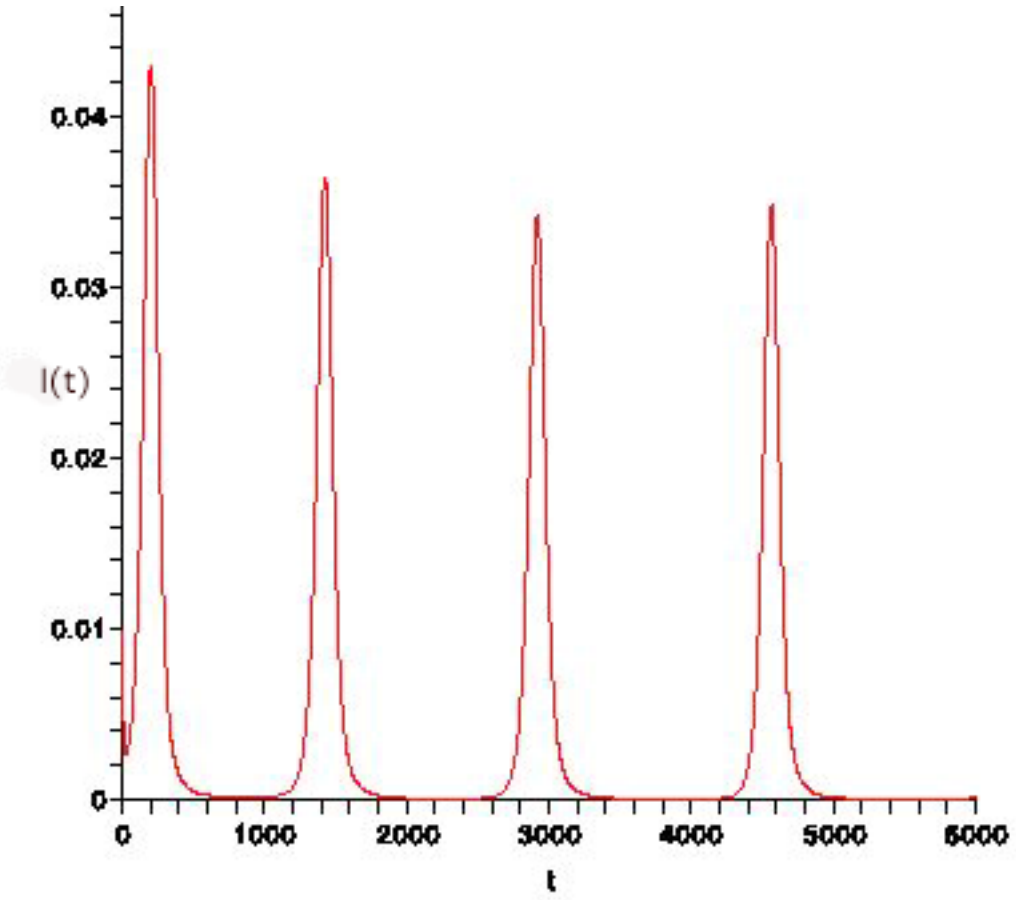

Fig 3.6 A Graph of *I(t),* K = 1.5

Fig 3.7 (See appendix 3.4 for maple file) and 3.8 below shows what happens in the long run in this model

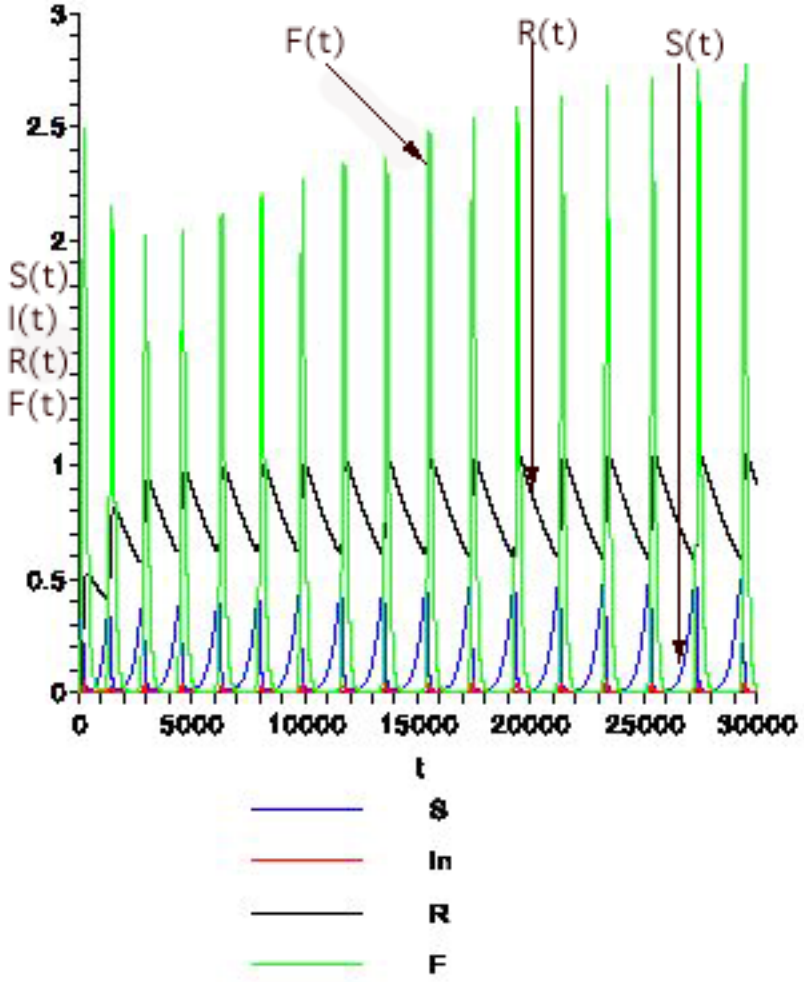

Fig 3.7 A Graph of *R(t), F(t), S(t), I(t),* K = 1.5

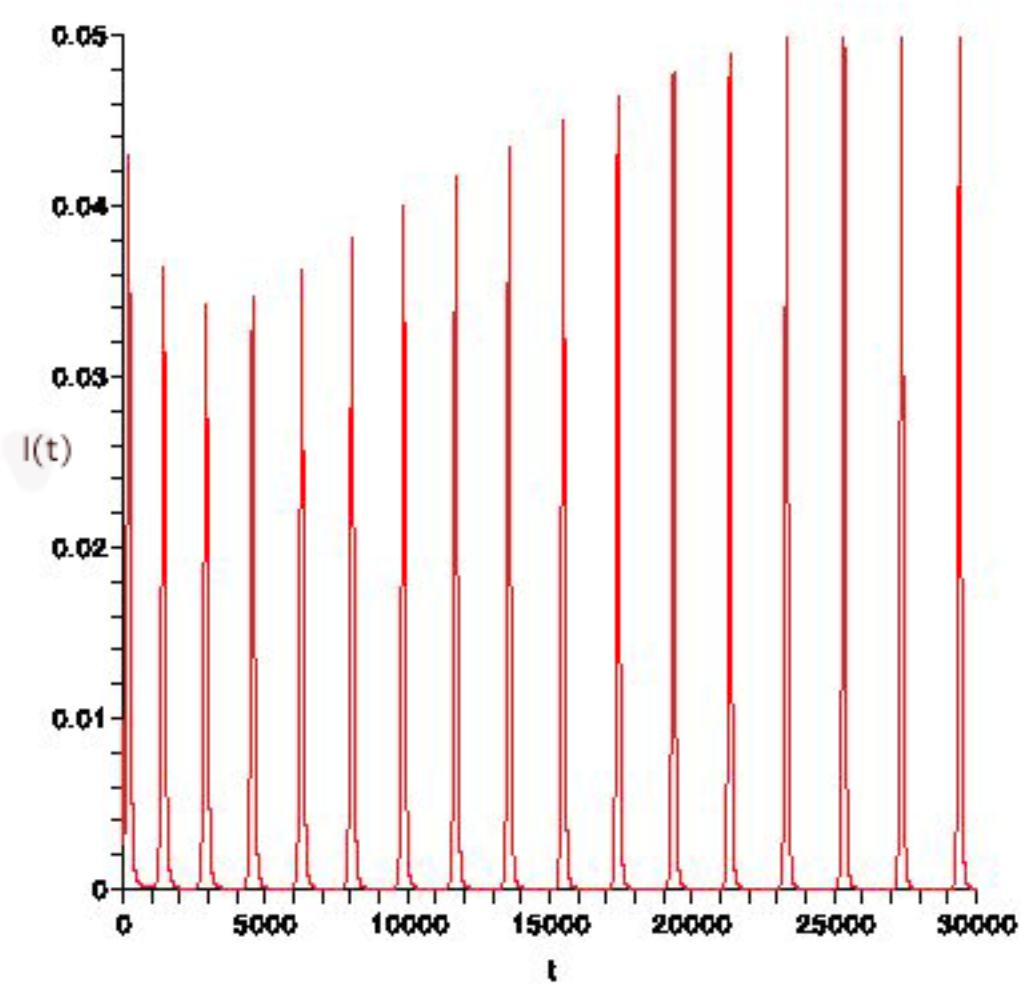

Fig 3.8 A Graph of *I(t),* K = 1.5

Figure 3.7 shows the dynamics of equation (3.3) over a period of 30000 days (82 years). The oscillation for *S(t), I(t) , F(t) and R(t)* will continue over the period and eventually its respective amplitude become damped. But the oscillation does not eventually die out but oscillated forever (Fiorello, 2004). It is evident that something very interesting occurs between K = 0.9 and K = 1.5. For K = 0.9, the eigenvalues are:

$$e3 := \begin{bmatrix} -0.1105026454649221 11 + 0.\,I \\ -0.00002867851358197499 16 + 0.00456226168440401151\,I \\ -0.00002867851358197499 16 - 0.00456226168440401151\,I \\ -0.0011403752174498598 8 + 0.\,I \end{bmatrix}$$

Whereas the eigenvalues for K = 1.5 are:

$$e3 := \begin{bmatrix} -0.1105777847875264 31 + 0.\,I \\ 0.00005899928172154709 38 + 0.00547450379910441754\,I \\ 0.00005899928172154709 38 - 0.00547450379910441754\,I \\ -0.0008377916834524360 87 + 0.\,I \end{bmatrix}$$

The real part of the complex eignevalues change from negative to positive as shown in e3 for K = 0.9 and K = 1.5 above. Making small changes in K can nail down the point of transition. From my investigation, I found that the point of interest are when K = 1.04 and 1.05

For K = 1.04 the eignevalues (See Appendix 3.5) are:

$$e3 := \begin{bmatrix} -0.1105225472523045 09 + 0.\,I \\ -9.042370716943930 24\ 10^{-7} + 0.00482284276253261250\,I \\ -9.042370716943930 24\ 10^{-7} - 0.00482284276253261250\,I \\ -0.0010404643580880440 8 + 0.\,I \end{bmatrix}$$

Whereas K = 1.05 the eigenvalues (See appendix 3.6) also are:

$$e3 := \begin{bmatrix} -0.1105239064028348 68 + 0.\,I \\ 8.38392515013593975\ 10^{-7} + 0.00484004914992496906\,I \\ 8.38392515013593975\ 10^{-7} - 0.00484004914992496906\,I \\ -0.0010342910207308795 9 + 0.\,I \end{bmatrix}$$

What then goes on inbetween when K = 1.04 and 1.05?

Hopf Birfurcation

Hopf birfurcation is the bifurcation of a fixed point to a limit cycle (Tabor, 1989 P. 197). What we have shown so far is that we have two type of equilibrium points. One is the spiral source whereas the other is spiral sink. For K = K_1, negative real parts result to a spiral sink. One the other hand, for K = K_2, positive real part results to a spiral source. To proof for the existence of hopf birfurcation, we can try to proof that N(t) is bounded.

$$\frac{dS}{dt} = bS(1-\frac{N}{K}) - \beta S(I+F) - dS$$

$$\frac{dI}{dt} = \beta S(I+F) - I(r+d_i+d)$$

$$\frac{dR}{dt} = rI - dR$$

$$\frac{dS}{dt} = bS(1-\frac{N}{K}) - dN - d_iI$$

If N get bigger than K, then

$N < \max(N(0), K)$

Hence N is bounded.

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APPENDICES

1.1

```
> restart:with(DEtools):with(plots):
Warning, the name changecoords has been redefined
> de1:= D(S)(t)=-beta*S(t)*In(t);
                                    de1 := D(S)(t) = -β S(t) In(t)
> de2:= D(In)(t)= (beta*S(t)-r)*In(t);
                                    de2 := D(In)(t) = (β S(t) - r) In(t)
> beta:=.3; r:=.6;
                                    β := 0.3
                                    r := 0.6
> PP1:=phaseportrait([de1,de2],[S(t),In(t)],t=0..50,
  [[S(0)=4,In(0)=.02]],S=0..6,In=0..2,linecolor=black,arrows=medium,stepsize=.01):
> PP2:=phaseportrait([de1,de2],[S(t),In(t)],t=0..50,
  [[S(0)=3,In(0)=.02]],S=0..6,In=0..2,linecolor=black,arrows=medium,stepsize=.01):
> PP3:=phaseportrait([de1,de2],[S(t),In(t)],t=0..50,
  [[S(0)=5,In(0)=.02]],S=0..6,In=0..2,linecolor=black,arrows=medium,stepsize=.01):
> display({PP1,PP2,PP3});
>
```

1.2a

```
> restart;with(LinearAlgebra):
  with(VectorCalculus):
  with(plots):
> N:=S+In+R;
```

$$N := S + In + R$$

```
> eqn1:= -beta*S*In;
```

$$eqn1 := -\beta\, S\, In$$

```
> eqn2:= (beta*S-r)*In;
```

$$eqn2 := (\beta\, S - r)\, In$$

```
>
> jc := Jacobian([eqn1, eqn2], [S, In]);
```

$$jc := \begin{bmatrix} -\beta\, In & -\beta\, S \\ \beta\, In & \beta\, S - r \end{bmatrix}$$

```
> sp:=solve({eqn1=0, eqn2=0},{S,In});
```

$$sp := \{In = 0, S = S\}$$

```
> p1:=eval(jc,[S=subs(sp[1],S), In=subs(sp[1],In)]);
```

$$p1 := \begin{bmatrix} 0 & -\beta\, S \\ 0 & \beta\, S - r \end{bmatrix}$$

```
> e1 := Eigenvectors(p1);
```

$$e1 := \begin{bmatrix} 0 \\ \beta\, S - r \end{bmatrix}, \begin{bmatrix} 1 & -\dfrac{\beta\, S}{\beta\, S - r} \\ 0 & 1 \end{bmatrix}$$

1.2b

```
> restart:with(plots):with(plottools):with(DEtools):
> N:=S(t)+In(t)+R(t);
```

$$N := S(t) + In(t) + R(t)$$

```
> eqn1:= diff(S(t),t)=-beta*S(t)*In(t), S(0)=ic1;
```

$$eqn1 := \frac{d}{dt} S(t) = -\beta\, S(t)\, In(t)\,,\ S(0) = ic1$$

```
> eqn2:= diff(In(t),t)=((beta*S(t)-r))*In(t), In(0)=ic2;
```

$$eqn2 := \frac{d}{dt} In(t) = (\beta\, S(t) - r)\, In(t)\,,\ In(0) = ic2$$

```
> eqn3:=diff(R(t),t)=r*In(t), R(0)=ic3;
```

$$eqn3 := \frac{d}{dt} R(t) = r\, In(t)\,,\ R(0) = ic3$$

```
> r:=.9;
```

$$r := 0.9$$

```
> beta:=.0067;
```

$$\beta := 0.0067$$

```
> ic1:=400; ic2:=7; ic3:=0;
```

$$ic1 := 400$$

$$ic2 := 7$$

$$ic3 := 0$$

```
> dsol:=dsolve([eqn1, eqn2, eqn3], numeric);
```

$$dsol := \mathbf{proc}(x\_rkf45)\ \ldots\ \mathbf{end\ proc};$$

```
> odeplot(dsol, [[t, S(t), color = blue], [t, In(t), color = red], [t, R(t),
  color = green]], t=0..10, view = [0..10, 0..500]);
```

2.1

R_0 ≔ expected number of secondary cases per primary case in a virgin' population. In other words, R_0 is the initial growth rate (more accurately: multiplication factor; note that R_0 is dimensionless) when we consider the population on a generation basis (with 'infecting another host' likened to 'begatting a child'). Consequently, R_0 has threshold value 1, in the sense that an epidemic will result from the introduction of the in the infective agents when R_0>1, while the number of inflecteds is expected to decline (on a generation basis) right after the introduction when R_0<1. The advantage of measuring growth on a generation basis is that for many models one has an explicit expression for R_0 in terms of the parameters (Diekmann, 2000, p. 4)

2.2

```
> restart;with(LinearAlgebra):
  with(VectorCalculus):
  with(plots):
> eqn1:= -0.0175*S*In + 5000 - 5*S;
> eqn2:= 0.0175*S*In - 5*In - 30*In;
> jc := Jacobian([eqn1, eqn2], [S, In]);
> sp:=solve({eqn1=0, eqn2=0},{S,In});
> p1:=eval(jc,[S=subs(sp[1],S), In=subs(sp[1],In)]);
> e1 := Eigenvectors(p1);
>
```

$$eqn1 := -0.0175\,S\,In + 5000 - 5\,S$$

$$eqn2 := 0.0175\,S\,In - 35\,In$$

$$jc := \begin{bmatrix} -0.0175\,In - 5 & -0.0175\,S \\ 0.0175\,In & 0.0175\,S - 35 \end{bmatrix}$$

$$sp := \{In = 0.,\ S = 1000.\},\ \{S = 2000.,\ In = -142.8571429\}$$

$$p1 := \begin{bmatrix} -5. & -17.5000 \\ 0. & -17.5000 \end{bmatrix}$$

$$e1 := \begin{bmatrix} -5. + 0.\,I \\ -17.5000000000000000 + 0.\,I \end{bmatrix}, \begin{bmatrix} 1. + 0.\,I & 0.813733471206735004 + 0.\,I \\ 0. + 0.\,I & 0.581238193719096463 + 0.\,I \end{bmatrix}$$

2.3

```
> restart:with(DEtools):with(plots):
Warning, the name changecoords has been redefined
> de1:= D(S)(t)=-beta*S(t)*In(t)+mu*N-mu*S(t);
```

$$de1 := D(S)(t) = -\beta\, S(t)\, In(t) + \mu\, N - \mu\, S(t)$$

```
> de2:= D(In)(t)= (beta*S(t)-r-mu)*In(t);
```

$$de2 := D(In)(t) = (\beta\, S(t) - r - \mu)\, In(t)$$

```
> mu:= 5;
```

$$\mu := 5$$

```
> N:=1000;
```

$$N := 1000$$

```
> beta:=(17.5)*10^(-3); r:=30;
```

$$\beta := 0.01750000000$$

$$r := 30$$

```
> R0 = beta*N/(mu + r);
```

$$R0 = 0.5000000000$$

```
> PP1:=phaseportrait([de1,de2],[S(t),In(t)],t=0..70,
  [[S(0)=200,In(0)=800]],S=-1..1000,In=-1..1000,linecolor=black,arrows=mediu
  m,stepsize=.01):

> display({PP1});
```

2.4

```
> restart:with(plots):with(plottools):with(DEtools):
Warning, the name changecoords has been redefined
Warning, the assigned name arrow now has a global binding
Warning, the previous binding of the name translate has been removed and it
now has an assigned value
>
> eqn1:= diff(S(t),t) = -beta*In(t)*S(t)+mu*N-mu*S(t), S(0)=ic1;
```

$$eqn1 := \frac{d}{dt} S(t) = -\beta\, In(t)\, S(t) + \mu\, N - \mu\, S(t),\ S(0) = ic1$$

```
> eqn2:= diff(In(t),t) = beta*S(t)*In(t)- mu*In(t)-r*In(t),In(0)=ic2;
```

$$eqn2 := \frac{d}{dt} In(t) = \beta\, In(t)\, S(t) - \mu\, In(t) - r\, In(t),\ In(0) = ic2$$

```
> beta:=(17.5)*10^(-3);
```

$$\beta := 0.01750000000$$

```
> mu:= 5;
```

$$\mu := 5$$

```
> r:=30;
```

$$r := 30$$

```
> ic1:=200; ic2:=800;
```

$$ic1 := 200$$

$$ic2 := 800$$

```
> N:=ic1+ic2;
```

$$N := 1000$$

```
> R0 = beta*N/(mu + r);
```

$$R0 = 0.5000000000$$

```
> dsol:=dsolve([eqn1, eqn2], numeric);
dsol := proc(x_rkf45) ... end proc;
> dsol:=dsolve([eqn1, eqn2], numeric);
dsol := proc(x_rkf45) ... end proc;
> odeplot(dsol,[[t, S(t), color = blue],[t,In(t), color = red]], t=0..200,
  view = [0..70, 0..N]);
```

2.5

Although R_0 is only defined at the time of invasion, σ and R are defined at all times. For most models, the contact number σ remains constant as the infection spreads, so it is always equal to the basic reproduction number R_0. In these models σ and R_0 can be used interchangeably (Hethcote, 2000, p. 604)

2.6

```
> restart;with(LinearAlgebra):
  with(VectorCalculus):
  with(plots):
> eqn1:= -0.003*S*In + 50 - 0.05*S;
> eqn2:= 0.003*S*In - In - 0.05*In;
> jc := Jacobian([eqn1, eqn2], [S, In]);
> sp:=solve({eqn1=0, eqn2=0},{S,In});
> p1:=eval(jc,[S=subs(sp[2],S), In=subs(sp[2],In)]);
> e1 := Eigenvectors(p1);
```

$$eqn1 := -0.003\,S\,In + 50 - 0.05\,S$$

$$eqn2 := 0.003\,S\,In - 1.05\,In$$

$$jc := \begin{bmatrix} -0.003\,In - 0.05 & -0.003\,S \\ 0.003\,In & 0.003\,S - 1.05 \end{bmatrix}$$

$$sp := \{S = 1000.,\ In = 0.\},\ \{S = 350.,\ In = 30.95238095\}$$

$$p1 := \begin{bmatrix} -0.1428571428 & -1.050 \\ 0.09285714285 & 0. \end{bmatrix}$$

$$e1 := \begin{bmatrix} -0.07142857140000000016 + 0.303970326150851622\,I \\ -0.07142857140000000016 - 0.303970326150851622\,I \end{bmatrix},$$

$$\begin{bmatrix} 0.958514475637036288 + 0.\,I & 0.958514475637036288 + 0.\,I \\ -0.0652050663437843958 - 0.277485674075907128\,I & -0.0652050663437843958 + 0.277485674075907128\,I \end{bmatrix}$$

2.7

```
> restart:with(DEtools):with(plots):
Warning, the name changecoords has been redefined
> de1:= D(S)(t)=-beta*S(t)*In(t)+mu*N-mu*S(t);
> de2:= D(In)(t)= (beta*S(t)-r-mu)*In(t);
> mu:= 5*10^(-2);
> N:=1000;
> beta:=3*10^(-3); r:=1;
> R0 = beta*N/(mu + r);
>
> PP1:=phaseportrait([de1,de2],[S(t),In(t)],t=0..70,
  [[S(0)=990,In(0)=10]],S=-1..1000,In=-1..1000,linecolor=black,arrows=medium
  ,stepsize=.01):
> display({PP1});
```

$$de1 := D(S)(t) = -\beta\, S(t)\, In(t) + \mu\, N - \mu\, S(t)$$

$$de2 := D(In)(t) = (\beta\, S(t) - r - \mu)\, In(t)$$

$$\mu := \frac{1}{20}$$

$$N := 1000$$

$$\beta := \frac{3}{1000}$$

$$r := 1$$

$$R0 = \frac{20}{7}$$

2.8

```
> restart:with(plots):with(plottools):with(DEtools):
Warning, the name changecoords has been redefined
Warning, the assigned name arrow now has a global binding
Warning, the previous binding of the name translate has been removed and it
now has an assigned value
```

```
> eqn1:= diff(S(t),t) = -beta*In(t)*S(t)+mu*N-mu*S(t), S(0)=ic1;
```

$$eqn1 := \frac{d}{dt} S(t) = -\beta\, In(t)\, S(t) + \mu\, N - \mu\, S(t),\ S(0) = ic1$$

```
> eqn2:= diff(In(t),t) = beta*S(t)*In(t)- mu*In(t)-r*In(t),In(0)=ic2;
```

$$eqn2 := \frac{d}{dt} In(t) = \beta\, In(t)\, S(t) - \mu\, In(t) - r\, In(t),\ In(0) = ic2$$

```
> beta:=3*10^(-3);
```

$$\beta := \frac{3}{1000}$$

```
> mu:=5*10^(-2);
```

$$\mu := \frac{1}{20}$$

```
> r:=1;
```

$$r := 1$$

```
> ic1:=990; ic2:=10;
```

$$ic1 := 990$$

$$ic2 := 10$$

```
> N:=ic1+ic2;
```

$$N := 1000$$

```
> R0 = beta*N/(mu + r);
```

$$R0 = \frac{20}{7}$$

```
> dsol:=dsolve([eqn1, eqn2], numeric);
```

dsol := proc(x_rkf45) ... end proc;

```
> dsol:=dsolve([eqn1, eqn2], numeric);
```

dsol := proc(x_rkf45) ... end proc;

```
> odeplot(dsol,[[t, S(t), color = blue],[t,In(t), color = red]], t=0..70,
  view = [0..70, 0..N]);
```

2.8

```
> restart;with(LinearAlgebra):
  with(VectorCalculus):
  with(plots):
> eqn1:= -0.05*S*In + 14.29 - 0.01429*S;
> eqn2:= 0.05*S*In - 10*In - 0.01429*In;
>
> jc := Jacobian([eqn1, eqn2], [S, In]);
> sp:=solve({eqn1=0, eqn2=0},{S,In});
> p1:=eval(jc,[S=subs(sp[2],S), In=subs(sp[2],In)]);
> e1 := Eigenvectors(p1);
```

$$eqn1 := -0.05\,S\,In + 14.29 - 0.01429\,S$$

$$eqn2 := 0.05\,S\,In - 10.01429\,In$$

$$jc := \begin{bmatrix} -0.05\,In - 0.01429 & -0.05\,S \\ 0.05\,In & 0.05\,S - 10.01429 \end{bmatrix}$$

$$sp := \{In = 0., S = 1000.\}, \{S = 200.2858000, In = 1.141160873\}$$

$$p1 := \begin{bmatrix} -0.07134804365 & -10.01429000 \\ 0.05705804365 & 0. \end{bmatrix}$$

$$e1 := \begin{bmatrix} -0.0356740218249999922 + 0.755065003897404718\,I \\ -0.0356740218249999922 - 0.755065003897404718\,I \end{bmatrix},$$

$$\begin{bmatrix} 0.997163285041178460 + 0.\,I & 0.997163285041178460 + 0.\,I \\ -0.00355220637645281817 - 0.0751848707902373903\,I & -0.00355220637645281817 + 0.0751848707902373903\,I \end{bmatrix}$$

2.9

```
> restart:with(DEtools):with(plots):
Warning, the name changecoords has been redefined
> de1:= D(S)(t)=-beta*S(t)*In(t)+mu*N-mu*S(t);
```

$$de1 := D(S)(t) = -\beta\, S(t)\, In(t) + \mu\, N - \mu\, S(t)$$

```
> de2:= D(In)(t)= (beta*S(t)-r-mu)*In(t);
```

$$de2 := D(In)(t) = (\beta\, S(t) - r - \mu)\, In(t)$$

```
> mu:= 1.429*10^(-2);
```

$$\mu := 0.01429000000$$

```
> N:=1000;
```

$$N := 1000$$

```
> beta:=5*10^(-2); r:=10;
```

$$\beta := \frac{1}{20}$$

$$r := 10$$

```
> R0 = beta*N/(mu + r);
```

$$R0 = 4.992865196$$

```
> PP1:=phaseportrait([de1,de2],[S(t),In(t)],t=0..70,
  [[S(0)=998,In(0)=2]],S=-1..1000,In=-20..600,linecolor=black,arrows=medium,
  stepsize=.01):

> display({PP1});
```

2.10

```
> restart:with(plots):with(plottools):with(DEtools):
Warning, the name changecoords has been redefined
Warning, the assigned name arrow now has a global binding
Warning, the previous binding of the name translate has been removed
and it now has an assigned value
> eqn1:= diff(S(t),t) = -beta*In(t)*S(t)+mu*N-mu*S(t), S(0)=ic1;
```

$$eqn1 := \frac{d}{dt} S(t) = -\beta\, In(t)\, S(t) + \mu\, N - \mu\, S(t),\ S(0) = ic1$$

```
> eqn2:= diff(In(t),t) = beta*S(t)*In(t)- mu*In(t)-r*In(t),In(0)=ic2;
```

$$eqn2 := \frac{d}{dt} In(t) = \beta\, In(t)\, S(t) - \mu\, In(t) - r\, In(t),\ In(0) = ic2$$

```
> beta:=5*10^(-2);
```

$$\beta := \frac{1}{20}$$

```
> mu:= 1.429*10^(-2);
```

$$\mu := 0.01429000000$$

```
> r:=10;
```

$$r := 10$$

```
> ic1:=998; ic2:=2;
```

$$ic1 := 998$$

$$ic2 := 2$$

```
> N:=ic1+ic2;
```

$$N := 1000$$

```
> R0 = beta*N/(mu + r);
```

$$R0 = 4.992865196$$

```
> dsol:=dsolve([eqn1, eqn2], numeric, method = rkf45, range = 0..200);
```

$$dsol := \mathbf{proc}(x\_rkf45)\ \ldots\ \mathbf{end\ proc}$$

```
> odeplot(dsol,[[t, S(t), color = blue],[t,In(t), color = red]],
  t=0..200, view = [0..200, 0..350], refine=10);
```

3.1

```
> restart;with(LinearAlgebra):
  with(VectorCalculus):
  with(plots):
> N:=S+In+R;
> b:=3/365;
> d:=1/(8*365);
> K:=0.9;
> beta:=.01;
> v:= 0;
> d1:=.01;
> r:=.09;
> alpha:=1/90;
> delta:=1;
> eqn1:= b*S*(1-N/K)-d*S-beta*(In+F)*S - v;
> eqn2:= -(d+d1+r)*In+beta*(In+F)*S;
> eqn3:=r*In+v-d*R;
> eqn4:=delta*In-alpha*F;
> Digits:=4;
> jc := Jacobian([eqn1, eqn2, eqn3, eqn4], [S, In, R, F]);
> sp:=solve({eqn1=0, eqn2=0, eqn3=0, eqn4=0},{S,In, R, F});
> subs(sp[3],S);
> p3:=eval(jc,[S=subs(sp[3],S),R=subs(sp[3],R),F=subs(sp[3],F),
  In=subs(sp[3],In)]);
> e3 := Eigenvectors(p3);
```

3.2

```
> restart:with(plots):with(plottools):with(DEtools):
Warning, the name changecoords has been redefined
Warning, the assigned name arrow now has a global binding
Warning, the previous binding of the name translate has been removed and it now has an assigned value
> N:=S(t)+In(t)+R(t);
```

$$N := S(t) + In(t) + R(t)$$

```
> eqn1:= diff(S(t),t)=b*S(t)*(1-N/K)-d*S(t)-beta*(In(t)+F(t))*S(t) - v, S(0)=ic1;
```

$$eqn1 := \frac{d}{dt} S(t) = b\, S(t) \left(1 - \frac{S(t) + In(t) + R(t)}{K} \right) - d\, S(t) - \beta\, (In(t) + F(t))\, S(t) - v,\ S(0) = ic1$$

```
> eqn2:= diff(In(t),t)=-(d+d1+r)*In(t)+beta*(In(t)+F(t))*S(t), In(0)=ic2;
```

$$eqn2 := \frac{d}{dt} In(t) = -(d + d1 + r)\, In(t) + \beta\, (In(t) + F(t))\, S(t),\ In(0) = ic2$$

```
> eqn3:=diff(R(t),t)=r*In(t)+v-d*R(t), R(0)=ic3;
```

$$eqn3 := \frac{d}{dt} R(t) = r\, In(t) + v - d\, R(t),\ R(0) = ic3$$

```
> eqn4:=diff(F(t),t)=delta*In(t)-alpha*F(t), F(0)=ic4;
```

$$eqn4 := \frac{d}{dt} F(t) = \delta\, In(t) - \alpha\, F(t),\ F(0) = ic4$$

```
> b:=3/(365);
```

$$b := \frac{3}{365}$$

```
> d:=1/(8*365);
```

$$d := \frac{1}{2920}$$

```
> K:=0.9;
```

$$K := 0.9$$

```
> beta:=0.01;
```

$$\beta := 0.01$$

```
> v := 0;
```

$$v := 0$$

```
> d1:=0.01;
```

$$d1 := 0.01$$

```
> r:=0.09;
```

$$r := 0.09$$

```
> alpha:=1/(90);
```

$$\alpha := \frac{1}{90}$$

```
> delta:=1;
```

$$\delta := 1$$

```
> ic1:=0.22; ic2:=0.01; ic3:=0; ic4:= 0;
```

$$ic1 := 0.22$$
$$ic2 := 0.01$$
$$ic3 := 0$$
$$ic4 := 0$$

```
> dsol:=dsolve([eqn1, eqn2, eqn3,eqn4], numeric, method = rkf45, range = 0..10000);
```

$$dsol := \text{proc}(x\_rkf45) \ \ldots \ \text{end proc};$$

```
> odeplot(dsol, [[t, S(t), color = blue], [t, In(t), color = red], [t, R(t), color =
  black],[t,F(t),color=green]], t=0..5000, view = [0..5000, 0...2], refine=10);
```

3.3

```
> restart;with(LinearAlgebra):
  with(VectorCalculus):
  with(plots):
> N:=S+In+R;
> b:=3/365;
> d:=1/(8*365);
> K:=1.5;
> beta:=.01;
> v:= 0;
> d1:=.01;
> r:=.09;
> alpha:=1/90;
> delta:=1;
> eqn1:= b*S*(1-N/K)-d*S-beta*(In+F)*S - v;
> eqn2:= -(d+d1+r)*In+beta*(In+F)*S;
> eqn3:=r*In+v-d*R;
> eqn4:=delta*In-alpha*F;
> jc := Jacobian([eqn1, eqn2, eqn3, eqn4], [S, In, R, F]);
> sp:=solve({eqn1=0, eqn2=0, eqn3=0, eqn4=0},{S,In, R, F});
> subs(sp[3],S);
> p3:=eval(jc,[S=subs(sp[3],S),R=subs(sp[3],R),F=subs(sp[3],
  F), In=subs(sp[3],In)]);
> e3 := Eigenvectors(p3);
>
```

3.4

```
> restart:with(plots):with(plottools):with(DEtools):
> N:=S(t)+In(t)+R(t);
```

$$N := S(t) + In(t) + R(t)$$

```
> eqn1:= diff(S(t),t)=b*S(t)*(1-N/K)-d*S(t)-beta*(In(t)+F(t))*S(t) - v, S(0)=ic1;
```

$$eqn1 := \frac{d}{dt} S(t) = b\, S(t) \left(1 - \frac{S(t) + In(t) + R(t)}{K} \right) - d\, S(t) - \beta\, (In(t) + F(t))\, S(t) - v,\ S(0) = ic1$$

```
> eqn2:= diff(In(t),t)=-(d+d1+r)*In(t)+beta*(In(t)+F(t))*S(t), In(0)=ic2;
```

$$eqn2 := \frac{d}{dt} In(t) = -(d + d1 + r)\, In(t) + \beta\, (In(t) + F(t))\, S(t),\ In(0) = ic2$$

```
> eqn3:=diff(R(t),t)=r*In(t)+v-d*R(t), R(0)=ic3;
```

$$eqn3 := \frac{d}{dt} R(t) = r\, In(t) + v - d\, R(t),\ R(0) = ic3$$

```
> eqn4:=diff(F(t),t)=delta*In(t)-alpha*F(t), F(0)=ic4;
```

$$eqn4 := \frac{d}{dt} F(t) = \delta\, In(t) - \alpha\, F(t),\ F(0) = ic4$$

```
> b:=3/(365);
```

$$b := \frac{3}{365}$$

```
> d:=1/(8*365);
```

$$d := \frac{1}{2920}$$

```
> K:=1.5;
```

$$K := 1.5$$

```
> beta:=0.01;
```

$$\beta := 0.01$$

```
> v := 0;
```

$$v := 0$$

```
> d1:=0.01;
```

$$d1 := 0.01$$

```
> r:=0.09;
```

$$r := 0.09$$

```
> alpha:=1/(90);
```

$$\alpha := \frac{1}{90}$$

```
> delta:=1;
```

$$\delta := 1$$

```
> ic1:=0.22; ic2:=0.01; ic3:=0; ic4:= 0;
```

$$ic1 := 0.22$$

$$ic2 := 0.01$$

$$ic3 := 0$$

$$ic4 := 0$$

```
> dsol:=dsolve([eqn1, eqn2, eqn3,eqn4], numeric, method = rkf45, range = 0..30000);
```

$$dsol := \text{proc}(x\_lsode)\ \ldots\ \text{end proc};$$

```
> odeplot(dsol, [[t, S(t), color = blue], [t, In(t), color = red], [t, R(t), color =
  black],[t,F(t),color=green]], t=0..30000, view = [0..30000, 0...2], refine=10);
```

3.5

```
> restart;with(LinearAlgebra):
  with(VectorCalculus):
  with(plots):
> N:=S+In+R;
```

$$N := S + In + R$$

```
> b:=3/365;
```

$$b := \frac{3}{365}$$

```
> d:=1/(8*365);
```

$$d := \frac{1}{2920}$$

```
> K:=1.04;
```

$$K := 1.04$$

```
> beta:=.01;
```

$$\beta := 0.01$$

```
> v:= 0;
```

$$v := 0$$

```
> d1:=.01;
```

$$d1 := 0.01$$

```
> r:=.09;
```

$$r := 0.09$$

```
> alpha:=1/90;
```

$$\alpha := \frac{1}{90}$$

```
> delta:=1;
```

$$\delta := 1$$

```
> eqn1:= b*S*(1-N/K)-d*S-beta*(In+F)*S - v;
```

$$eqn1 := \frac{3}{365} S (-0.9615384615 S - 0.9615384615 In - 0.9615384615 R + 1) - \frac{1}{2920} S - (0.01 In + 0.01 F) S$$

```
> eqn2:= -(d+d1+r)*In+beta*(In+F)*S;
```

$$eqn2 := -0.1003424658 In + (0.01 In + 0.01 F) S$$

```
> eqn3:=r*In+v-d*R;
```

$$eqn3 := 0.09 In - \frac{1}{2920} R$$

```
> eqn4:=delta*In-alpha*F;
```

$$eqn4 := In - \frac{1}{90} F$$

```
> jc := Jacobian([eqn1, eqn2, eqn3, eqn4], [S, In, R, F]);
```

$$jc := \left[\left[-0.01580611170\,S - 0.01790305585\,In - 0.007903055848\,R + 0.007876712329 - 0.01\,F,\right.\right.$$

$$-0.01790305585\,S,\ -0.007903055848\,S,\ -0.01\,S\Big],$$

$$\left[0.01\,In + 0.01\,F,\ -0.1003424658 + 0.01\,S,\ 0,\ 0.01\,S\right], \left[0,\ 0.09,\ \frac{-1}{2920},\ 0\right], \left[0,\ 1,\ 0,\ \frac{-1}{90}\right] \Big]$$

```
> sp:=solve({eqn1=0, eqn2=0, eqn3=0, eqn4=0},{S,In, R, F});
```

$$sp := \{R = 0.,\ S = 0.,\ F = 0.,\ In = 0.\},\ \{R = 0.,\ S = 0.9966666667,\ F = 0.,\ In = 0.\},$$

$$\{S = 0.1102664459,\ F = 0.2105211830,\ In = 0.002339124256,\ R = 0.6147218544\}$$

```
> subs(sp[3],S);
```

$$0.1102664459$$

```
> p3:=eval(jc,[S=subs(sp[3],S),R=subs(sp[3],R),F=subs(sp[3],F),
  In=subs(sp[3],In)]);
```

$$p3 := \begin{bmatrix} -0.000871441880 & -0.001974106339 & -0.0008714418801 & -0.001102664459 \\ 0.002128603073 & -0.09923980134 & 0 & 0.001102664459 \\ 0 & 0.09 & \frac{-1}{2920} & 0 \\ 0 & 1 & 0 & \frac{-1}{90} \end{bmatrix}$$

```
> e3 := Eigenvectors(p3);
```

$$e3 := \begin{bmatrix} -0.110522547252304509 + 0.\,I \\ -9.04237071694393024\,10^{-7} + 0.00482284276253261250\,I \\ -9.04237071694393024\,10^{-7} - 0.00482284276253261250\,I \\ -0.00104046435808804408 + 0.\,I \end{bmatrix}, [[0.00883885249260626948 + 0.\,I,$$

$$-0.0103993057410875795 + 0.236925038764249529\,I,$$

$$-0.0103993057410875795 - 0.236925038764249529\,I,\ -0.0325830667325580914 + 0.\,I],\ [$$

$$-0.0985985831514201022 + 0.\,I,\ 0.0105282424297778420 + 0.00457021713279598397\,I,$$

$$0.0105282424297778420 - 0.00457021713279598397\,I,\ 0.00614122662589943088 + 0.\,I],$$

$$[0.0805397160984860499 + 0.\,I,\ 0.0987049219472773548 - 0.189479122762922834\,I,$$

$$0.0987049219472773548 + 0.189479122762922834\,I,\ -0.791850288293182602 + 0.\,I],$$

$$[0.991823345267655032 + 0.\,I,\ 0.947618937175559339 + 0.\,I,\ 0.947618937175559339 + 0.\,I,$$

$$0.609814521004407360 + 0.\,I]]$$

3.6

```
> restart;with(LinearAlgebra):
  with(VectorCalculus):
  with(plots):
> N:=S+In+R;
```

$$N := S + In + R$$

```
> b:=3/365;
```

$$b := \frac{3}{365}$$

```
> d:=1/(8*365);
```

$$d := \frac{1}{2920}$$

```
> K:=1.05;
```

$$K := 1.05$$

```
> beta:=.01;
```

$$\beta := 0.01$$

```
> v:= 0;
```

$$v := 0$$

```
> d1:=.01;
```

$$d1 := 0.01$$

```
> r:=.09;
```

$$r := 0.09$$

```
> alpha:=1/90;
```

$$\alpha := \frac{1}{90}$$

```
> delta:=1;
```

$$\delta := 1$$

```
> eqn1:= b*S*(1-N/K)-d*S-beta*(In+F)*S - v;
```

$$eqn1 := \frac{3}{365} S (-0.9523809524 S - 0.9523809524 In - 0.9523809524 R + 1) - \frac{1}{2920} S - (0.01 In + 0.01 F) S$$

```
> eqn2:= -(d+d1+r)*In+beta*(In+F)*S;
```

$$eqn2 := -0.1003424658 In + (0.01 In + 0.01 F) S$$

```
> eqn3:=r*In+v-d*R;
```

$$eqn3 := 0.09 In - \frac{1}{2920} R$$

```
> eqn4:=delta*In-alpha*F;
```

$$eqn4 := In - \frac{1}{90} F$$

```
> jc := Jacobian([eqn1, eqn2, eqn3, eqn4], [S, In, R, F]);
```

$$jc := \left[\begin{array}{c} [-0.01565557730\ S - 0.01782778865\ In - 0.007827788650\ R + 0.007876712329 - 0.01\ F,\ -0.01782778865\ S, \\ -0.007827788650\ S,\ -0.01\ S],\ [0.01\ In + 0.01\ F,\ -0.1003424658 + 0.01\ S,\ 0,\ 0.01\ S],\left[0,\ 0.09,\ \frac{-1}{2920},\ 0 \right],\left[0,\ 1,\ 0,\ \frac{-1}{90} \right] \end{array} \right]$$

```
> sp:=solve({eqn1=0, eqn2=0, eqn3=0, eqn4=0},{S,In, R, F});
```

$$sp := \{S = 0.,\ F = 0.,\ R = 0.,\ In = 0.\},\ \{F = 0.,\ R = 0.,\ In = 0.,\ S = 1.006250000\},$$
$$\{S = 0.1102664459,\ F = 0.2121773173,\ In = 0.002357525747,\ R = 0.6195577664\}$$

```
> subs(sp[3],S);
```

$$0.1102664459$$

```
> p3:=eval(jc,[S=subs(sp[3],S),R=subs(sp[3],R),F=subs(sp[3],F), In=subs(sp[3],In)]);
```

$$p3 := \begin{bmatrix} -0.000863142434 & -0.001965806893 & -0.0008631424337 & -0.001102664459 \\ 0.002145348430 & -0.09923980134 & 0 & 0.001102664459 \\ 0 & 0.09 & \frac{-1}{2920} & 0 \\ 0 & 1 & 0 & \frac{-1}{90} \end{bmatrix}$$

```
> e3 := Eigenvectors(p3);
```

$$e3 := \begin{bmatrix} -0.110523906402834868 + 0.\ I \\ 8.38392515013593975\ 10^{-7} + 0.00484004914992496906\ I \\ 8.38392515013593975\ 10^{-7} - 0.00484004914992496906\ I \\ -0.00103429102073087959 + 0.\ I \end{bmatrix}, [[0.00883941612608903082 + 0.\ I,$$
$$-0.0103104360675405678 + 0.236008898592388439\ I,\ -0.0103104360675405678 - 0.236008898592388439\ I,$$
$$-0.0319477194427438480 + 0.\ I],\ [-0.0985999167362004298 + 0.\ I,$$
$$0.0105337783227464083 + 0.00458821422828377662\ I,\ 0.0105337783227464083 - 0.00458821422828377662\ I,$$
$$0.00610846724845998460 + 0.\ I],\ [0.0805398119134644596 + 0.\ I,$$
$$0.0987138573132724934 - 0.188872291221261090\ I,\ 0.0987138573132724934 + 0.188872291221261090\ I,$$
$$-0.794654486243344782 + 0.\ I],\ [0.991823199889530604 + 0.\ I,\ 0.947968519773158546 + 0.\ I,$$
$$0.947968519773158546 + 0.\ I,\ 0.606189968032800785 + 0.\ I]]$$