ISTANBUL TECHNICAL UNIVERSITY ★ GRADUATE SCHOOL OF SCIENCE ENGINEERING AND TECHNOLOGY

STABILITY ANALYSIS OF A MATHEMATICAL MODEL OF CRIMEAN CONGO HAEMORRHAGIC FEVER DISEASE

M.Sc. THESIS

Miray ALIN

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İSTANBUL TEKNİK ÜNİVERSİTESİ ★ FEN BİLİMLERİ ENSTİTÜSÜ

KIRIM-KONGO KANAMALI ATEŞİNİN MATEMATİKSEL MODELİNİN KARARLILIK ANALİZİ

YÜKSEK LİSANS TEZİ

Miray ALIN
(509161293)

Matematik Mühendisliği Anabilim Dalı

Matematik Mühendisliği Programı

Tez Danışmanı: Doç. Dr. Saadet Seher ÖZER

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Miray ALIN, a M.Sc. student of ITU Graduate School of Science Engineering and Technology 509161293 successfully defended the thesis entitled "STABILITY ANALYSIS OF A MATHEMATICAL MODEL OF CRIMEAN CONGO HAEMORRHAGIC FEVER DISEASE", which he/she prepared after fulfilling the requirements specified in the associated legislations, before the jury whose signatures are below.

Thesis Advisor :	**Doç. Dr. Saadet Seher ÖZER** Istanbul Technical University	.....
Jury Members :	**Doç. Dr. Ali Ercengiz** Istanbul Technical University	.....
	Doç.Dr. Mustafa Taylan Şengül Marmara University	.....
		.....

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To my beloved father,

FOREWORD

I would not have been able to write this thesis without the support of the people around me. I would like to thank my family, boyfriend and colleagues for their mental support.

This thesis would not have been possible without support of my advisor Assoc. Prof. Dr. Saadet Seher Özer. I deeply thank for being supportive and helpful all the time.

July 2020 Miray ALIN

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STABILITY ANALYSIS OF A MATHEMATICAL MODEL OF CRIMEAN CONGO HAEMORRHAGIC FEVER DISEASE

SUMMARY

Today, ticks are harmful parasitic creatures feared by humans. Ticks do not always carry dangerous diseases. However, we should not ignore the pathogens and viruses that may be carried because these creatures can carry various viruses and seriously threaten human health. If it is not diagnosed early, it can result in fatal consequences.

Ticks can get viruses from their hosts at various stages of their lives. Ticks can transmit these viruses to humans in the adult tick stage. Here we can say that the animals that ticks use as hosts are only vectors. Cattle, bovine or chickens do not show symptoms of diseases which are caused by ticks.

In this thesis, the spread of Crimean-Congo haemorrhagic fever disease is investigated by considering the problem as an epidemic model. Before stating the problem, in first chapter, some information about dynamic systems is given. The definition of systems of differential equations and their stability analysis are mentioned. Besides, the autonomous systems of equations are briefly explained. And how their stability can be analysed is mentioned. Then, to guide our own problem, information about the well-known SI, SIR, SIS epidemic models and Prey-Predator model and their stability is given in the second chapter. And finally in the third chapter the original problem of the thesis is examined. The system of equation of these models is non-linear. After writing system of equation we found the equilibrium points first. Then, we do linearisation by substituting the equilibrium point in to the Jacobian matrix. We investigated sign of the eigenvalues of these Jacobian matrices which are evaluated by equilibrium points of epidemic models. If all eigenvalues are negative the equilibrium point is stable. If at least one eigenvalue is positive, then the equilibrium point is called unstable. It is not always possible to determine the sign of eigenvalues. In such a case, we could talk about *basic reproduction number*. Basic reproduction number is represented by R_0. If $R_0 < 1$, all eigenvalues are negative and the equilibrium point is a stable equilibrium point. The disease disappear over time. Otherwise, if $R_0 > 1$, at least one of the eigenvalues is positive. Also, the endemic equilibrium point exist when $R_0 > 1$. In addition to, when $R_0 > 1$ disease free equilibrium point is unstable and endemic equilibrium point is stable. And the disease becomes endemic.

The problem is expressed as the combination of the variation of population dynamics of human, tick and birds(chicken). In all dynamics of human and tick we considered the in and outs to the compartments, outs as both in the meaning of transfers between compartments and removals such as death. The inputs to the system are either taken constants or logistic growth effects.

In this thesis, we investigate the problem in three different ways.

- The model which takes logistic growth both in tick and chicken populations,

- The model which takes logistic growth only in chicken population,
- The model which takes logistic growth only in tick population.

We use a system of five ODEs to represent the interaction between chicken population, susceptible and infected populations of humans and ticks. It can be said that there is *SI* model between infected tick and susceptible tick, *SIS* model between infected human and susceptible human, and Prey-Predator model between tick and chicken. We have determined the equilibrium points for each model and investigate the stability of the equilibrium points. During the studies the reproduction numbers were found and the stability is investigated with respect to the reproduction numbers. The bifurcation analysis has also been done for tick logistic - chicken logistic model and tick logistic - chicken constant model.

According to the results of the first and second models, it was observed that there was a decrease in the number of ticks when the chicken population in the environment was increased. In addition, if the frequency of unleashing of chickens into the environment is increased, then ticks can be more likely to increase among chickens is. Therefore, the number of ticks in the environment may decrease. Due to this decrease, it has mathematically shown that the Crimean Congo Haemorrhagic Fever disease decreases over time.

KIRIM-KONGO KANAMALI ATEŞİNİN MATEMATİKSEL MODELİNİN KARARLILIK ANALİZİ

ÖZET

Günümüzde keneler insanlar için tehlikeli, parazit taşıyan canlılar olmuşlardır. Bu nedenle insanlar bu canlılardan uzak durmak için bir çok önlem almaktadırlar. Keneler her zaman tehlikeli hastalık yayacak virüs taşımazlar. Ancak, insan sağlığını tehdit edebilecek, tehlikeli virüs ve patojenleri taşıyabileceklerini göz ardı etmemeliyiz. Bu tip insan sağlığını tehdit eden virüs ve patojenler, erken teşhis edilmediği takdirde, sonuçları ölümcül olabilir.

Keneler hayatları boyunca 3 evreden geçerler. Bunlar, larva evresi, nymph evresi ve yetişkin evre olmak üzere üçe ayrılır. Larvalar ve nympler genelde küçük baş hayvan, taşvan veya kuş gibi canlılardan beslenirler. Yetişkin evreye geldiklerinde ise daha çok büyük baş hayvanları veya insanları tercih ederler. Bu nedenle keneler insanlara Kırım-Kongo Kanamalı Ateşi hastalığını yetişkin evresinde bulaştırırlar. Keneler yaşamlarının çeşitli evrelerinde konak olarak kullandıkları hayvanlardan virüs alabilirler. O halde, kenelerin konak olarak kullandıkları büyük baş, küçük baş veya tavukların Kırım-Kongo Kanamalı Ateşi hastalığının semptomlarını göstermediğini ve hastalıktan etkilenmedi yalnızca taşıyıcı oldukları söylenebilir.

Köylerde, kırsal kesimlerde yaşayan veya çalışan insanların kene ısırığına maruz kalma ihtimalleri, şehirlerde yaşayan insanlara göre daha fazladır. Bu nedenle köy yerleri, kırsal kesimler gibi yerlerde yaşayan insanların daha fazla önlem almaları gerekmektedir. Kenelerden korunmanın çeşitli yolları vardır. Bunlardan bazılarını şöyle sıralayabiliriz,

- Özellikle tarlada çalışan insanlar veya doğa yürüyüşüne çıkanlar uzun kollu t-shirt ve pantolon tercih etmelidirler.
- Köy yerlerinde yaşayan, özellikle tarlada çalışan veya kenelerin çok görüldüğü yerlerde ikamet eden insanların pantolon paçarını çoraplarının içine sokmaları kenelerden korunmalarına yardımcı olacaktır.
- Doğa yürüyüşüne çıkıldığında, patikaların ortasından yürümeye özen gösterilmelidir. Çünkü keneler genellikle toprağa yakın yaprak altlarında bulunurlar.
- Kene sokmasına maruz kalan insanların, keneleri kendi uğraşları ile çıkarmaya çalışmamalılar. Uzman olmayan bir kişi keneyi çıkarmaya çalışırken kenenin ısırdığı yerden içeriye kusmasına ve virüs taşıyorsa insana bulaşmasına sebebiyet verebilir. Bu nedenle kene sokmasına maruz kalan kişi hemen bir hastaneye gitmeli ve doktor tarafından çıkarılmalıdır.
- Hayvanlar üzerinde olan keneler çıkarılırken çıplak elle çıkarılmamalıdır. Çıplak elle yapılan temasta hayvanın kanı insana sürüldüğünde, kene herhangi bir virüs

veya patojen taşıyorsa bu insana geçebilir. Bu nedenle ken ısırığına maruz kalan bir hayvanın kan veya vücut sıvısı ile temas edilmemesine özen gösterilmelidir.

- Kenelerin yoğun görüldüğü zamanlar özellikle Nisan-Ekim dönemlerinde ilaçlama yapılması kenelerin üremesine engel olacak önlemlerden bir tanesidir.
- Kırsal kesimlerde yaşıyan insanlar, kene sokmasına karşı, hayvanlarını belirli periyodlarda parazit aşılarını yapmalıdırlar.

Bu tezde, kenelerin yoğun olarak bulunduğu yerlerdeki tavukların, burada bulunan keneler üzerindeki etkisi epidemik bir matematiksel bir model üzerinde araştırılmıştır. Ayrıca, bu etkinin dolaylı olarak Kırım-Kongo Kanamalı Ateşi hastalığının insan populayonuna etkisi de incelenmiştir.

Problemimize başlamadan önce, problemin çözümünü ve anlaşılmasını kolaylaştırmak adına çeşitli bilgiler verdik. İlk olarak, dinamik sistemler hakkında bir takım tanımlamalar yaptık. Diferansiyel denklem sisteminin, sabit homojen denklem sisteminin ve otonom denklem sisteminin tanımını yaptık. Ardından bu denklem sistemlerinin stabilite analizi, neden stabilite analizine ihtiyaç duyduğumuz hakkında bilgiler verdik. Stabilite analizine başlayabilmek için ilk olarak denge noktalarının bulunması gerektiğini söyeledik. Ardından denge noktalarını, sisteme ait yazılan Jacobian matriste yerine koyarak stabilite analizini yapabileceğimizi söyledik.

Denge noktaları, Jacobian matriste yazıldıktan sonra elde edilen matrisin özdeğerlerinin işaretlerine göre bulunan denge noktasının stable mı yoksa unstable mı olduğunun kararının nasıl verileceğini açıkladık. Denge noktasının Jacobian matriste yerine yazıldıktan sonra elde edilen matrisin tüm özdeğerleri negatif reel kısma sahip ise bu denge noktasının stable, en az birinin reel kısmı pozitif ise bu denge noktasının unstable olduğunu söyledik.

Bu tanımlamaları yaptıktan sonra, üçüncü bölümde çok iyi bilinen birkaç matematiksel modelin analizlerini kendi problemlerimize yol göstermesi adına tekrar yaptık. Bu modeller arasında *SI*, *SIR*, *SIS* epidemik modelleri ve Prey-Predator (Av-Avcı) modelini inceledik. Bu modellerin model diyagramlarını çizdik. Ardından bu modellerin lineer olmayan denklem sistemlerini yazdık. Denklem sistemlerini yazdıktan sonra stabilite analizlerini yapabilmek için ilk olarak bu modellerin denge noktalarını bulduk ve sonrasında denklem sistemine ait Jacobian matriste bu noktalarını yerine koyduk. Elde edilen matrisin özdeğerlerinin işaret incelemesini yaparak denge noktalarının stable mı yoksa unstable mı olduğuna karar verdik. Fakat her zaman denge noktasının Jacobian matriste yerine koyularak elde edilen matrisin özdeğerlerinin kesin olarak negatif ya da kesin olarak pozitif olduğunu söylemek mümkün olmuyordu. Bazı özdeğerler belli koşullar sağlandığında negatif ya da pozitif oluyordu. İşte bu durumda *basic reproduction number*'dan söz edebiliyorduk. R_0 ile temsil edilen bu terim, işaret analizini yapamadığımız özdeğerden elde edilmektedir. Eğer $R_0 < 1$ ise, bütün özdeğerler negatif olmaktadır. Bu durumda denge noktasının stable olduğu söylenmektedir. Ayrıca $R_0 < 1$ durumunda hastalık durumu zamanla ortadan kalkmaktadır. Ayrıca, model incelemelerimizde $R_0 > 1$ olduğu durumlarda endemik denge noktası adını verdiğimiz denge noktası var olacak aynı zamanda stable olacaktır. Bu da hastalığın populasyonda varlığını sürdürüp endemik bir hal alacağını işaret etmektedir.

Biz bu tezde, problemlerimizi üç farklı şekilde ele aldık.

- Hem kene hem de tavuk populasyonlarının lojistik büyüme olarak alındığı model.
- Yalnızca tavuk populasyonunun lojistik büyüme olarak alındığı model.
- Yalnızca kene populasyonunun logistik büyüme olarak alındığı model.

Problemlerimizde beş tane adi diferansiyel denklemden meydana gelen bir denklem sistemi oluşturduk. Bu denklemler yazılırken duyarlı insan ve kene populasyonları, infekte insan ve kene populasyonları ve tavuk populasyonu arasındaki ilişkiler göz önüne alınmıştır. Burada infekte kene ve duyarlı kene arasında *SI* model olduğu söylenebilir. Çünkü bir kene infekte olduktan sonra iyileşme şansı bulunmadığından tekrar duyarlı olamamaktadır. İnfekte insan ve duyarlı insan arasında ise *SIS* modeli bulunmaktadır. Çünkü Kırım-Kongo Kanamalı Ateşi hastalığına yakalanan bir insan iyileştikten sonra virüsü taşıyan bir kene tarafından ısırılırsa tekrar hastalığa yakalanma riski bulunmaktadır. Kene populasyonu ve tavuk populasyonu arasında ise Prey-Predator modeli ilişkisi vardır. Burada keneler avı, tavuklar ise avcıyı temsil etmektedir.

İncelemiş olduğumuz üç modelden ilk ve üçüncü modelin sonuçlarına baktığımızda, tavuk populasyonuna eklenen tavuk miktarının sayısı artırıldığında ortamdaki kene sayısının daha hızlı düştüğü sonucuna varılmıştır. Buna ek olarak, eğer tavukların kümeslerinden salınma sıklığı da artırıldığında, daha fazla kene bulup yiyeceklerinden yine kene sayısında bir düşüş olacağı matematiksel olarak görülmüştür. Bu düşüşlere bağlı olarak Kırım-Kongo Kanamalı Ateşi hastalığının ortamda bir süre kalıcığını sürdükten sonra zaman içerisinde yok olduğu verilerimizce gösterilmiştir.

1. INTRODUCTION TO DYNAMICAL SYSTEMS

1.1 What is a System of Differential Equation?

Equations that are associated with one or more variables according to their derivatives are called differential equations. Differential equations are both at the center of many theories of physic and are necessary for the mathematical explanation of many things in nature. For example, they are used to describe many problems in classical mechanics such as Newton's and Lagrange's classical mechanical equations, Maxwell's classical electromagnetism equation, Schrödinger's quantum mechanics equation, and Einstein's general gravitation theory.

1.1.1 Linear Homogeneous System of Differential Equation with Constant Coefficient

A special form system of differential equation is written as [1, 2],

$$\dot{x}(t) = Ax(t) + f(t)$$

where $x \in R^n$ and f is a function depends on the independent variable t. If $f(t) = 0$, system of equations become homogeneous

$$\dot{x} = Ax. \tag{1.1}$$

where A is a constant coefficient *nxn* matrix and

$$\dot{x} = \frac{dx}{dt} = \begin{pmatrix} \frac{dx_1}{dt} \\ \frac{dx_2}{dt} \\ \vdots \\ \frac{dx_n}{dt} \end{pmatrix}.$$

equation (1.1) is called a system of linear homogeneous differential equation with constant coefficient. It is shown that the general solution of this linear system (1.1) is given by

$$x(t) = e^{At}c$$

where e^{At} is an *nxn* matrix and constant valued vector $c = x(0)$ which is $x(t)$ at time $t = 0$. Also, it can be seen that (1.1) has a unique solution each point x_0 in the phase space R^n.

Definition 1.1.1. [3] If $det(A) \neq 0$ then $Ax = 0$ if and only if $x = 0$. The origin is called an equilibrium point of the linear system (1.1).

1.1.2 Stability Analysis of Equilibrium Point in System of Linear Equations

In this section, we are going to look at the stability analysis of equilibrium point.

Suppose, A is a constant coefficient *nxn* matrix and $\lambda_i,\ i = 1,2,...$ are eigenvalues of this matrix. These eigenvalues give us information about the system of differential equation's behaviour around the equilibrium point. If all eigenvalues of the linear system (1.1) have negative real parts for $t \to \infty$, the flow gradually approaches the origin that origin is the equilibrium point of this linear system. On the other hand if all eigenvalues of the linear system (1.1) has a positive real part for $t \to \infty$ the flow moves away from the origin. Here we shall use these information to give the following definitions.

Definition 1.1.2. [3] Suppose that some of the eigenvalues of A have negative real part, some have positive real part and these eigenvalues are distinct. Also let $\{w_1,...,w_n\}$ are eigenvectors corresponding to these eigenvalues. Let us denote these eigenvalues as $\lambda_j = a_j + ib_j$ and eigenvectors as $w_j = u_j + iv_j,\ j = 1,2,....$ *Stable*, *unstable subspace* of the linear system (1.1) represent by E^s and E^u respectively. They are linear subspaces that are shown below;

$$E^s = Span\{u_j, v_j | a_j < 0\},$$
$$E^u = Span\{u_j, v_j | a_j > 0\}.$$

- If all the solution curves of the system (1.1) are decreasing functions then it means all eigenvalues of matrix A have negative real part. All solutions in E^s approach the equilibrium point when $t \to \infty$ then this equilibrium point is called a stable equilibrium point.

- If all eigenvalues of the matrix A has positive real part then all solutions in E^u move away from the equilibrium point when $t \to \infty$. As a result, such an equilibrium point is called an unstable equilibrium point.

In conclusion, if all eigenvalues of the matrix corresponding to the system of the linear equations have negative real parts, the equilibrium point of the system is called stable. Otherwise, if all eigenvalues of the matrix has positive real part, then the equilibrium point is called unstable. Perko (2013) mentioned this topic in detail in [3].

1.2 System of Autonomous Differential Equations

An autonomous system is a system of ordinary differential equations that contains explicitly only the dependent variable.

In the previous section we have considered a special autonomous system that is called the constant coefficient system, it is said that system (1.1) has a unique solution for every x_0 and this solution is $x(t) = e^{At}x_0$. In this section, we examine system of non-linear autonomous differential equations.

Definition 1.2.1. The most generalized form of system of first order ordinary differential equations (ODEs) can be defined as follows,

$$\begin{aligned}
\frac{dx_1}{dt} &= f_1(x_1, ..., x_n) \\
\frac{dx_2}{dt} &= f_2(x_1, ..., x_n) \\
&\vdots \\
\frac{dx_n}{dt} &= f_n(x_1, ..., x_n)
\end{aligned}$$

or, in vector notation,

$$\dot{x} = f(x) \tag{1.2}$$

where $f : E \to R^n$ and E is an open subset of R^n. Under certain conditions, we show that the unique solution at each $x_0 \in E$ point in the maximal interval $(\alpha, \beta) \subset R$ on the function f. In general, it is not possible to solve a non-linear system, but a lot of qualitative information can be obtained about the local behaviour of the solution [4].

1.2.1 Linearisation

The stability analysis of the non-linear system of differential equation is not as easy as system of linear equations. As mentioned in Section (1.2), the local behaviour of the solution of the system (1.2) can be obtained through qualitative information. Linearisation is a method to deal with the system of non-linear equations. Before starting to the linearisation, equilibrium points must be found.

Definition 1.2.2. $x_0 \in R^n$ is called an *equilibrium point* of $\dot{x} = f(x)$ if $f(x_0) = 0$. And also, an equilibrium point x_0 is called a *hyperbolic equilibrium point* of $\dot{x} = f(x)$ if none of the eigenvalues of the matrix $Df(x_0)$ have zero real part.

Detailed information about this definition is given in [3].

Consider the system (1.2)

$$\begin{aligned} \dot{x_1} &= f_1(x_1, x_2, ..., x_n), \\ \dot{x_2} &= f_2(x_1, x_2, ..., x_n), \\ &\vdots \\ \dot{x_n} &= f_n(x_1, x_2, ..., x_n) \end{aligned} \tag{1.3}$$

and assume that $x^* = (x_1^*, x_2^*, ..., x_n^*)$ is equilibrium point of the system (1.3) according to the definition (1.2.2). Namely,

$$\begin{aligned} f_1(x_1^*, x_2^*, ..., x_n^*) &= 0, \\ f_2(x_1^*, x_2^*, ..., x_n^*) &= 0, \\ &\vdots \\ f_n(x_1^*, x_2^*, ..., x_n^*) &= 0. \end{aligned}$$

Let us define

$$\varepsilon = x - x^* \tag{1.4}$$

for linearisation, (1.4) represent the components of a small perturbation nearby equilibrium point. To understand how this perturbation behaves near the equilibrium, we need to derive differential equations for ε. When this derivation is done,

$$\dot{\varepsilon} = \dot{x}$$

is obtained. It can be written

$$\dot{\varepsilon} = f(x^* + \varepsilon) \tag{1.5}$$

by substitution. When Taylor series is expanded to (1.5),

$$\begin{aligned} \dot{\varepsilon} &= f(x^*) + \varepsilon \frac{\partial f}{\partial x} + O(\varepsilon^2), \\ &= \varepsilon \frac{\partial f}{\partial x} + O(\varepsilon^2), \end{aligned} \tag{1.6}$$

equation (1.6) is written because we know $f(x^*) = 0$. Remember that these partial derivatives in (1.6) are evaluated at the equilibrium point x^*. Thus, they are not functions, they are constants. Moreover, $O(\varepsilon^2)$ denotes quadratic term in ε. Since, ε is small, this quadratic term is extremely small. So, this term can be neglected. The disturbance $\varepsilon = (\varepsilon_1, \varepsilon_2, ..., \varepsilon_n)$ evolves according to

$$\begin{pmatrix} \dot{\varepsilon_1} \\ \dot{\varepsilon_2} \\ \vdots \\ \dot{\varepsilon_n} \end{pmatrix} = \begin{pmatrix} \frac{\partial f_1}{\partial x_1} & \frac{\partial f_1}{\partial x_2} & \cdots \frac{\partial f_1}{\partial x_n} \\ \frac{\partial f_2}{\partial x_1} & \frac{\partial f_2}{\partial x_2} & \cdots \frac{\partial f_2}{\partial x_n} \\ \vdots & \vdots & \vdots \\ \frac{\partial f_n}{\partial x_1} & \frac{\partial f_n}{\partial x_2} & \cdots \frac{\partial f_n}{\partial x_n} \end{pmatrix} \begin{pmatrix} \varepsilon_1 \\ \varepsilon_2 \\ \vdots \\ \varepsilon_n \end{pmatrix}. \tag{1.7}$$

The matrix

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

is called the *Jacobian matrix* at the equilibrium point x^* [5]. Also (1.7) is called *linearized system*. Stability analysis can be performed as described in the subsection (1.1.2) using Jacobian matrix. Let us give the formal definition of Jacobian Matrix.

Definition 1.2.3. If $f : R^n \to R^n$ is differentiable at x_0, then the partial derivatives $\frac{\partial f_i}{\partial x_j}$ $i,\ j = 1, ..., n$, all exist at x_0 and for all $x \in R^n$,

$$Df(x_0)x = \sum_{i,j=1}^{n} \frac{\partial f_i}{\partial x_j}(x_0) x_j.$$

Thus, if f is a differentiable function, the derivative Df is called the *nxn* Jacobian matrix

$$Df = \left[\frac{\partial f_i}{\partial x_j} \right].$$

After finding the equilibrium points of the non-linear system of equation, the Jacobian matrix of the system (1.2) should be written. To analyse the stability of the equilibrium points, these points are substituted in the Jacobian matrix.

Consider, x^* is the equilibrium point of system (1.2) and, λ_i, $i = 1,2,...$ are the eigenvalues of the Jacobian matrix which is evaluated at the x^*. If all eigenvalues of this matrix less than zero, the solution of the system start from initial condition converge to this equilibrium point at $t \to \infty$. In that case, the equilibrium point x^* called *locally asymptotically stable*.

Theorem 1.2.1. *[6] A necessary and sufficient condition for an equilibrium to be locally asymptotically stable is that all eigenvalues of the Jacobian have negative real part.*

These theorems are given in detail by Martcheva (2010) in [6].

Perko (2013) mentioned another important theorem in [3] about local qualitative of ordinary differential equations. The Hartman-Grobman Theorem is a very important result in the local qualitative theory of ordinary differential equations. The theorem shows that a hyperbolic equilibrium point has the same qualitative structure as the linear system $\dot{x} = Ax$ with the non-linear $\dot{x} = f(x)$ system where $A = Df(x_0)$ near x_0.

In other words, the theorem states that the behaviour of a dynamic system in an area near the hyperbolic equilibrium is qualitatively the same as behaviour its linearization around this equilibrium point. Hence, simpler linearization of the system can be used to analyze the behaviour around the hyperbolic equilibrium point when dealing with these dynamic systems.

Theorem 1.2.2 (The Hartman-Grobman Theorem [3])**.** *Let E be an open subset of R^n containing the origin, let $f \in C^1(E)$, and let ϕ_t be the flow of the non-linear system* (1.2)*. Suppose that $f(0) = 0$ and that the matrix $A = Df(0)$ has no eigenvalue with zero real part. Then there exists a homomorphism H of an open set U containing the origin onto an open set V containing the origin such that for each $x_0 \in U$, there is an open interval $I_0 \subset R$ containing zero such that for all $x_0 \in U$ and $t \in I_0$*

$$H \circ \phi_t(x_0) = e^{At} H(x_0);$$

i.e., H maps trajectories of (1.2) *near the origin onto trajectories of* (1.1) *near the origin and preserves the parametrization by time.*

Proof of the theorem is given in [3] by (Perko, 2013). We shall use the Hartman-Grobman theorem in our problem in section three.

2. EPIDEMIC MODELS

Epidemiology is the scientific area that examines disease and health patterns on a population basis. The word "epidemiology" consists of Greek terms. "epi" meaning "upon", "demos", "people" and "logos", that is "study". This etymology applies only to the human population of the subject of epidemiology [6]. The father of epidemiology is often considered to be the Greek doctor Hippocrates (460–377 BC), who described the connection between the disease and the environment [7]. The term "epidemiology" appears to have been used for the first time in 1802 by the Spanish doctor de Villalba to describe the work of epidemics in Epidemiologia Espanola [8]. Until the twentieth century epidemiological studies were mostly related to infectious diseases. Today, the leading causes of deaths worldwide are diseases such as stroke and coronary heart disease, positioning diseases that are not transmitted from one person to another as the main concern of epidemiology. Infectious diseases include low respiratory infections and HIV in the world as the dominant causes of death.

According to the Centers for Disease Control and Prevention, an epidemic is an increase in the number of disease cases beyond what is normally expected in a geographic area. Often, the increase in cases occurs quickly. On the other hand, a pandemic is used to describe a disease that has spread to many countries and affects a large number of people. While a pandemic may be described as a kind of epidemic, it cannot be said that an epidemic is a type of pandemic.

Mathematical epidemiology was raised to a new level by the model of the outspread of infectious diseases, published by Kermack and McKendrick in 1927. In their article, “A contribution to the mathematical theory of epidemics” [9], Kermack and McKendrick published for the first time a deterministic *epidemic* model that included susceptible, infected, and removed individuals. This model does not contain natural birth, natural death, or disease-related death and, as a result, models only disease outbreaks. Kermack and McKendrick published Part II and Part III of their “A contribution to the mathematical theory of epidemics” in 1932 and 1933, respectively,

to capture epidemic modeling of diseases that can be established in a population and persist.

Mathematical model is a definition of a system using mathematical tools and language. In addition, the process of developing mathematical models is called mathematical modelling. In general mathematical modelling can be applied to biological or any other system but we will deal with the modelling of infectious diseases and their spread in populations. Mathematical models have been developed to explain a system, to study the effects of its different components, and to make predictions about its behaviour. The modelling process requires that a biological scenario be translated in a math problem. The modelling process begins with a clear definition of methods based on understanding the system. Translation to mathematical equations must be done with a specific aim or a biological question in mind. Then the verbal description of the system is coded with mathematical equations.

In this section, we will analyze *SI*, *SIR*, *SIS* and Predator-Prey models first and finally, we will begin to interpret our mathematical model. To understand the phenomena of a general epidemic model we will give different, simple, very well known models explicitly.

2.1 *SI* Model

SI model is the simplest epidemic model. There are only susceptible individuals S and infective individuals I in the population. When susceptible individual contact with an infective person the disease is transmitted and the susceptible person becomes infectious immediately. Besides, this model, which has already been analyzed before, is not containing naturally occurring or disease-related deaths and also there is no source term for the susceptible population. Detailed research of this model is investigated in [10].

The diagram of the model is given as,

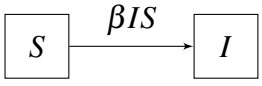

Figure 2.1 : Transfer diagram of SI model.

where β is a non-dimensional parameter representing infectious contact rate. So the system of differential equations according to the figure 2.1 can be written as

$$\frac{dS}{dt} = -\beta IS, \tag{2.1}$$

$$\frac{dI}{dt} = \beta IS. \tag{2.2}$$

As we can see from (2.1) and (2.2), the population is constant. So that,

$$S + I = N.$$

If we reduce the system into one equation, we can analyse the system easily. Let us write $N - I$ instead of S;

$$\begin{aligned} -\frac{dI}{dt} &= -\beta I(N-I) \\ \Rightarrow \frac{dI}{dt} &= \beta IN - \beta I^2 \\ \Rightarrow \frac{dI}{dt} &= \beta IN(1-\frac{I}{N}). \end{aligned}$$

Which is a logistic growth equation given usually as below,

$$\frac{dI}{dt} = rI(1-\frac{I}{K}) \tag{2.3}$$

where r represents growth rate of infective population and K is called carrying capacity. Here we will investigate the stability of this model. First find the equilibrium points;

$$\begin{aligned} \frac{dI}{dt} = 0 \Rightarrow \beta NI(1-\frac{I}{N}) &= 0, \\ I_0 &= 0, \\ I_1 &= N. \end{aligned}$$

A typical application of the logistic equation is a widespread population growth model, in which the growth rate is proportional to both the current population and the number of available resources, originally due to Pierre-François Verhulst in 1838. The Verhulst equation was published after reading Verhulst Thomas Malthus' "An Essay on the Principle of Population" [11]. Verhulst obtained the logistic equation to describe the self-limiting growth of a biological population. The equation was rediscovered in 1911 by A. G. McKendrick for bacterial growth in broth and experimentally tested using a technique for non-linear parameter estimation.

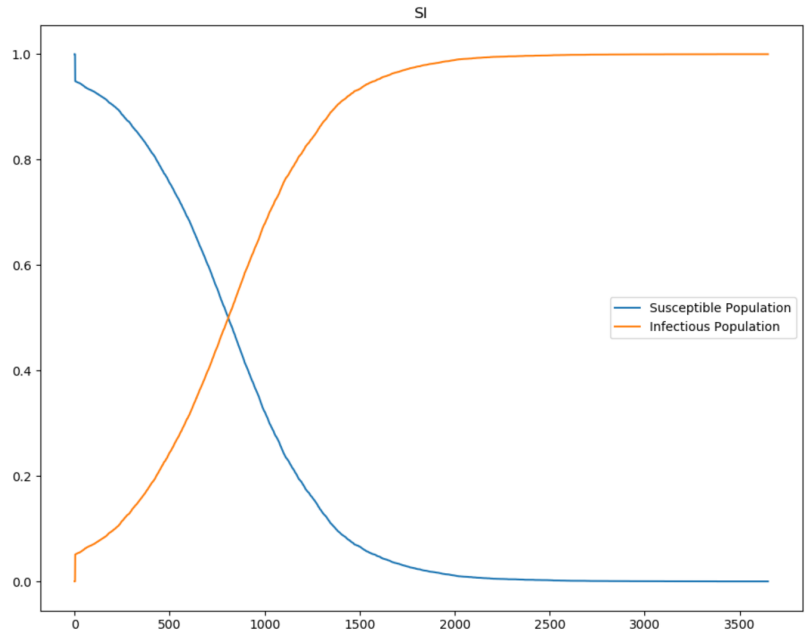

Figure 2.2 : SI outbreak showing logistic grown.

After finding the equilibrium points, the stability analysis of these equilibrium points can be done. The equilibrium point I_0 is an unstable and the equilibrium point I_1 is a stable equilibrium point by the [6].

One can see the solution curves of the SI system given by (2.1)-(2.2) in the Figure 2.2, where the horizontal axis represents time, while the vertical axis represents susceptible and infective populations. This figure shows the change in the number of susceptible and infected people over time. In addition to this, when the parameter β, which is called the contact rate increases the infected human population grows rapidly, and the same ratio decreases in the susceptible population. So, if parameter β in the SI model decreases the spread of the disease decreases accordingly.

So, in the Figure 2.2, the susceptible population in the SI model decreases over time while the infective population grows logistically, therefore the disease spreads and all population is infected over time.

2.2 *SIR* Model

The *SI* model express the spread of disease when infected people do not get treatment. But normally infected people may recover and become healthy. To consider such case we analyse the model with S, I, R where R represents the recovered population or at some studies it is called the removed population.

This outbreak model has very different dynamics. While the susceptible class always decreases independently of the initial condition the recover class always increases independently of the initial condition. In addition, the infective class either monotonically decreases to zero, depending on the initial condition, or it increases non-monotonously to reach the top point first and then monotonically decreases to zero. This topic is mentioned the work of (Martcheva, 2010) [6].

In this section, one of the most commonly reviewed versions, *SIR*, will be discussed. In my model here, I also add the natural and disease-related deaths. On the other hand, the SI model in the subsection 2.1 does not contain any of these. The susceptible individuals S becomes infected and stay infected with no chance of recovery. Everyone in the population are infected after a while in this model.

In this model, individuals leave the susceptible compartment in rate β and enter the infected compartment. Also, they can be leave from the susceptible compartment by dying in a natural way at the rate of d. Infected people can be treated and separated from the infected class at the rate of α, or they can be leave by natural death or disease-related death at the rate of q. In the very well-known SIR model, total population enter in the susceptible compartment at the rate of γ. For convenience, let's write $\gamma N = \Lambda$.

Therefore, the diagram should be like this;

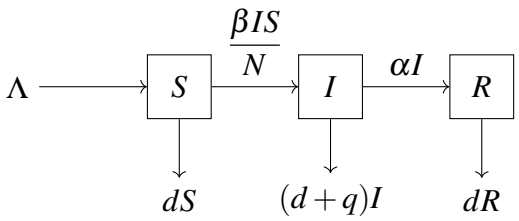

Figure 2.3 : Transfer diagram of SIR model.

Let us write the system of the differential equations [10, 12];

$$\frac{dS}{dt} = \Lambda - \frac{\beta IS}{S+I+R} - dS, \tag{2.4}$$

$$\frac{dI}{dt} = \frac{\beta IS}{S+I+R} - (d+q)I - \alpha I, \tag{2.5}$$

$$\frac{dR}{dt} = \alpha I - dR. \tag{2.6}$$

We assume that the population size $N = S+I+R$ is constant,

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt}.$$

Let us substitute the equations (2.4), (2.5) and (2.6) in the above equation.

$$\frac{dN}{dt} = \Lambda - \frac{\beta IS}{S+I+R} - dS + \frac{\beta IS}{S+I+R} - (d+q)I - \alpha + \alpha I - dR$$

$$\frac{dN}{dt} = \Lambda - dN - qI$$

The reader should be careful not to confuse the parameter d and differential d. It can be said that population is constant, if $\Lambda = dN - qI$. Since the system is non-linear, we need to write the Jacobian matrix to do the stability analysis. Then the stability of the equilibrium points can be investigated by substituting in the Jacobian matrix.

Let us find the equilibrium points of the system;

$$\frac{dS}{dt} = 0 \Rightarrow \Lambda - \frac{\beta IS}{S+I+R} - dS = 0,$$

$$\frac{dI}{dt} = 0 \Rightarrow \frac{\beta IS}{S+I+R} - (d+q)I - \alpha I = 0,$$

$$\frac{dR}{dt} = 0 \Rightarrow \alpha I - dR = 0.$$

Solution of these equations are given in the table (2.1).

Equilibrium Points	**Description**
$E_0 = (\frac{\Lambda}{d}, 0, 0)$	Disease-free equilibrium point. The only non-zero population is susceptible human population at $\frac{\Lambda}{d}$.
$E^* = (S^*, I^*, R^*)$	Endemic Equilibrium point which exist $d+q+\alpha < \beta$ and $q < \beta$. $S^* = -\frac{(d+\alpha)\Lambda}{d(q-\beta)}, I^* = -\frac{d(d+q+\alpha-\beta)}{(d+\alpha)(d+q+\alpha)} S^*, R^* = \frac{\alpha}{d} I^*.$

Table 2.1 : Equilibrium points of SIR model.

Let us write the Jacobian matrix of the system.

$$\mathrm{J} = \begin{pmatrix} -d + \frac{IS\beta}{(S+I+R)^2} - \frac{I\beta}{(S+I+R)} & \frac{IS\beta}{(S+I+R)^2} - \frac{I\beta}{(S+I+R)} & \frac{IS\beta}{(S+I+R)^2} \\ -\frac{IS\beta}{(S+I+R)^2} + \frac{I\beta}{(S+I+R)} & -d-q-\alpha - \frac{IS\beta}{(S+I+R)^2} + \frac{I\beta}{(S+I+R)} & -\frac{IS\beta}{(S+I+R)^2} \\ 0 & \alpha & -d \end{pmatrix}.$$

Now we can investigate stability analysis of the equilibrium points.

1. Equilibrium point $E_0 = (\frac{\Lambda}{d}, 0, 0)$

   Let us do the stability analysis by substituting the disease-free equilibrium point in the Jacobian matrix.

$$J(E_0) = \begin{pmatrix} -d & -\beta & 0 \\ 0 & -d-q-\alpha+\beta & 0 \\ 0 & \alpha & -d \end{pmatrix} \tag{2.7}$$

   Eigenvalues of this matrix are,

$$\lambda_1 = -d,$$
$$\lambda_2 = -d,$$
$$\lambda_3 = -d-q-\alpha+\beta.$$

   If we look at the eigenvalues which are found, it can be said that the eigenvalues λ_1 and λ_2 are negative. But the eigenvalue λ_3 becomes negative when the condition

$$\frac{\beta}{d+q+\alpha} < 1 \tag{2.8}$$

   is satisfied. In this case, all eigenvalues will be negative thus the equilibrium point E_0 is called a stable equilibrium point.

   So we can say that,

$$R_0 = \frac{\beta}{d+q+\alpha}. \tag{2.9}$$

   R_0 is a threshold value. When it is less than 1, the eigenvalue is negative therefore the equilibrium point is stable, so the disease will disappear. But if it is greater than 1, as the eigenvalue will be positive,the equilibrium point will be unstable, therefore the disease will spread. When the flow passes from stable to unstable equilibrium points or vice versa, R_0 will be equal to 1, when it happens we call it bifurcation.

   If we look at the basic reproduction number, it can be said that the basic reproduction number is the ratio of an individual becoming infective to the by sum of the proportion of individuals who enter treatment, the natural death rate and the

disease-related death rate. The interpretation is very straightforward: the rate of infection is greater than the rate of the sum of natural death, disease-related death and transmission rate.

2. Equilibrium point $E^* = (S^*, I^*, R^*)$

   Now, let us do the stability analysis by substituting the endemic equilibrium point in the Jacobian matrix.

$$J(E^*) = \begin{pmatrix} -d + \frac{I^*S^*\beta}{(S^*+I^*+R^*)^2} - \frac{I^*\beta}{(S^*+I^*+R^*)} & \frac{I^*S^*\beta}{(S^*+I^*+R^*)^2} - \frac{I^*\beta}{(S^*+I^*+R^*)} & \frac{I^*S^*\beta}{(S^*+I^*+R^*)^2} \\ -\frac{I^*S^*\beta}{(S^*+I^*+R^*)^2} + \frac{I^*\beta}{(S^*+I^*+R^*)} & -d-q-\alpha - \frac{I^*S^*\beta}{(S^*+I^*+R^*)^2} + \frac{I^*\beta}{(S^*+I^*+R^*)} & -\frac{I^*S^*\beta}{(S^*+I^*+R^*)^2} \\ 0 & \alpha & -d \end{pmatrix} \tag{2.10}$$

   from which we can write the characteristic polynomial

$$P(\lambda) = (\lambda + d)(\lambda^2 + h_1\lambda + h_2) \tag{2.11}$$

   where we have defined,

$$h_1 = \frac{d(\beta - q)}{\alpha + d},$$
$$h_2 = \frac{d(d+q+\alpha)(q-\beta)(d+q+\alpha-\beta)}{\beta(\alpha+d)}.$$

   As seen in Table 2.1, the equilibrium point E^* exist if

$$q < \beta \tag{2.12}$$

   and

$$R_0 = \frac{\beta}{d+q+\alpha} > 1. \tag{2.13}$$

   It is clear that h_1 and h_2 are positive according to (2.12), (2.13) and also it is obvious that $\lambda_1 = -d < 0$, in addition we also know that

$$\lambda_2 + \lambda_3 = -h_1,$$

$$\lambda_2\lambda_3 = h_2.$$

   So, the other two roots of this characteristic equation (2.11) are negative. For more detailed calculations, see [13]. For the existence of this equilibrium point and more detailed information the reader may look at (Martcheva, 2015)'s and (Britton, 2012)'s work [6, 10].

Basically, for the existence of the equilibrium point E^*, the threshold value must be greater than 1 $R_0 > 1$. In this case, all eigenvalues of the Jacobian matrix (2.10) are negative. Therefore, according to the Theorem 1.2.1 the equilibrium point E^* is a *locally asymptotically stable* for $R_0 > 1$.

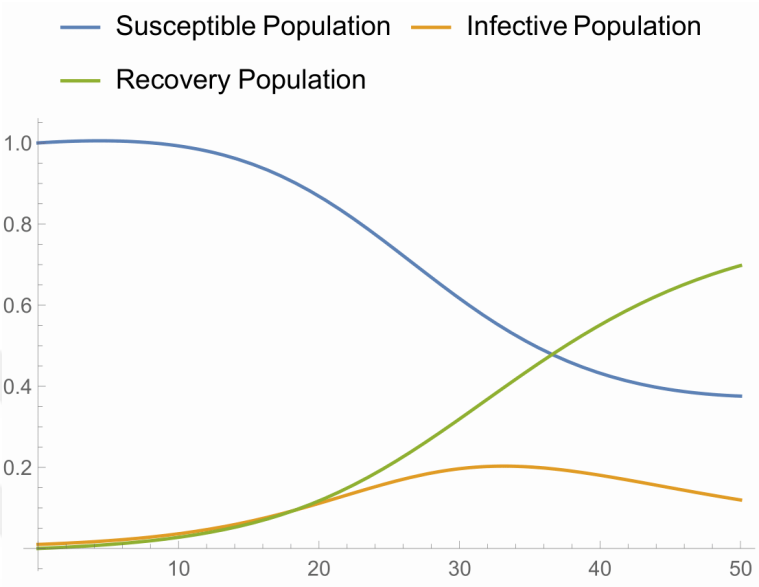

Figure 2.4 : SIR model when $\beta = 0.28, \alpha = 0.14, \Lambda = 0.015, d = 0.01, q = 0.002$

The graphic that appeared in the numerical solution is given by assigning certain values to the parameters of the well-known *SIR* model in Figure 2.4. In this graph, the horizontal axis represents the time, the vertical axis expresses susceptible, infective and recovery populations at time t. The graph is plotted when the threshold value is $R_0 < 1$,therefore the disease first spreads and then decreases. Reader can see [14] for more details.

2.2.1 Bifurcation Analysis

In this section, we shall examine bifurcation analysis of the well-known *SIR* model. As is known, bifurcation occurs when the threshold value is $R_0 = 1$. The equilibrium point is locally asymptotically stable by Theorem 1.2.1 when the threshold value $R_0 < 1$, and the unstable state occurs when the threshold value is $R_0 > 1$.

2.2.1.1 Castillo-Chavez and Song Theorem

Castillo-Chavez and Song bifurcation theorem is a useful method in determining the direction of the bifurcation at critical point which is called basic reproduction number R_0.

Theorem 2.2.1 (Castillo-Chavez and Song [6]). *Consider the following general system of ODEs with a parameter ϕ:*

$$\frac{dx}{dt} = f(x,\phi), \qquad f : R^n xR \to R^n, \qquad f \in C^2(R^n xR) \tag{2.14}$$

where 0 is an equilibrium point of the system. Assume the following conditions:

A_1. *$\mathscr{A} = D_x f(0,0) = (\frac{\partial f_i}{\partial x_j}(0,0))$ is the linearisation matrix of system that we define above around the equilibrium 0 with ϕ evaluated at 0. Zero is a simple eigenvalue of $\mathscr{A}$, and other eigenvalues have negative real parts.*

A_2. *The matrix $\mathscr{A}$ has a non-negative right eigenvector w and a left eigenvector v each corresponding to the zero eigenvalue.*

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \tag{2.15}$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0), \tag{2.16}$$

where f_k is the kth component of f.

The local dynamics of around 0 are totally determined by a and b.

i. *$a > 0$, $b > 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0 < \phi \ll 1$, 0 is unstable and there exists a negative and locally asymptotically stable equilibrium;*

ii. *$a < 0$, $b < 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is unstable; when $0 < \phi \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium;*

iii. *$a > 0$, $b < 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi \ll 1$, 0 is stable, and a positive unstable equilibrium appears;*

iv. $a < 0$, $b > 0$. *When* ϕ *changes from negative to positive,* 0 *changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.*

Particularly, if $a > 0$ *and* $b > 0$*, then a backward bifurcation occurs at* $\phi = 0$*. If* $a < 0$ *and* $b > 0$*, then a forward bifurcation occurs at* $\phi = 0$*.*

The proof of the theorem can be found at [15].

Remark. In practice, the following two observations are important.

1. In fact, the equilibrium point 0 by the theorem (2.2.1) is the disease free equilibrium point and ϕ is one of the parameter of the basic reproduction number R_0, also the critical value of ϕ is value of parameter which makes the basic reproduction number $R_0 = 1$.

2. It is known that if the disease free equilibrium point has positive entries, the right eigenvector w need not to be non-negative. It means components of the right eigenvector could be negative that correspond to positive entries in the disease free equilibrium point. In addition, components of the right eigenvector that correspond to zero entries in the disease free equilibrium point has to be non-negative [6].

We set $S = x_1$, $I = x_2$, $R = x_3$. By calling the system of equations as $\dot{x} = f(t)$ we shall write (2.4)-(2.6) as follows in therms of the new variables:

$$\begin{aligned} f_1 &= \Lambda - \frac{\beta x_1 x_2}{(x_1 + x_2 + x_3)} - d x_1, \\ f_2 &= \frac{\beta x_1 x_2}{(x_1 + x_2 + x_3)} - (d + q + \alpha) x_2, \\ f_3 &= \alpha x_2 - d x_3. \end{aligned} \tag{2.17}$$

The parameter ϕ which is given in theorem (2.2.1) is represented by β with critical value obtained from $R_0 = 1$,

$$\tilde{\beta} = d + q + \alpha.$$

The disease free equilibrium point of this model is $[\tilde{x_1} = \frac{\Lambda}{d}, \tilde{x_2} = 0, \tilde{x_3} = 0]$. The linearisation around the disease free equilibrium evaluated at $\tilde{\beta}$ is given by above

$$\mathscr{A} = \begin{pmatrix} -d & -\tilde{\beta} & 0 \\ 0 & -d - q - \alpha + \tilde{\beta} & 0 \\ 0 & \alpha & -d \end{pmatrix}.$$

Eigenvalues of this matrix are,

$$\lambda_1 = -d,$$
$$\lambda_2 = -d,$$
$$\lambda_3 = 0.$$

$\lambda_3 = 0$ is a simple eigenvalue of $D_x f$. The right and left eigenvectors can now be calculated to use in the theorem.

The right eigenvector w corresponding to the zero eigenvalue is found as

$$w = (-\frac{d+q+\alpha}{\alpha}, \frac{d}{\alpha}, 1)^T,$$

whereas the left eigenvector v corresponding to the zero eigenvalue is evaluated as

$$v = (0, 1, 0).$$

The second derivatives are evaluated at the disease free equilibrium $(\tilde{S}, \tilde{I}, \tilde{R}) = (\frac{\Lambda}{\mathrm{d}}, 0, 0)$ and with $\beta = \tilde{\beta}$.

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_2} = \frac{\partial^2 f_2}{\partial x_2 \partial x_1} = \frac{(d+q+\alpha)d}{\Lambda}$$

$$\frac{\partial^2 f_2}{\partial x_2 \partial \beta} = 1$$

By using these derivatives, a and b are found as follows:

$$b = \frac{\partial^2 f_2}{\partial x_2 \partial \beta} v_2 w_2 = (1)(\frac{d}{\alpha})(1) = \frac{d}{\alpha} > 0,$$
$$a = \frac{\partial^2 f_2}{\partial x_1 \partial x_2} v_2 w_1 w_2 + \frac{\partial^2 f_2}{\partial x_2 \partial x_1} v_2 w_2 w_1 = -2\frac{((d+q+\alpha)d)^2}{\alpha^2 \Lambda} < 0.$$

It is clearly seen that $a < 0$, $b > 0$. This shows according to the condition *iv* of the theorem 2.2.1, the sign of at least one eigenvalue of the matrix (2.7) changes from negative to positive. So the equilibrium point E_0 becomes unstable. When $R_0 < 1$, the equilibrium point E^* does not exist because its components are negative. When R_0 cross the value 1, the negative unstable equilibrium point E^* becomes positive and locally asymptotically stable.

2.3 *SIS* Models

In the *SIR* model which we have examined in the previous subsection, the recovering individuals leave the general population. And, they do not enter susceptible compartment again. In the very well-known *SIS* model that we shall examine now, we consider that not every disease will immunize. Therefore the infected individuals are possible to become infected again after recovery [16]. Therefore, we do not use the recovery class in this model. But consider the recovered infected individuals directly transfer to the susceptible compartment. Therefore in the *SIS* model, population divide into two subgroups. These are susceptible S and infected I classes.

The diagram of this model is given in the figure (2.5), Here Λ is the number of

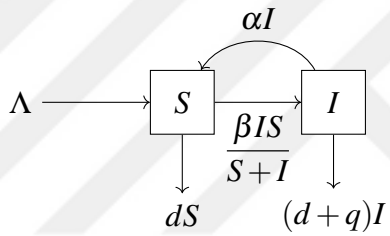

Figure 2.5 : Transfer diagram of SIS model.

susceptible individuals enter into the susceptible compartment either by birth or immigration. β is the transmission rate of disease. $\frac{\beta IS}{S+I}$ is the average of the transmission number per day from susceptible to infected compartments when they interact, which is called standard incidence [16]. d is the natural death rate, α is the recovery rate and q is the disease-related death rate. So the system of the differential equations can be written according to the figure 2.5

$$\frac{dS}{dt} = \Lambda - \frac{\beta IS}{S+I} - dS + \alpha I, \tag{2.18}$$

$$\frac{dI}{dt} = \frac{\beta IS}{S+I} - (d+q+\alpha)I. \tag{2.19}$$

It is assumed that the population size is constant. First we should find out which condition must be provided for this assumption. So it is known that,

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} = 0 \tag{2.20}$$

Then, let us substitute the equations (2.18) and (2.19) in the equation (2.20),

$$\frac{dN}{dt} = \Lambda - \frac{\beta IS}{S+I} - dS + \alpha I + \frac{\beta IS}{S+I} - (d+q+\alpha)I,$$
$$\frac{dN}{dt} = \Lambda - dN - qI.$$

Therefore, the population size remains constant when $\Lambda = dN + qI$ is provided.

Since, this system of equations is non-linear, we should do linearisation. Let us find the equilibrium points of the system,

$$\frac{dS}{dt} = 0 \Rightarrow \Lambda - \frac{\beta IS}{S+I} - dS + \alpha I = 0$$
$$\frac{dI}{dt} = 0 \Rightarrow \frac{\beta IS}{S+I} - (d+q+\alpha)I = 0.$$

Solution is determined as:

$$E_0 = (\frac{\Lambda}{d}, 0),$$
$$E^* = (\frac{\Lambda(d+q+\alpha)}{\beta(d+q) - q(d+q+\alpha)}, \frac{\Lambda(d+q+\alpha-\beta)}{q(d+q+\alpha) - (d+q)\beta}).$$

Then let us write the Jacobian matrix of system,

Equilibrium Point / (S,I)	**Description**
$E_0 = (\frac{\Lambda}{d}, 0)$	Disease-free equilibrium point. The only non-zero population is susceptible human population $\frac{\Lambda}{d}$.
$E^* = (S^*, I^*)$	Endemic Equilibrium point.

Table 2.2 : Equilibrium points of SIS model.

$$J = \begin{pmatrix} -d + \frac{IS\beta}{(I+S)^2} - \frac{I\beta}{I+S} & \alpha + \frac{IS\beta}{(I+S)^2} - \frac{S\beta}{I+S} \\ -\frac{IS\beta}{(I+S)^2} + \frac{I\beta}{I+S} & -d - q - \alpha - \frac{IS\beta}{(I+S)^2} + \frac{S\beta}{I+S} \end{pmatrix}. \tag{2.21}$$

Let us investigate stability analysis of the equilibrium points by substituting equilibrium points in the Jacobian matrix.

1. Equilibrium point $E_0 = (\frac{\Lambda}{d}, 0)$

   The Jacobian matrix is

$$J(E_0) = \begin{pmatrix} -d & \alpha - \beta \\ 0 & -d - q - \alpha + \beta \end{pmatrix}. \tag{2.22}$$

Eigenvalues of matrix (2.22) are,

$$\lambda_1 = -d,$$
$$\lambda_2 = -d - q - \alpha + \beta.$$

It is clear that the eigenvalue λ_1 is negative. But the eigenvalue λ_2 becomes negative when the condition

$$\frac{\beta}{d+q+\alpha} < 1$$

is satisfied. In this case, all eigenvalues are negative then it can be said that the equilibrium point E_0 is a locally asymptotically stable according to the theorem (1.2.1). If

$$\frac{\beta}{d+q+\alpha} > 1,$$

then λ_2 is positive. Therefore equilibrium point E_0 is called unstable equilibrium point.So the threshold value will be

$$R_0 = \frac{\beta}{d+q+\alpha}. \tag{2.23}$$

As one can see easily the basic reproduction number R_0 in this *SIS* model is the same as in the very well-known *SIR* model. The interpretation is very straightforward: the rate of infection is greater than the rate of the sum of natural death, disease-related death and transmission rate.

In Figure (2.6) it is shown that, when $R_0 < 1$, the solution of the system starts from the initial condition that adequately close the equilibrium point E_0 and converge to this equilibrium point at $t \to \infty$. In other words, while the infectious individual population size decrease to zero over time, the susceptible individual population size is steady state.

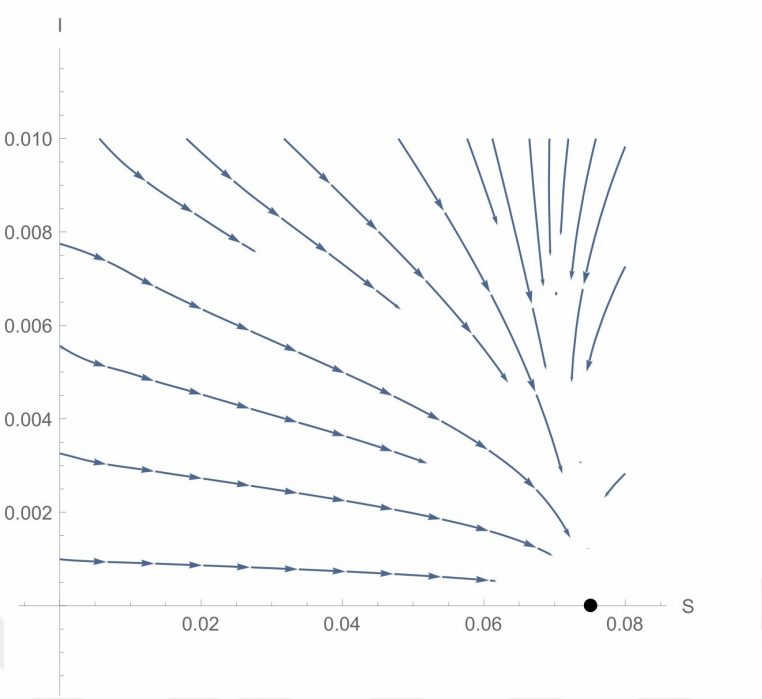

Figure 2.6 : Phase portrait of equilibrium point $E_0 = (\frac{\Lambda}{d}, 0)$ while $R_0 < 1$. When $\Lambda = 0.03$, $\beta = 0.4$, $d = 0.4$, $q = 0.03$, $\alpha = 0.1$

Therefore, it can be seen that the equilibrium point is asymptotically stable by the theorem 1.2.1.

2. Equilibrium point $E^* = (S^*, I^*)$

   Solution of the system is found as

$$S^* = \frac{\Lambda}{d + (q+d)(R_0 - 1)},$$
$$I^* = \frac{(R_0 - 1)\Lambda}{d + (q+d)(R_0 - 1)}.$$

   So it is clear that the equilibrium point E^* exists if and only if when $R_0 > 1$. After writing this equilibrium point, now we can investigate stability analysis of the endemic equilibrium point E^* by substituting it in the Jacobian matrix,

$$J(E^*) = \begin{pmatrix} -d + \frac{I^* S^* \beta}{(I^* + S^*)^2} & \alpha + \frac{I^* S^* \beta}{(I^* + S^*)^2} - \frac{S^* \beta}{I^* + S^*} \\ -\frac{I^* S^* \beta}{(I^* + S^*)^2} + \frac{I^* \beta}{I^* + S^*} & -d - q - \alpha - \frac{I^* S^* \beta}{(I^* + S^*)^2} + \frac{S^* \beta}{I^* + S^*} \end{pmatrix}. \quad (2.24)$$

   The characteristic polynomial of this matrix (2.24) is

$$P(\lambda) = (\lambda^2 + h_1 \lambda + h_2) \quad (2.25)$$

where

$$h_1 = \beta - q - \alpha,$$
$$h_2 = \frac{(d+q+\alpha-\beta)(q(d+q+\alpha)-(d+q)\beta)}{\beta}.$$

We can write h_2 as

$$h_2 = \frac{(d+q+\alpha)(R_0-1)((d+q)R_0-q)}{R_0}. \tag{2.26}$$

Since the existence of the equilibrium point E^* depends on being $R_0 > 1$, so h_2 is always positive. If $\frac{q+\alpha}{\beta} < 1$, one can see that $h_1 > 0$. And the sum of the eigenvalues are negative. $\lambda_1\lambda_2 > 0$ and $\lambda_1 + \lambda_2 < 0$ all eigenvalues of matrix (2.24) are negative. Therefore, endemic steady state equilibrium point E^* of the system is locally asymptotically stable by the theorem (1.2.1).

If $\frac{q+\alpha}{\beta} > 1$, one can see that $h_1 < 0$. And the sum of the eigenvalues are positive. $\lambda_1\lambda_2 > 0$ and $\lambda_1 + \lambda_2 > 0$ all eigenvalues of matrix (2.24) are positive. Therefore, endemic equilibrium point is an unstable equilibrium point.

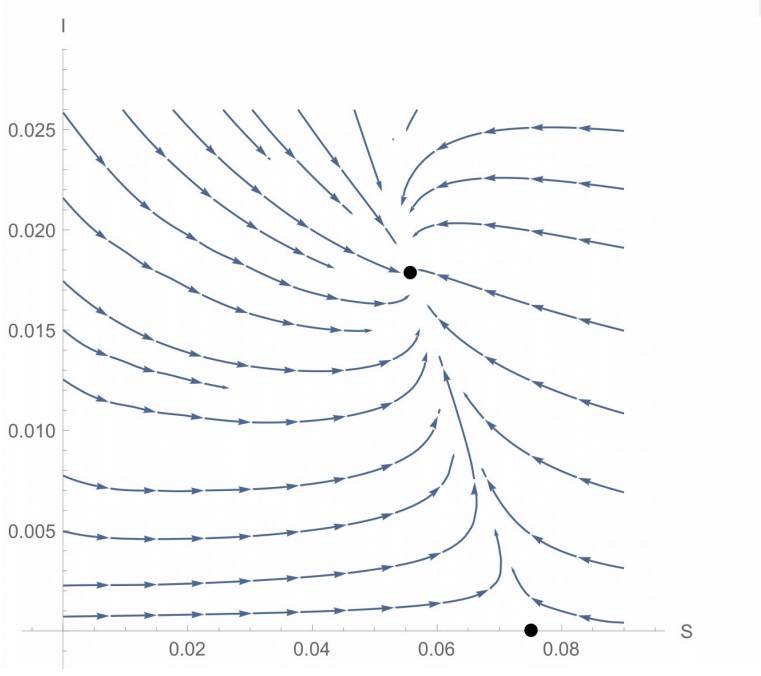

Figure 2.7 : Phase portrait of equilibrium points $E_0 = (\frac{\Lambda}{d}, 0)$ and $E^* = (S^*, I^*)$ while $R_0 > 1$. When $\Lambda = 0.03$, $\beta = 0.7$, $d = 0.4$, $q = 0.03$, $\alpha = 0.1$

In Figure (2.7), it is shown that when $R_0 > 1$ the solution of the system starts from the initial condition that adequately closes the equilibrium point E^* and converges to

this equilibrium point at $t \to \infty$. In other words, the infectious individual population size increases when the susceptible individual population size decreases. Hence, it can be seen that the equilibrium point E^* is asymptotically stable according to the theorem (1.2.1). Also, the flow of the system converges to equilibrium point E^* and the flow moves away from the equilibrium point E_0 and this equilibrium point is called an unstable equilibrium point.

2.4 Prey-Predator Models

In this section, we will examine the of prey-predator equations, also known as Lotka-Volterra equation. In this type of model, we have two types of population. One of them is prey and the other is a predator.

The Lotka–Volterra predator-prey model was initially recommended by Alfred J. Lotka in 1910. In 1925, Lotka used the equations to analyse predator-prey interactions in his book Elements of Physical Biology [17], and reproduce the equations that we know today. Vito Volterra, who was interested in the statistical analysis of fish catches in the Adriatic, independently investigated the equations in 1926 [18]. The equations are based on the investigation that the predator-prey dynamics are often oscillatory. More detailed explanations are described by (Martcheva, 2015) in the reference [6]. Also, the Lotka-Volterra model makes some assumptions for the environment and evolution of the prey-predator populations:

- The prey population always can find sufficient food.
- The food supply of the predator population depends on the prey population.
- The environment does not change in favor of a species, in addition to genetic adaptation is slow enough.

If we explain the prey-predator model a little more, we can say that if there is no predator in the environment, the number of prey will gradually increase. This increment continues until the food stock of the previous ones is exhausted. We call that *carrying capacity*. As a predator enters the environment, the number of prey will decrease while the number of predators will increase for a while. Likewise, in the

absence of prey, the number of predators will begin to decrease, and in the presence of prey, it will continue to increase.

Then, let's describe the model's diagram,

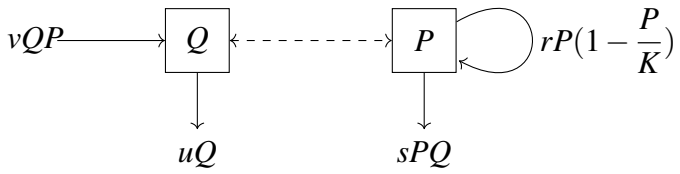

Figure 2.8 : Transfer diagram of Prey-Predator model.

where P represents prey and Q represents predator. We can write the system of differential equation according to the diagram shown in figure 2.8;

$$\frac{dP}{dt} = rP(1-\frac{P}{K}) - sPQ, \tag{2.27}$$

$$\frac{dQ}{dt} = vQP - uQ. \tag{2.28}$$

Here, predators eat preys at the rate of s. Where v represents the growing rate of predators. The predators die or leave from the population at a rate of u for various other reasons. Also, K is the *carrying capacity* of the prey in the absence of the predator, and r is the growth rate of the prey population.

Since the prey-predator model is a non-linear system, we should do linearisation. First equilibrium points must be found; then, linearisation is done by finding the Jacobian matrix. After, it can be talked about the stability analysis of the equilibrium points.

System of equation (2.27)-(2.28) has three equilibrium points. The first equilibrium point which corresponds to the disappearance of both prey and predator is called the *extinction equilibrium point*. It is given by $E_0 = (0,0)$.

The second equilibrium point which corresponds to the absence of the predator only and existence of the prey population. This equilibrium point called the *predator-extinction equilibrium* which is given by $E_1 = (K,0)$.

The third and last equilibrium point which corresponds to a predator-prey coexistence is given by $E^* = (\frac{u}{v}, \frac{r(Kv-u)}{Ksv})$.

These equilibrium points can be seen more clearly in the Table 2.3. For more detailed information the reader may look at again (Martcheva, 2015)'s book [6].

Equilibrium Point / (P,Q)	**Description**
$E_0 = (0,0)$	Extinction equilibrium.
$E_1 = (K,0)$	Predator-extinction equilibrium point.
$E^* = (\frac{u}{v}, \frac{r(Kv-u)}{Ksv})$	Predator–prey coexistence equilibrium point.

Table 2.3 : Equilibrium points of Prey-Predator Model.

Now we can write the Jacobian matrix of non-linear system of equation,

$$J = \begin{pmatrix} -u+Pv & Qv \\ -Ps & -\frac{Pr}{K} + (1+\frac{P}{K})r - Qs \end{pmatrix}$$

To investigate stability of the equilibrium points.

1. Equilibrium point $E_0 = (0,0)$

   We can do the stability analysis by substituting the first found equilibrium point E_0 in the Jacobian matrix. This matrix also called the *community matrix* in some literatures

   $$J(E_0) = \begin{pmatrix} -u & 0 \\ 0 & r \end{pmatrix} \tag{2.29}$$

   whose eigenvalues of matrix (2.29) are

   $$\lambda_1 = -u$$
   $$\lambda_2 = r.$$

   Since $\lambda_1 < 0$ but $\lambda_2 > 0$ this equilibrium point is called unstable equilibrium point. Let us consider the other equilibrium points.

2. Equilibrium point $E_1 = (K,0)$

   Substitution of the second equilibrium point E_1 in the Jacobian matrix gives

   $$J(E_1) = \begin{pmatrix} -u+Kv & 0 \\ -Ks & -r \end{pmatrix} \tag{2.30}$$

   whose eigenvalues of this matrix are,

   $$\lambda_1 = -r,$$
   $$\lambda_2 = Kv - u.$$

When we consider these eigenvalues, it can be easily seen that the first eigenvalue $\lambda_1 < 0$ but the other eigenvalue's sign depends on some conditions. If $\frac{Kv}{u} < 1$, then $\lambda_2 < 0$. It can be said that the basic reproduction number is

$$R_0 = \frac{Kv}{u}. \tag{2.31}$$

If $R_0 < 1$, the number of prey increases and the prey only equilibrium point is called locally asymptotically stable from the theorem (1.2.1). Otherwise when $R_0 > 1$, the number of predators increases, the number of prey decreases and equilibrium point E_1 is called unstable equilibrium point. If $R_0 = 1$, bifurcation may occur.

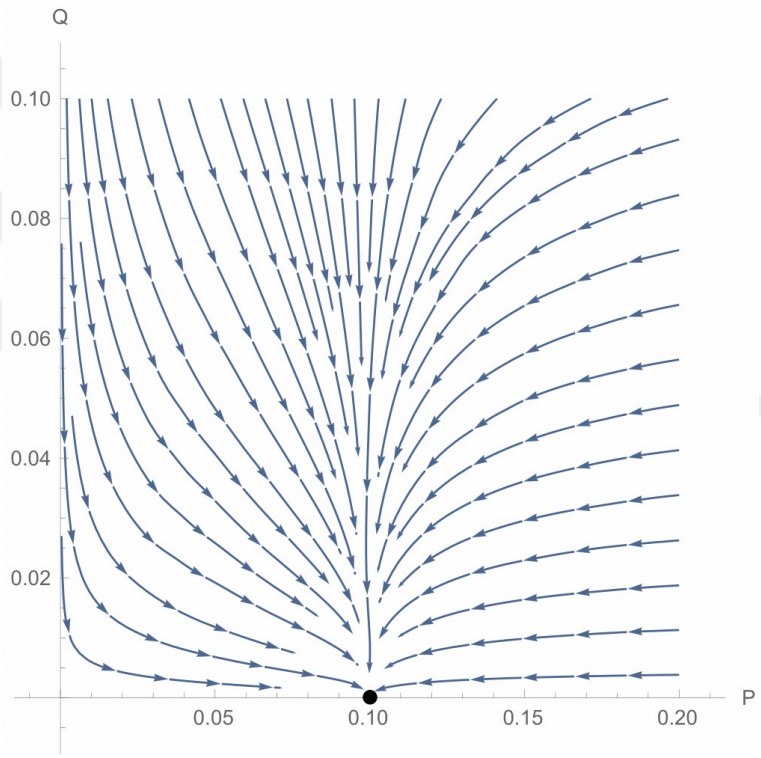

Figure 2.9 : Phase portrait of equilibrium point $E_1 = (K, 0)$ while $R_0 < 1$. When $r = 0.7$, $K = 0.1$, $s = 0.2$, $v = 0.4$, $u = 0.3$

In figure (2.9) it is seen that, when $R_0 < 1$ for the solution of the system starts from the initial condition that adequately closes the equilibrium point E_1 and converge to this equilibrium point at $t \to \infty$. In other words, when the predator population size decreases to zero over time, the prey population size is steady state. Therefore, it can be seen that the equilibrium point is asymptotically stable from the Theorem 1.2.1.

3. Equilibrium point $E^* = (\frac{u}{v}, \frac{r(Kv-u)}{Ksv})$

   If the equilibrium point E^* is written as;

$$E^* = (\frac{u}{v}, \frac{ru(R_0-1)}{Ksv}),$$

   the stability analysis can be done more easily. Hence it can be seen that, equilibrium point E^* exists when $R_0 > 1$. Substitution of the equilibrium point E^* gives

$$J(E^*) = \begin{pmatrix} 0 & \frac{r(Kv-u)}{Ks} \\ -\frac{su}{v} & -\frac{ru}{Kv} \end{pmatrix}. \tag{2.32}$$

   The characteristic equation of matrix (2.32) is

$$P(\lambda) = \lambda^2 + h_1\lambda + h_2$$

   where

$$h_1 = \frac{r(Kv-u)}{Kv} + \frac{2ru}{Kv} - r,$$
$$h_2 = \frac{ru(Kv-u)}{Kv}.$$

   Let us simplify h_1 and h_2 as follows:

$$h_1 = r + \frac{1}{R_0},$$
$$h_2 = ru(1 - \frac{1}{R_0})$$

   where R_0 is given by the equation (2.31). Existence of the equilibrium point E^* depends on $R_0 > 1$. It is obvious that $h_1 > 0$ and $h_2 > 0$ and therefore $\lambda_1 + \lambda_2 < 0$ because $\lambda_1 + \lambda_2 = -h_1$ and $\lambda_1\lambda_2 > 0$ as $\lambda_1\lambda_2 = h_2$. Thus all eigenvalues of matrix (2.32) are negative. As a result, according to the theorem (1.2.1) the equilibrium point E^* is called locally asymptotically stable equilibrium point. And if $R_0 = 1$, bifurcation may occur.

In Figure (2.10) it can be seen that, when $R_0 > 1$ for the solution of the system starts from the initial condition that adequately closes the equilibrium point E^* and converge to this equilibrium point at $t \to \infty$.

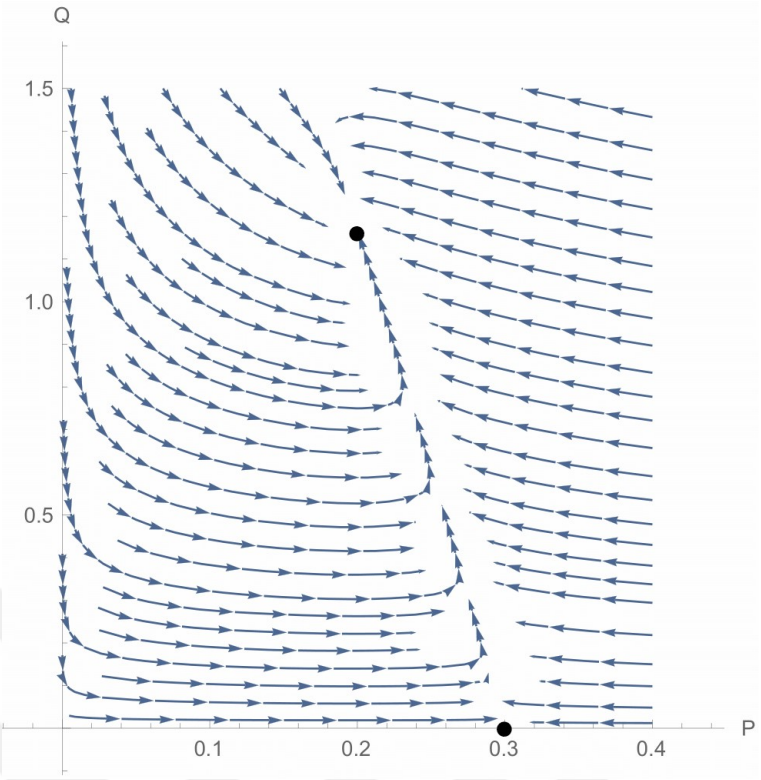

Figure 2.10 : Phase portrait of equilibrium point $E^* = (K,0)$ and $E^* = (\frac{u}{v}, \frac{r(Kv-u)}{Ksv})$ while $R_0 > 1$. When $r = 0.7$, $K = 0.3$, $s = 0.2$, $v = 0.5$, $u = 0.1$

In other words, when the predator population size increases by feeding with preys and the prey population size decrease depend on this situation. Therefore, it can be seen that the equilibrium point E^* is locally asymptotically stable from the theorem (1.2.1). Also, while flow of the system converges to equilibrium point E^*, it moves away from the equilibrium point E_0 and equilibrium point E_0 is called unstable equilibrium point.

3. STABILITY ANALYSIS OF A MATHEMATICAL MODEL OF CRIMEAN CONGO HAEMORRHAGIC FEVER DISEASE

3.1 Introduction

Here, we will present a mathematical model expressing the spread of Crimean Congo haemorrhagic fever disease, by considering the effect of chickens on the tick population.

> Chickens were shown to be natural predators of ticks. *Rhipicephalus appendiculatus* (the brown ear tick) were recovered in large numbers from the crops and gizzards of chickens which had scavenged for 30 min- 1 hour among tick-infested cattle. Other ticks recovered were *Amblyomma variegatum* (tropical bont tick) and *Boophilus decoloratus* (blue cattle tick). The numbers of ticks recovered ranged from 3 to 331, with an average of 81 per chicken. Cattle facilitated the predation of ticks by certain behavioural actions. Chickens also picked up both engorged and unengorged ticks seeded on vegetation, but unengorged ticks were preferred [19].

In the nature, birds and chickens eat ticks. They keep many creatures, especially cattle, away from them. Therefore, birds are natural predators of ticks. Veterinarians around the world used the tick control method for cattle. Chickens are natural predators of ticks that feed on cattle, so chickens can be used as part of the tick control plan. If chickens are allowed to access pastures, they can eat a significant number of insects, especially ticks. Ticks climb the grass and wait for a suitable host. Thus, ticks that climb to the top of the grass can be noticed by chickens and eaten. Large ticks are easily eaten by chickens. This issue has been tackled more extensively by Sahito (2013) in [20].

Let us give some more information about the ticks, they have three stages after hatching. These are larval, nymph and adult stages. If an adult female tick is infected, then the Crimean-Congo haemorrhagic fever (CCHF) virus can pass into the eggs after

mating. Then ticks hatch as larvae and they are fed by the blood of small mammals and birds [21]. If they do not have CCHF virus, they can get CCHF virus from their hosts. After the larvae stage, they turn into nymphs. Nymphs also engorge by the blood of small mammals and birds and may get CCHF virus from their hosts. After the nymphs stage, they turn into adult female or male ticks. Adults are fed by blood of cattle to mate and lay eggs and may get CCHF virus. Only at the adult stage, a tick can bite and feed on humans and transfer CCHF virus [22]. People living in rural areas where the reproduction rate of ticks is more are likely to be exposed to bites of ticks. There are several ways to be protected against tick bites.

- Precautions like wearing long-sleeved clothing and trousers should be taken during trekking or fieldwork.
- Trouser cuff should be inserted into the socks during the periods when ticks are dense.
- While hiking, care should be taken while walking in the middle of the paths because ticks are usually found under the leaf, close to the soil.
- Insect repellent sprays can be a preventive method for ticks and other insects.
- Ticks on animals should not be removed with bare hands. If the tick carries a virus, the disease can be transmitted by contact with the blood or body fluid of the host animal.
- Care should be taken while applying pesticides in the fields during the breeding time of ticks.
- With the possibility of tick bites, animals should be given parasite vaccines in a timely manner.

Crimean Congo haemorrhagic fever disease was first described in the 12th century in Tajikistan. During the years 1944-45, it was often seen among the Soviet soldiers who helped collect products on the Western Crimean steppes in the Crimean region of Russia [23]. Congo virus was detected from a patient with a fever in Zaire in 1956. In 1969, Congo virus and Crimean haemorrhagic fever viruses were identified to be the same virus, and the disease was renamed Crimean-Congo Haemorrhagic Fever. The

disease first attracted attention in Turkey in 2002, and in 2003, a definitive diagnosis was made. The cases of Crimean Congo haemorrhagic fever are more common in the spring and summer, beginning from the time ticks are activated. The Crimean Congo Haemorrhagic Fever cases, which attracted attention for the first time around Tokat province, are mostly concentrated in the north of Central Anatolia, the Central Black Sea, and north of Eastern Anatolia. It is mentioned by (Tartar, Balın, Akbulut and Demirdag, 2019) in reference [24].

According to data from the Ministry of Health of Turkey Crimean-Congo haemorrhagic fever begin to appear in the spring with the fatality rate hover around 4-5% in Turkey. Considering the incidence of cases by years, it can be mentioned that there is an increase and decrease tendency and the highest case was 1318 individuals in 2009. Although 343 Crimean Congo haemorrhagic fever cases were identified in 2017, it still remains important in Turkey.

3.2 Problem

This study investigates the effects of chickens on ticks within the framework of the spread of the disease. We examine three types of epidemiological models in the thesis; Tick Logistic Growth - Chicken Logistic Growth Model, Tick Constant - Chicken Logistic Growth Model and Tick Logistic Growth - Chicken Constant Model. We first consider the growth of both tick and chicken populations as the logistic growth equation. We analyse this model in section 3.2.1. Then we consider the growth of the chicken population as logistic growth; details are given in section 3.2.2. Finally, we consider the growth of ticks as logistic growth, the work of which is given in detail in section 3.2.3. We use a system of four ODE's to represent the interactions between infected and susceptible populations of humans and ticks. We add a fifth ODE which models the dynamics of a chicken population and its effect on the ticks. The model assumes finite total population of humans which is denoted by N, and finite total population of ticks are represented by T. The human population is mutually divided into two sub-populations. These are the susceptible class which is represented by S and the infected human class which is represented by I. Similarly we subdivide the tick population into susceptible and infected ticks compartment, they are denoted by T_s and T_i, respectively. The chicken population is denoted by B.

Then, we do the following model assumptions:

- Crimean Congo haemorrhagic fever is an *SIS* model for humans; there is no immunity on the recovery. Someone with treatment can be infected again when bitten by a tick carrying the virus.
- The age structure is not included in the model because there is no age group specifically exposed to Crimean Congo haemorrhagic fever disease. Only the people living in rural areas may be exposed to this disease are included in the model.
- All ticks are considered in the model are adult ticks and also somehow infective ticks have no way of recovering and gaining immunity.
- All parameters are constant. In reality, the parameters depend on the region being modelled, the population growth rates depends on, the season, temperature, etc.

Let us list the parameters and variables common in all 3 models for a better understanding.

- $N \rightarrow$ Total population size.
- $S \rightarrow$ Susceptible human population size.
- $I \rightarrow$ Infected human population size.
- $T_s \rightarrow$ Susceptible tick population size.
- $T_i \rightarrow$ Infected tick population size.
- $B \rightarrow$ Size of the bird population.
- $\Lambda_1 \rightarrow$ Humans: Population growth.
- $\Lambda_2 \rightarrow$ Ticks: Population growth.
- $\Lambda_3 \rightarrow$ Chickens: Population growth.
- $\gamma \rightarrow$ Recovery rate of humans.
- $\beta_1 \rightarrow$ Transmission rate: Tick to human.
- $\beta_2 \rightarrow$ Transmission rate: Tick to tick.

- $\beta_3 \rightarrow$ Transmission rate: Bird to tick.
- $d \rightarrow$ Natural death rate of humans.
- $\mu \rightarrow$ Natural death rate of chickens.
- $q_0 \rightarrow$ Natural death rate of ticks.
- $q_1 \rightarrow$ Birds caused death rate of ticks.
- $\alpha \rightarrow$ Crimean Congo haemorrhagic fever death rate of humans.
- $r_1 \rightarrow$ Growth rate of chickens.
- $r_2 \rightarrow$ Growth rate of ticks.
- $K_1 \rightarrow$ Carrying capacity of chickens.
- $K_2 \rightarrow$ Carrying capacity of ticks.

3.2.1 Tick logistic growth - chicken logistic growth model

In this subsection, we consider the growth of both susceptible ticks and chicken populations as logistic growth.

The diagram of the Tick Logistic Growth - Chicken Logistic Growth model is shown in the figure 3.1.

Figure (3.1) represents dynamics the model. The bidirectional dotted arrows between the boxes represent the interaction between the classes.

So, we can write the system of differential equation;

$$\frac{dS}{dt} = \Lambda_1 + \gamma I - \beta_1 S T_i - dS, \tag{3.1}$$

$$\frac{dI}{dt} = \beta_1 S T_i - (\alpha + d + \gamma) I, \tag{3.2}$$

$$\frac{dT_s}{dt} = r_2 T (1 - \frac{T}{K_2}) - \beta_2 T_s T_i - \beta_3 T_s B - (q_0 + q_1 B) T_s, \tag{3.3}$$

$$\frac{dT_i}{dt} = \beta_2 T_s T_i + \beta_3 T_s B - (q_0 + q_1 B) T_i, \tag{3.4}$$

$$\frac{dB}{dt} = r_1 B (1 - \frac{B}{K_1}). \tag{3.5}$$

The variation of the susceptible human population, S: Equation (3.1) represents the susceptible human population dynamics. The increments of the susceptible population

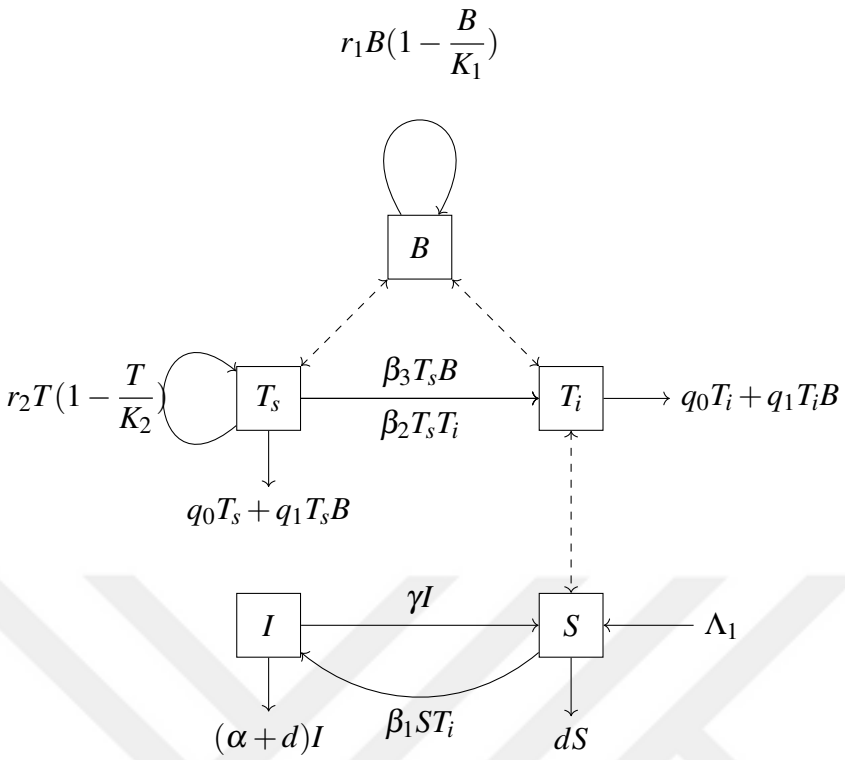

Figure 3.1 : Tick Logistic Growth - Chicken Logistic Growth Model Chart.

are shown by Λ_1 and γI. Here, Λ_1 represents the number of individuals entering the environment. γ represents the recovery rate of infected individuals. Terms that cause decrements of the susceptible human population are shown by $\beta_1 ST_i$ and dS. β_1 represents the transmission rate of disease between susceptible and infective individuals. And d represents the natural death rate.

The variation of the infected human population, I : Equation (3.2) represents the dynamics of the infected human population. The source term of infected people compartment is the term $\beta_1 ST_i$. Individuals leave the compartment of infected people either with the healing condition which is represented by γI, or leave with disease-related death or natural death which are represented by αI and dI, respectively.

The variation of the susceptible tick population, T_s: Equation (3.3) represents the dynamics of the susceptible tick population. In this model, we write the source term of the susceptible tick population with the logistic equation because the tick population varies depending on the temperature of the environment, the season and the number of nutrients in the environment. The parameters of growth rate and carrying capacity are represented by r_2 and K_2, respectively in this logistic equation. Terms that cause decrements of susceptible tick population are represented by $\beta_3 T_sB$, $\beta_2 T_sT_i$,

q_0T_s and q_1T_sB. The parameter β_3 is the transmission rate between ticks and chickens. Transmission rate between ticks is denoted by β_2.

The variation of the infected tick population, T_i: Equation (3.4) represents the dynamics of the infected tick compartment. The source terms of the infected tick population are given by β_3T_sB and $\beta_2T_sT_i$. Terms that cause the decrements of infected tick population are similar to the susceptible tick population.

The variation of the chicken population, B: Equation (3.5) describes the dynamics of the chicken population. In this model we assume, the source term of the chickens as logistic growth. Where r_1 is the growth rate and K_1 is the carrying capacity of chickens.

Since, this system of equations is a non-linear system, we should do linearisation. We must find equilibrium points first. After finding equilibrium points we should write the Jacobian matrix of the system. Then, we can investigate the stability analysis of the equilibrium points by substituting equilibrium points in the Jacobian matrix.

Since $S+I=N$ and $T_s+T_i=T$, to analyse more easily, we can write the following equations:

$$\frac{dN}{dt} = \Lambda_1 - dN - \alpha I, \tag{3.6}$$

$$\frac{dI}{dt} = \beta_1(N-I)T_i - (\alpha+d+\gamma)I, \tag{3.7}$$

$$\frac{dT}{dt} = r_2T(1-\frac{T}{K_2}) - (q_0+q_1B)T, \tag{3.8}$$

$$\frac{dT_i}{dt} = \beta_2(T-T_i)T_i + \beta_3(T-T_i)B - (q_0+q_1B)T_i, \tag{3.9}$$

$$\frac{dB}{dt} = r_1B(1-\frac{B}{K_1}). \tag{3.10}$$

The equilibrium points of the system (3.6)-(3.10) are given in the table (3.1).

Equilibrium Points / (N,I,T,T_i,B)	**Description**
$E_0 = (\frac{\Lambda_1}{d},0,0,0,0)$	Disease free equilibrium point.
$E_1 = (\frac{\Lambda_1}{d},0,0,0,K_1)$	Disease free equilibrium point.
$E_2 = (\frac{\Lambda_1}{d},0,\frac{K_2(r_2-q_0)}{r_2},0,0)$	Disease free equilibrium point which exist $r_2 > q_0$.
$E_3 = (\bar{N},\bar{I},\bar{T},\bar{T}_i,0)$	Endemic Equilibrium point.
$E^* = (N^*,I^*,T^*,T_i^*,K_1)$	Endemic Equilibrium point.

Table 3.1 : Equilibrium Points of Tick Logistic Growth - Chicken Logistic Growth Model.

After finding equilibrium points of the system (3.6)-(3.10) which we are shown in the Table 3.1, we can write the Jacobian matrix of the system,

$$J = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ T_i\beta_1 & -d-\alpha-T_i\beta_1-\gamma & 0 & (N-I)\beta_1 & 0 \\ 0 & 0 & r_2-q_0-Bq_1-\frac{2r_2T}{K_2} & 0 & -q_1T \\ 0 & 0 & T_i\beta_2+B\beta_3 & (T-2T_i)\beta_2-B(q_1+\beta_3)-q_0 & \beta_3(T-T_i)-q_1T_i \\ 0 & 0 & 0 & 0 & r_1-\frac{2Br_1}{K_1} \end{pmatrix}. \tag{3.11}$$

Let us investigate the stability of the equilibrium points.

1. Equilibrium Point $E_0 = (\frac{\Lambda_1}{d},0,0,0,0)$

   This equilibrium point contains only the susceptible human population $N = \frac{\Lambda_1}{d}$.

   Stability analysis can be done by substituting the disease-free equilibrium point E_0 in the Jacobian matrix,

$$J(E_0) = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ 0 & -d-\alpha-\gamma & 0 & \frac{\beta_1\Lambda_1}{d} & 0 \\ 0 & 0 & r_2-q_0 & 0 & 0 \\ 0 & 0 & 0 & -q_0 & 0 \\ 0 & 0 & 0 & 0 & r_1 \end{pmatrix}.$$

   Eigenvalues of this matrix are,

$$\lambda_1 = -d,$$

$$\lambda_2 = -q_0,$$

$$\lambda_3 = r_1,$$

$$\lambda_4 = r_2 - q_0,$$

$$\lambda_5 = -d-\alpha-\gamma.$$

It is obvious that, λ_1, λ_2 and λ_5 are less than zero, λ_3 is greater than zero. Hence we can say that the equilibrium point E_0 is an unstable equilibrium point.

2. Equilibrium Point $E_1 = (\frac{\Lambda_1}{d}, 0, 0, 0, K_1)$

   This equilibrium point contains only susceptible human and chicken populations $N = \frac{\Lambda_1}{d}$ and $B = K_1$, respectively. If we substitute equilibrium point E_1 in Jacobian matrix, we obtain;

$$J(E_1) = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ 0 & -d-\alpha-\gamma & 0 & \frac{\beta_1\Lambda_1}{d} & 0 \\ 0 & 0 & r_2 - q_0 - K_1 q_1 & 0 & 0 \\ 0 & 0 & K_1\beta_3 & -q_0 - K_1(q_1+\beta_3) & 0 \\ 0 & 0 & 0 & 0 & -r_1 \end{pmatrix}.$$

   Eigenvalues of this matrix are,

$$\begin{aligned} \lambda_1 &= -d, \\ \lambda_2 &= -r_1, \\ \lambda_3 &= r_2 - q_0 - K_1 q_1, \\ \lambda_4 &= -q_0 - K_1(q_1+\beta_3), \\ \lambda_5 &= -d-\alpha-\gamma. \end{aligned}$$

   It is clear that the sign of the eigenvalues λ_1, λ_2, λ_4, λ_5 are negative. If

$$\frac{r_2}{q_0 + K_1 q_1)} < 1, \tag{3.12}$$

   the sign of λ_3 becomes negative too. And the equilibrium point E_1 is called locally asymptotically stable by the theorem (1.2.1). The basic reproduction number is defined as

$$R_0 = \frac{r_2}{q_0 + K_1 q_1}. \tag{3.13}$$

   If the growth rate of ticks which is represented by r_2 is greater than the denominator in R_0, then, ticks in the environment increase. If $R_0 = 1$, bifurcation may occur. If the growth rate of ticks r_2 is less than the denominator in R_0, then equilibrium point is a stable equilibrium point. In this case, flow approaches the equilibrium point, the spread of ticks decreases. The spread of the disease also decreases correspondingly.

3. Equilibrium point $E_2 = (\frac{\Lambda_1}{d}, 0, \frac{K_2(r_2-q_0)}{r_2}, 0, 0)$

   If we substitute the equilibrium point E_2 in the Jacobian matrix,

$$J(E_1) = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ 0 & -d-\alpha-\gamma & 0 & \frac{\beta_1\Lambda_1}{d} & 0 \\ 0 & 0 & q_0-r_2 & 0 & \frac{K_2q_1(q_0-r_2)}{r_2} \\ 0 & 0 & 0 & K_2\beta_2 - \frac{q_0(r_2+K_2\beta_2)}{r_2} & \beta_3(K_2 - \frac{K_2q_0}{r_2}) \\ 0 & 0 & 0 & 0 & r_1 \end{pmatrix}$$

   is obtained. Eigenvalues of this matrix are

$$\begin{aligned} \lambda_1 &= -d, \\ \lambda_2 &= r_1, \\ \lambda_3 &= q_0 - r_2, \\ \lambda_4 &= K_2\beta_2 - \frac{q_0(r_2+K_2\beta_2)}{r_2}, \\ \lambda_5 &= -d-\alpha-\gamma. \end{aligned}$$

   Since $\lambda_2 > 0$, the equilibrium point E_2 is an unstable equilibrium point.

4. Equilibrium point $E_3 = (\bar{N}, \bar{I}, \bar{T}, \bar{T}_i, 0)$

   The $\bar{N}$, $\bar{I}$, $\bar{T}$ and $\bar{T}_i$ are found as

$$\begin{aligned} \bar{N} &= \frac{(d+\alpha+\bar{T}_i\beta_1+\gamma)\Lambda_1}{(d+\alpha)(d+\bar{T}_i\beta_1)+d\gamma}, \\ \bar{I} &= \frac{\bar{T}_i\beta_1\Lambda_1}{(d+\alpha)(d+\bar{T}_i\beta_1)+d\gamma}, \\ \bar{T} &= \frac{K_2(r_2-q_0)}{r_2}, \\ \bar{T}_i &= \frac{K_2r_2\beta_2 - q_0(r_2+K_2\beta_2)}{K_2r_2\beta_2}. \end{aligned}$$

   The equilibrium point E_3 exists when the necessary conditions are provided. These conditions are

   - $r_2 > q_0$,
   - $K_2r_2\beta_2 > q_0(r_2+K_2\beta_2)$.

   When we substitute the equilibrium point E_3 in the Jacobian matrix, one of the eigenvalues is found as r_1. Recall that, r_1 represents a growth rate of chickens and

it is a non-negative constant. As one eigenvalue of being positive is a sufficient condition to say the equilibrium point E_3 is an unstable equilibrium point.

5. Equilibrium point $E^* = (N^*, I^*, T^*, T_i^*, K_1)$

   The components of equilibrium point E^* are given;

$$
\begin{aligned}
N^* &= \frac{(d+\alpha+T_i^*\beta_1+\gamma)\Lambda_1}{(d+\alpha)(d+T_i^*\beta_1)+d\gamma}, \\
I^* &= \frac{T_i^*\beta_1\Lambda_1}{(d+\alpha)(d+T_i^*\beta_1)+d\gamma}, \\
T^* &= -\frac{K_2(q_0+K_1q_1-r_2)}{r_2} \\
T_i^* &= -\frac{1}{2r_2\beta_2}\left[q_0(r_2+K_2\beta_2)+K_1(q_1r_2+K_2q_1\beta_2+r_2\beta_3)-K_2r_2\beta_2\right. \\
&\quad -\{(K_1(q_1r_2+K_2q_1\beta_2+r_2\beta_3)+q_0(r_2+K_2\beta_2)-K_2r_2\beta_2)^2 \\
&\quad \left.-4K_1K_2(q_0+K_1q_1-r_2)r_2\beta_2\beta_3\}^{1/2}\right].
\end{aligned}
$$

   This equilibrium point exists if

$$\frac{r_2}{q_0+K_1q_1} > 1.$$

   Note that this condition refers $R_0 > 1$ (see (3.13)). As seen here, the existence of equilibrium point E^* depends on the basic reproduction number $R_0 > 1$. Recall, when $R_0 = 1$, bifurcation might occur. An exchange in stability occurs between equilibrium E_1 and one of the other equilibrium points. Since the existence of E^* depends on $R_0 > 1$, the change in stability is between E_1 and E^*. Investigation of the stability analysis of equilibrium point E^* can be done by substituting this in the Jacobian matrix;

$$
J(E^*) = \begin{pmatrix}
-d & -\alpha & 0 & 0 & 0 \\
T_i^*\beta_1 & -d-\alpha-T_i^*\beta_1-\gamma & 0 & (N^*-I^*)\beta_1 & 0 \\
0 & 0 & r_2-q_0-K_1q_1-\dfrac{2r_2T^*}{K_2} & 0 & -q_1T^* \\
0 & 0 & T_i^*\beta_2+K_1\beta_3 & (T^*-2T_i^*)\beta_2-K_1(q_1+\beta_3)-q_0 & (T^*-T_i^*)\beta_3-q_1T_i^* \\
0 & 0 & 0 & 0 & -r_1
\end{pmatrix}. \tag{3.14}
$$

   We can write matrix (3.14) as a block matrix;

$$
J(E^*) = \left(\begin{array}{cc|ccc}
-d & -\alpha & 0 & 0 & 0 \\
T_i^*\beta_1 & -d-\alpha-T_i^*\beta_1-\gamma & 0 & (N^*-I^*)\beta_1 & 0 \\
\hline
0 & 0 & r_2-q_0-K_1q_1-\dfrac{2r_2T^*}{K_2} & 0 & -q_1T^* \\
0 & 0 & T_i^*\beta_2+K_1\beta_3 & (T^*-2T_i^*)\beta_2-K_1(q_1+\beta_3)-q_0 & (T^*-T_i^*)\beta_3-q_1T_i^* \\
0 & 0 & 0 & 0 & -r_1
\end{array}\right).
$$

We can write

$$J_1(E^*) = \begin{pmatrix} -d & -\alpha \\ T_i^*\beta_1 & -d-\alpha-T_i^*\beta_1-\gamma \end{pmatrix}, \tag{3.15}$$

$$J_2(E^*) = \begin{pmatrix} r_2 - q_0 - K_1 q_1 - \dfrac{2r_2T^*}{K_2} & 0 & -q_1T^* \\ T_i^*\beta_2 + K_1\beta_3 & (T^*-2T_i^*)\beta_2 - K_1(q_1+\beta_3) - q_0 & (T^*-T_i^*)\beta_3 - q_1T_i^* \\ 0 & 0 & -r_1 \end{pmatrix}.$$

First, let us find the sign of eigenvalues of matrix $J_1(E^*)$.

Theorem 3.2.1. *[3] Let $\delta = detA$ and $\tau = traceA$ where A is a $2x2$ matrix and consider the linear system*

$$\dot{x} = Ax. \tag{3.16}$$

a. If $\delta < 0$ (3.16) *has a saddle at the origin.*

b. If $\delta > 0$ an $\tau^2 - 4\delta \geq 0$, (3.16) *has a node at the origin; it is stable if $\tau < 0$ and unstable if $\tau > 0$.*

c. If $\delta > 0$ an $\tau^2 - 4\delta < 0$, and $\tau \neq 0$, (3.16) *has a focus at the origin; it is stable if $\tau < 0$ and unstable if $\tau > 0$.*

d. If $\delta > 0$ and $\tau = 0$, (3.16) *has a center at the origin.*

Note that in case (b), $\tau \geq 4|\delta| > 0$; $\tau \neq 0$.

The proof of the theorem can be found at [3].

Since

$$Trace(J_1) = -2d - \alpha - T_i^*\beta_1 - \gamma < 0$$

and

$$det(J_1) = d^2 + T_i^*\alpha\beta_1 + d(\alpha + T_i^*\beta_1 + \gamma) > 0,$$

both eigenvalues of $J_1(E^*)$ have negative real parts by the theorem (3.2.1). Eigenvalues of $J_2(E^*)$ are

$$\begin{aligned} \lambda_1 &= -r_1, \\ \lambda_2 &= r_2 - q_0 - K_1 q_1 - \frac{2r_2T^*}{K_2}, \\ \lambda_3 &= (T^*-2T_i^*)\beta_2 - q_0 - K_1(q_1+\beta_3). \end{aligned}$$

It is clear that the eigenvalue λ_1 is negative. If we substitute T^* in eigenvalue λ_2, we obtain

$$\lambda_2 = -(q_0 + K_1 q_1)(R_0 - 1).$$

Remember that, the equilibrium point E^* exists,when $R_0 > 1$. So, we can say that the eigenvalue λ_2 is negative. Now, we can investigate sign of last eigenvalue λ_3. We can write λ_3 as

$$\lambda_3 = -\frac{1}{R_0(q_0 + K_1 q_1)} \left[\{(q_0 + K_1 q_1)^2 (4K_1 K_2 (R_0 - 1) R_0 \beta_2 \beta_3 + [K_2 \beta_2 + R_0(q_0 - K_2 \beta_2 + K_1(q_1 + \beta_3))]^2)\}^{1/2} \right].$$

It is clear that the last eigenvalue of $J_2(E^*)$ is negative. As a result, all eigenvalues of matrix $J(E^*)$ are negative. Hence, this equilibrium point is a locally asymptotically stable by the theorem (1.2.1).

Equilibrium points of the system has five non-negative equilibria. E_0, E_1 and E_2 are disease-free equilibrium points and E_3, E^* are endemic equilibrium points. Equilibrium points E_0 and E_1 exists without any conditions, whereas E_2, E_3 and E^* exist when necessary conditions are provided. Also, E_0, E_2 and E_3 are unstable.

R_0 Sensitivity Analysis

To examine the sensitivity of R_0 to each of its parameters, following Arriola and Hyman [25], the normalised forward sensitivity index with respect to each of the parameters are calculated:

$$A_{r_2} = \frac{\frac{\partial R_0}{R_0}}{\frac{\partial r_2}{r_2}} = \frac{r_2}{R_0} \frac{\partial R_0}{\partial r_2} = r_2 \left(\frac{q_0 + K_1 q_1}{r_2} \right) \left(\frac{1}{q_0 + q_1 K_1} \right) = 1, \tag{3.17}$$

$$
\begin{aligned}
A_{q_0} &= \frac{\frac{\partial R_0}{R_0}}{\frac{\partial q_0}{q_0}} = \frac{q_0}{R_0}\frac{\partial R_0}{\partial q_0} = q_0\left(\frac{q_0+K_1q_1}{r_2}\right)\left(-\frac{r_2}{(q_0+K_1q_1)^2}\right) \\
&= -\frac{q_0}{q_0+K_1q_1} < 0, \\
A_{K_1} &= \frac{\frac{\partial R_0}{R_0}}{\frac{\partial K_1}{K_1}} = \frac{K_1}{R_0}\frac{\partial R_0}{\partial K_1} = K_1\left(\frac{q_0+K_1q_1}{r_2}\right)\left(-\frac{r_2q_1}{(q_0+K_1q_1)^2}\right) \\
&= -\frac{K_1q_1}{q_0+K_1q_1} < 0, \\
A_{q_1} &= \frac{\frac{\partial R_0}{R_0}}{\frac{\partial q_1}{q_1}} = \frac{q_1}{R_0}\frac{\partial R_0}{\partial q_1} = q_1\left(\frac{q_0+K_1q_1}{r_2}\right)\left(-\frac{K_1r_2}{(q_0+K_1q_1)^2}\right) \\
&= -\frac{K_1q_1}{q_0+K_1q_1} < 0.
\end{aligned}
$$

It is seen here, R_0 is most sensitive to changes in r_2. An increase or decrease in r_2 will bring about increase or decrease of the same proportion in basic reproduction number R_0. One can also see that q_0, K_1 and q_1 have an inversely proportional relationship with R_0; an increase in any of these parameters will bring about a decrease in R_0, however, the size of decrease will be proportionally smaller. In other words, if the precautions are taken in the breeding time of ticks, the increase of infective ticks may be prevented. Consequently, the spread of the disease can decrease.

Bifurcation Analysis

In this model, we shall examine bifurcation analysis. It is known that, bifurcation occurs when $R_0 = 1$. We use the Castillo-Chavez and Song bifurcation theorem (2.2.1) which is given in the second chapter. Let us recall,

$$
\begin{aligned}
a &= \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \\
b &= \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0)
\end{aligned}
\tag{3.18}
$$

from the theorem. We set $S = x_1$, $I = x_2$, $T_s = x_3$, $T_i = x_4$ and $B = x_5$. Therefore $N = x_1 + x_2$ and $T = x_3 + x_4$. System (3.6)-(3.10) becomes

$$f_1 = \Lambda_1 - d(x_1 + x_2) - \alpha x_2, \tag{3.19}$$

$$f_2 = \beta_1 x_1 x_4 - (\gamma + d + \alpha)x_2, \tag{3.20}$$

$$f_3 = r_2(x_3 + x_4)(1 - \frac{x_3 + x_4}{K_2}) - (q_0 + q_1 x_5)(x_3 + x_4), \tag{3.21}$$

$$f_4 = \beta_2 x_3 x_4 + \beta_3 x_3 x_5 - (q_0 + q_1 x_5)x_4, \tag{3.22}$$

$$f_5 = r_1 x_5(1 - \frac{x_5}{K_1}). \tag{3.23}$$

Let us consider the parameter ϕ which is given in theorem (2.2.1) is represented by r_2. Here r_2 is the obvious choice of bifurcation parameter because basic reproduction number R_0 is more sensitive to changes in r_2 as seen in the equation (3.17). When $R_0 = 1$, we can write

$$\tilde{r_2} = q_0 + K_1 q_1.$$

So, the disease free equilibrium point E_1 of this model is $(\tilde{x_1} = \frac{\Lambda_1}{d}, \tilde{x_2} = 0, \tilde{x_3} = 0, \tilde{x_4} = 0, \tilde{x_5} = K_1)$. The Jacobian matrix for the disease free equilibrium point is evaluated

$$J = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ 0 & -d-\alpha-\gamma & 0 & \frac{\beta_1 \Lambda_1}{d} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & K_1\beta_3 & -q_0 - K_1(q_1 + \beta_3) & 0 \\ 0 & 0 & 0 & 0 & -r_1 \end{pmatrix}.$$

Eigenvalues of this matrix are

$$\lambda_1 = 0,$$

$$\lambda_2 = -d,$$

$$\lambda_3 = -q_0 - K_1(q_1 + \beta_3),$$

$$\lambda_4 = -r_1,$$

$$\lambda_5 = -d - \alpha - \gamma.$$

It is clear that λ_1 is a simple eigenvalue of $D_x f$. So we can write right eigenvector and left eigenvector according to the theorem (2.2.1).

The right eigenvector w corresponding to the zero eigenvalue is

$$w = (-\frac{\alpha\beta_1\Lambda_1}{d^2(d+\alpha+\gamma)}, \frac{\beta_1\Lambda_1}{d(d+\alpha+\gamma)}, \frac{q_0 + K_1(q_1+\beta_3)}{K_1\beta_3}, 1, 0)^T$$

and the left eigenvector v corresponding to the zero eigenvalue is

$$v = (0,0,1,0,0).$$

The second derivatives in formulas (3.18) are evaluated at disease free equilibrium point $E_0 = (\frac{\Lambda_1}{d},0,0,0,K_1)$, and $r_2 = \tilde{r_2}$. The non-zero derivatives are given as follows,

$$\begin{aligned}
&\frac{\partial^2 f_2}{\partial x_1 \partial x_4} = \frac{\partial^2 f_2}{\partial x_4 \partial x_1} = \beta_1, && \frac{\partial^2 f_3}{\partial x_3 \partial x_4} = \frac{\partial^2 f_3}{\partial x_4 \partial x_3} = -2\frac{r_2}{K_2},\\
&\frac{\partial^2 f_3}{\partial x_3 \partial x_5} = \frac{\partial^2 f_3}{\partial x_5 \partial x_3} = -q_1, && \frac{\partial^2 f_3}{\partial x_4 \partial x_5} = \frac{\partial^2 f_3}{\partial x_5 \partial x_4} = -q_1,\\
&\frac{\partial^2 f_3}{\partial {x_3}^2} = -2\frac{r_2}{K_2}, && \frac{\partial^2 f_3}{\partial {x_4}^2} = -2\frac{r_2}{K_2},\\
&\frac{\partial^2 f_4}{\partial x_3 \partial x_4} = \frac{\partial^2 f_4}{\partial x_3 \partial x_3} = \beta_2, && \frac{\partial^2 f_4}{\partial x_3 \partial x_5} = \frac{\partial^2 f_4}{\partial x_5 \partial x_3} = \beta_3,\\
&\frac{\partial^2 f_4}{\partial x_4 \partial x_5} = \frac{\partial^2 f_4}{\partial x_5 \partial x_4} = -q_1, && \frac{\partial^2 f_4}{\partial x_3 \partial {x_5}^2} = -2\frac{r_1}{K_1},\\
&\frac{\partial^2 f_3}{\partial x_3 \partial r_2} = 1, && \frac{\partial^2 f_3}{\partial x_4 \partial r_2} = 1.
\end{aligned}$$

If we substitute these derivatives in a and b given by (3.18),

$$\begin{aligned}
a &= v_2 w_1 w_4 \frac{\partial^2 f_2}{\partial x_1 \partial x_4} + v_2 w_4 w_1 \frac{\partial^2 f_2}{\partial x_4 \partial x_1} + 2v_3 {w_3}^2 \frac{\partial^2 f_3}{\partial {x_3}^2} + v_3 w_3 w_4 \frac{\partial^2 f_3}{\partial x_3 \partial x_4}\\
&+ v_3 w_4 w_3 \frac{\partial^2 f_3}{\partial x_4 \partial x_3} + v_3 w_3 w_5 \frac{\partial^2 f_3}{\partial x_3 \partial x_5} + 2v_3 {w_4}^2 \frac{\partial^2 f_3}{\partial {x_4}^2} + v_3 w_5 w_3 \frac{\partial^2 f_3}{\partial x_5 \partial x_3}\\
&+ v_3 w_4 w_5 \frac{\partial^2 f_3}{\partial x_4 \partial x_5} + v_3 w_5 w_4 \frac{\partial^2 f_3}{\partial x_5 \partial x_4} + v_4 w_3 w_4 \frac{\partial^2 f_4}{\partial x_3 \partial x_4} + v_4 w_4 w_3 \frac{\partial^2 f_4}{\partial x_4 \partial x_3}\\
&+ v_4 w_3 w_5 \frac{\partial^2 f_4}{\partial x_3 \partial x_5} + v_4 w_5 w_3 \frac{\partial^2 f_4}{\partial x_5 \partial x_3} + v_4 w_4 w_5 \frac{\partial^2 f_4}{\partial x_4 \partial x_5} + v_4 w_5 w_4 \frac{\partial^2 f_4}{\partial x_5 \partial x_4}\\
&+ 2v_5 w_5 w_5 \frac{\partial^2 f_5}{\partial x_5 \partial x_5}\\
&= \frac{4r_2}{{K_1}^2 K_2 {\beta_3}^2}\left[-(q_0 + K_1 q_1)^2 - 3K_1(q_0 + K_1 q_1)\beta_3 - 3{K_1}^2 {\beta_3}^2\right],\\
b &= v_3 w_3 \frac{\partial^2 f_3}{\partial x_3 \partial r_2} + v_3 w_4 \frac{\partial^2 f_3}{\partial x_4 \partial r_2} = 2 + \frac{q_0 + K_1 q_1}{K_1 \beta_3}
\end{aligned}$$

are obtained. It is obvious that $a < 0$ and $b > 0$. This shows, according to the result of *iv.* in the theorem (2.2.1), the stability of the equilibrium point E_1 is changed from stable to unstable.

When $R_0 < 1$, equilibrium point E_1 is stable and components of the equilibrium point E^* are negative. It is mean, when $R_0 < 1$, the equilibrium point E^* does not exist.

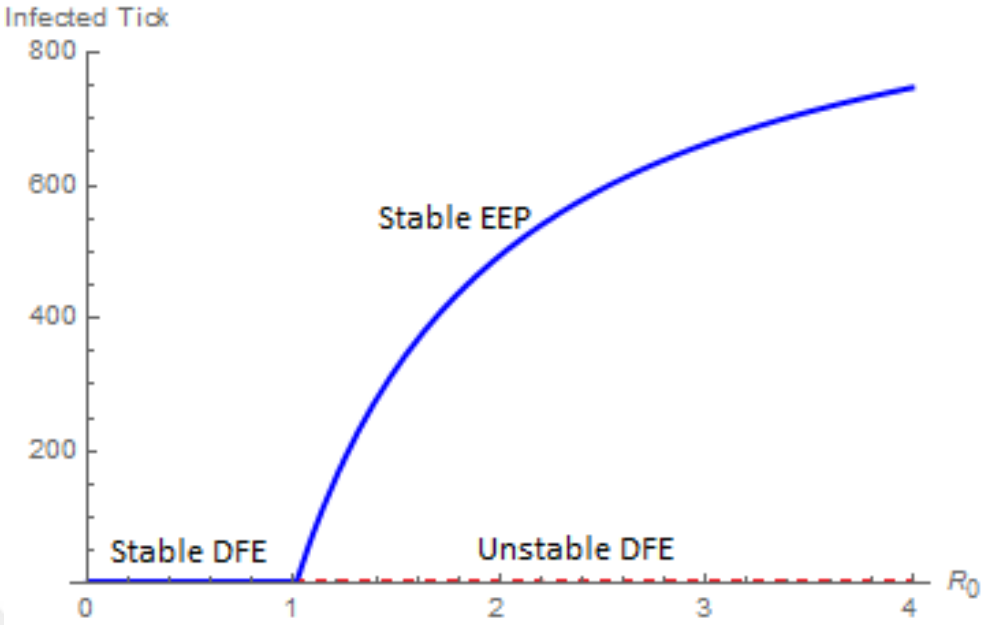

Figure 3.2 : Bifurcation diagram of the Infected Tick Population. Where $K_2 = 1000$, $q_0 = 0.0025$, $q_1 = 0.0063$, $\beta_3 = 0.004$, $K_1 = 2$, $\beta_2 = 0.49$.

Therefore, when $R_0 < 1$, there only exists the disease-free equilibrium point. When $R_0 > 1$, the disease-free equilibrium point becomes unstable, endemic equilibrium point E^* becomes positive and stable. As a result, we can say that forward bifurcation occurs.

Simulations

Here, to better understand the stability analysis of the system we simulate the model. Let us fix the following parameters and total human population in the system (3.6) - (3.10) as follows:

$$N = 5000, \quad r_1 = 0.2, \quad r_2 = 0.75, \quad K_2 = 300,$$
$$q_0 = 0.025, \quad \beta_2 = 0.0023, \quad \beta_3 = 0.0015, \quad \beta_1 = 0.008,$$
$$\alpha = 0.0018, \quad d = 0.011, \quad \gamma = 0.017.$$

We choose the initial conditions as,

$$B(0) = 2, \quad T(0) = 100, \quad T_i(0) = 0, \quad I(0) = 0.$$

Here we shall consider various cases by choosing the rest of the parameters differently.

1. *Case 1*

   If we choose $K_1 = 20$ and $q_1 = 0.063$, then $R_0 = 0.583 < 1$. And if we choose

the parameter $K_1 = 20$ and $q_1 = 0.073$, then $R_0 = 0.505 < 1$. Note that because $R_0 < 1$ the stability will point out that the disease vanish. When the parameters are $K_1 = 20$ and $r_1 = 0.2$, the graphic of the chicken population over time is given in figure (3.13).

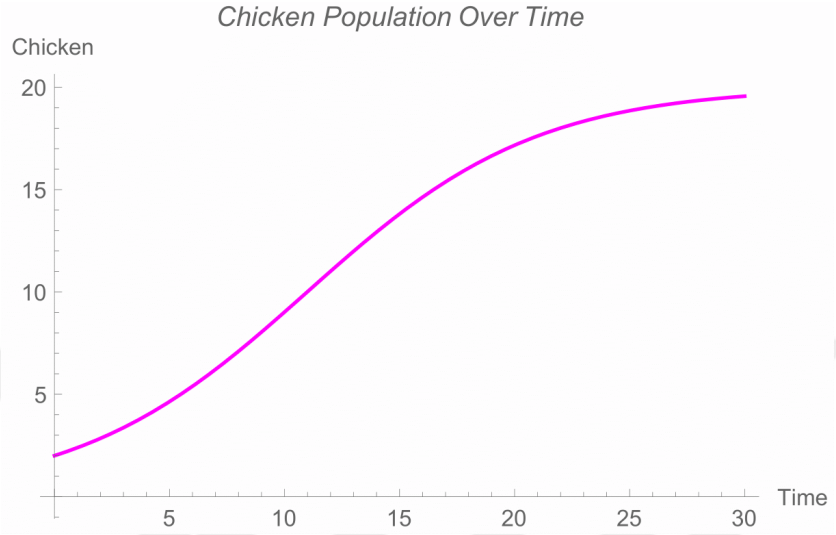

Figure 3.3 : Chicken Population Over Time. We choose $K_1 = 20$, so that $R_0 < 1$.

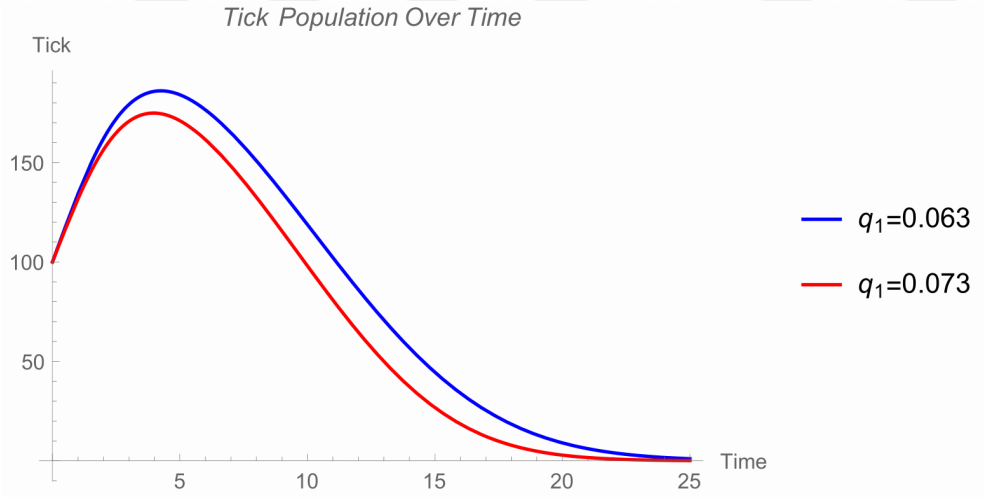

Figure 3.4 : Tick Population Over Time.

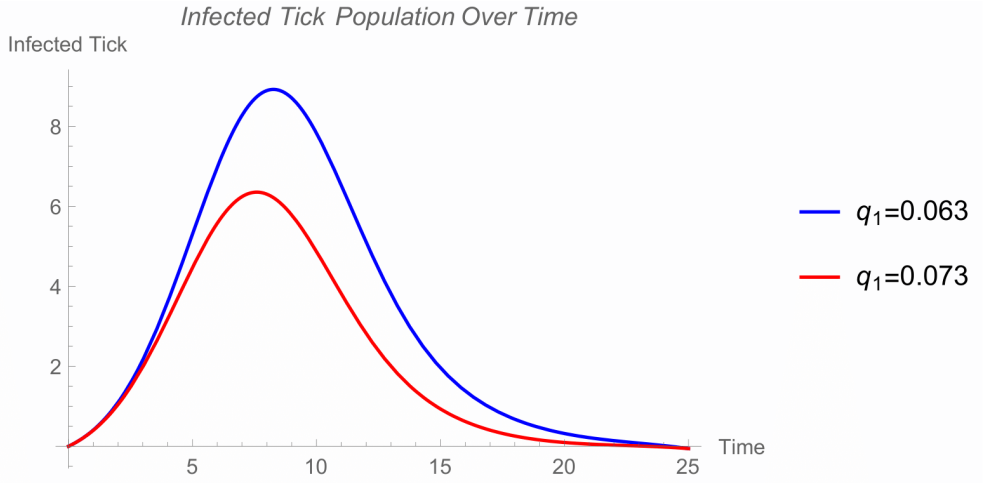

Figure 3.5 : Infected Tick Population Over Time.

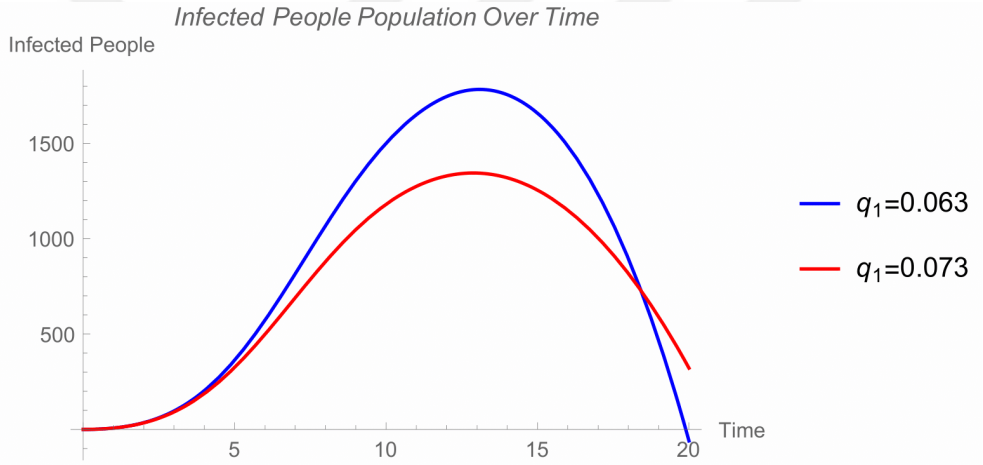

Figure 3.6 : Infected People Population Over Time.

Figures (3.4), (3.5) and (3.6) show that the tick population, infected tick population decrease and eventually vanish, which means the disease eventually dies out. Recall that, q_1 is chicken-related death rate of ticks. According to figures (3.5) and (3.6) an increase in q_1 causes the tick population and infected tick population increase less and vanish faster. Therefore, we can say that if we increase the frequency of the unleashed chickens in the environment, chicken related-death rate q_1 of ticks also increases. Thus, the tick population vanish rapidly.

2. *Case 2*

   If we choose parameter $K_1 = 5$ and $q_1 = 0.063$, then $R_0 = 2.205$. And if we choose $q_1 = 0.073$, then $R_0 = 1.923$. Which means as $R_0 > 1$, the equilibrium points are unstable. When the parameters are $K_1 = 5$ and $r_1 = 0.2$, the graphic of the chicken population over time is given in figure (3.7).

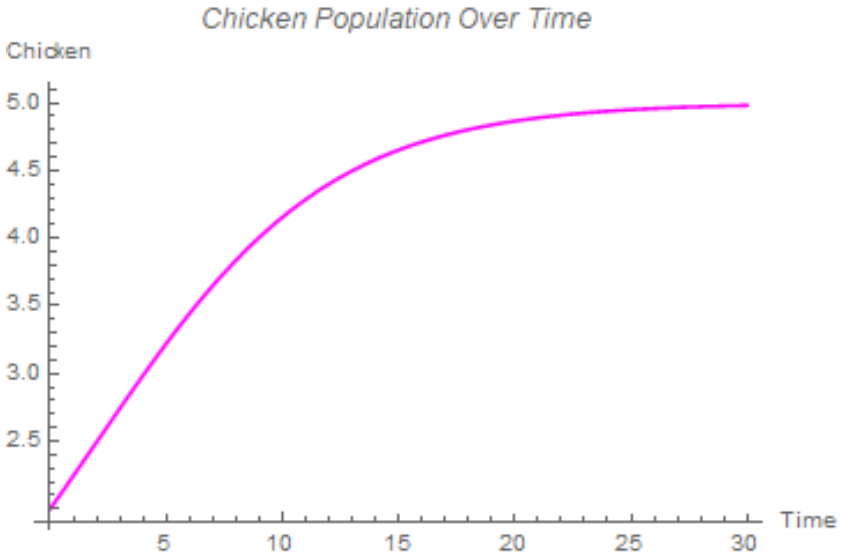

Figure 3.7 : Chicken Population Over Time. We choose $K_1 = 5$, so that $R_0 > 1$.

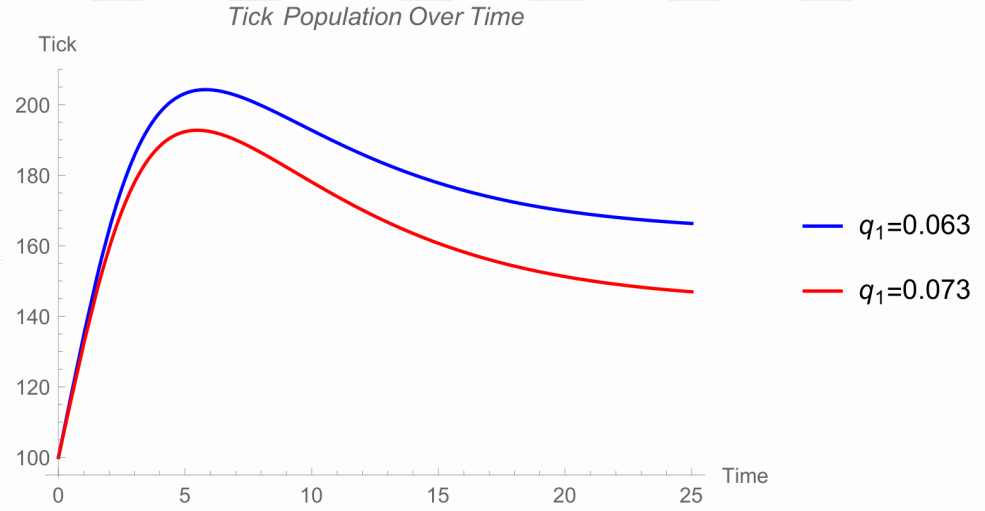

Figure 3.8 : Tick Population Over Time.

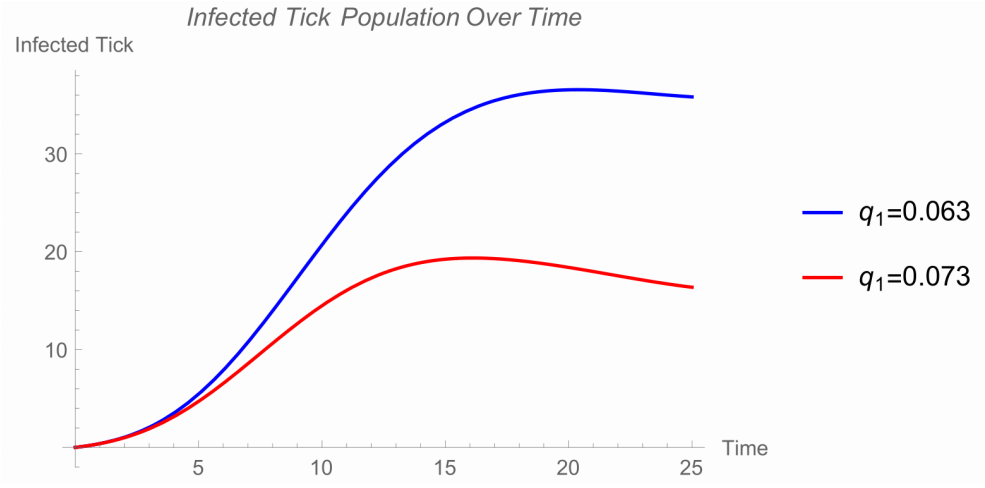

Figure 3.9 : Infected Tick Population Over Time.

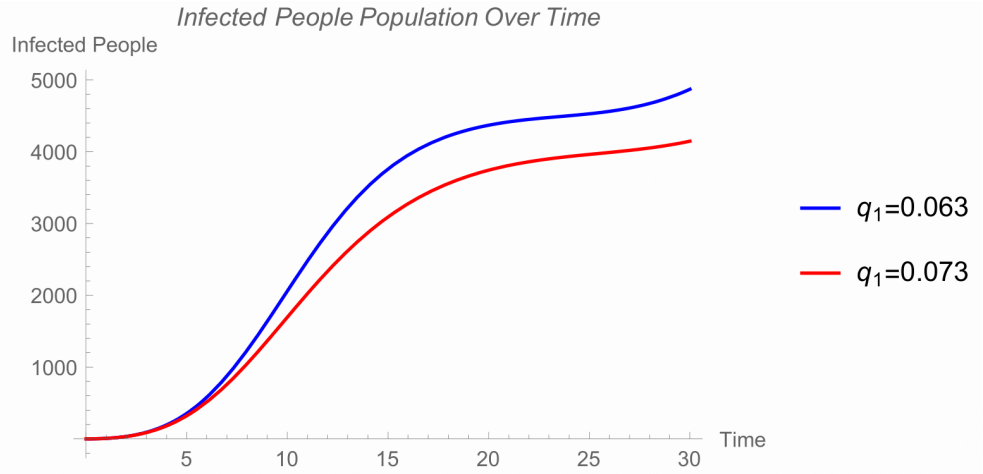

Figure 3.10 : Infected People Population Over Time.

As we can see in the figures (3.8) and (3.9) first, the tick population increases for a while, then it slightly decreases and eventually remains constant. Besides, according to figure (3.10), first the spread of the disease increases due to the increase of tick population. After, it decreases for a while due to the chicken-related death rate q_1. And eventually, the disease spreads and becomes endemic.

3.2.2 Tick constant - chicken logistic growth model

In this subsection, we will consider the growth of only chicken population as logistic growth whereas we take the number of the ticks entering to the system as constant.

The diagram of that we call tick constant - chicken logistic growth model is shown in the figure 3.11.

The bidirectional dotted arrows between the boxes represent the interaction between the classes.

So let us write the system of non-linear differential equation;

$$\frac{dS}{dt} = \Lambda_1 + \gamma I - \beta_1 S T_i - dS, \tag{3.24}$$

$$\frac{dI}{dt} = \beta_1 S T_i - (\alpha + d) I - \gamma I, \tag{3.25}$$

$$\frac{dT_s}{dt} = \Lambda_2 - \beta_2 T_s T_i - \beta_3 T_s B - (q_0 + q_1 B) T_s, \tag{3.26}$$

$$\frac{dT_i}{dt} = \beta_2 T_s T_i + \beta_3 T_s B - (q_0 + q_1 B) T_i, \tag{3.27}$$

$$\frac{dB}{dt} = r_1 B (1 - \frac{B}{K_1}). \tag{3.28}$$

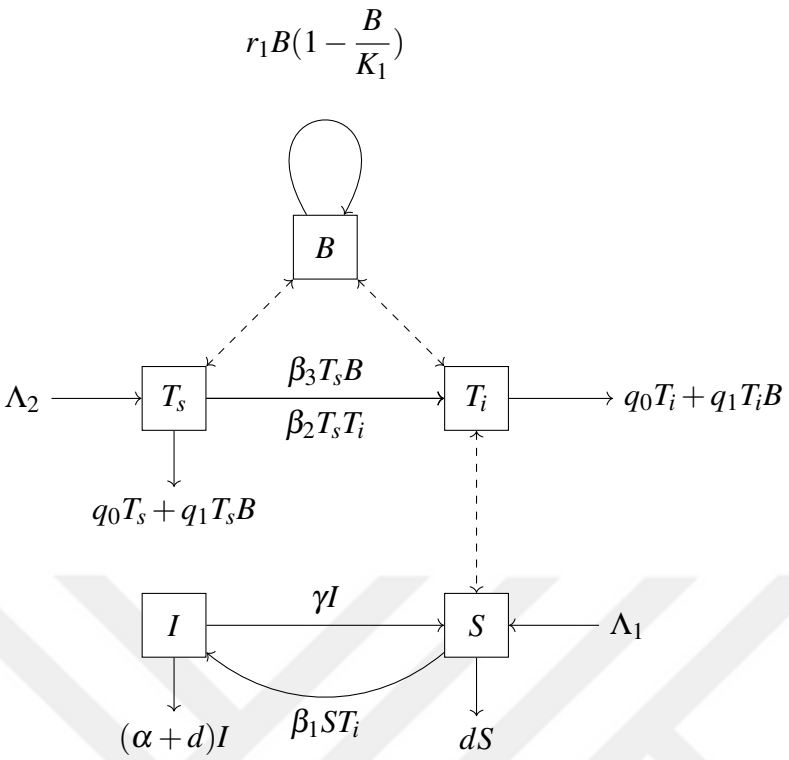

Figure 3.11 : Tick Constant - Chicken Logistic Growth Model Chart.

The variation of the susceptible human population, S: Equation (3.24) represents the susceptible human population dynamics. The increments of the susceptible population are shown by Λ_1 and γI. Here, Λ_1 represents the number of individuals entering the environment. γ represents the recovery rate of infected individuals. Terms that cause the decrements of the susceptible human population are shown by $\beta_1 ST_i$ and dS. β_1 represents the transmission rate of disease between susceptible and infected individuals, and d represents the natural death rate.

The variation of the infected human population, I : Equation (3.25) represents the dynamics of the infected human population. The source term of infected people compartment is the term $\beta_1 ST_i$. Individuals leave the compartment of infected people either with the healing condition which is represented by γI, or leave with disease-related death or natural death which are represented by αI and dI, respectively.

The variation of the susceptible tick population, T_s: Equation (3.26) represents the dynamics of the susceptible tick population. We write the source term of the susceptible tick population with Λ_2, where Λ_2 represents the number of ticks enter the susceptible tick compartment. Terms that cause decrements of susceptible tick population are represented by $\beta_3 T_sB$, $\beta_2 T_sT_i$, q_0T_s and q_1T_sB.

The variation of the infected tick population, T_i: Equation (3.27) represents the dynamics of the infected tick compartment. The source terms of the infected tick population are given by $\beta_3 T_s B$ and $\beta_2 T_s T_i$. Terms that cause the decrements of infected tick population are similar to the susceptible tick population.

The variation of the chicken population, B: Eq. (3.28) represents the dynamic of the chicken population. We assume, the source term of the chicken population is logistic growth equation where r_1 represents growth rate of chickens and carrying capacity of chickens is represented by K_1.

Since $S+I=N$ and $T_s+T_i=T$, to analyse more easily, we can write the following equations:

$$\frac{dN}{dt} = \Lambda_1 - dN - \alpha I, \tag{3.29}$$

$$\frac{dI}{dt} = \beta_1(N-I)T_i - (\alpha+d)I - \gamma I, \tag{3.30}$$

$$\frac{dT}{dt} = \Lambda_2 - (q_0+q_1B)T, \tag{3.31}$$

$$\frac{dT_i}{dt} = \beta_2(T-T_i)T_i + \beta_3(T-T_i)B - (q_0+q_1B)T_i, \tag{3.32}$$

$$\frac{dB}{dt} = r_1 B(1-\frac{B}{K_1}). \tag{3.33}$$

All parameters in this system are non-negative constants. Since this system of equations is a non-linear, we use Hartman Grobman theorem given in theorem (1.2.2). We should linearise it so that we can perform stability analysis. As in the previous model examples, we must substitute the equilibrium points of the system (3.29)-(3.33) in the Jacobian matrix. Then we can talk about the behaviour of the flow locally, near the equilibrium points according to the sign of the eigenvalues by applying the Hartman Grobman theorem. Equilibrium points of the system is given in the table (3.2).

Equilibrium Points / (N,I,T,T_i,B)	**Description**
$E_0 = (\frac{\Lambda_1}{d}, 0, \frac{\Lambda_2}{q_0}, 0, 0)$	Disease free equilibrium point.
$E_1 = (\bar{N}, \bar{I}, \bar{T}, \bar{T}_i, 0)$	Endemic equilibrium point which exist when $\beta_2\Lambda_2 > {q_0}^2$.
$E^* = (N^*, I^*, T^*, T_i^*, K_1)$	Endemic Equilibrium point.

Table 3.2 : Equilibrium points of the Tick Constant - Chicken Logistic Growth Model Table.

Jacobian matrix can be written as follows,

$$J = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ T_i\beta_1 & -d-\alpha-T_i\beta_1-\gamma & 0 & (-I+N)\beta_1 & 0 \\ 0 & 0 & -q_0-Bq_1 & 0 & -q_1T \\ 0 & 0 & T_i\beta_2+B\beta_3 & (T-T_i)\beta_2-q_0-Bq_1-T_i\beta_2-B\beta_3 & (T-T_i)\beta_3-q_1T_i \\ 0 & 0 & 0 & 0 & r_1-\frac{2Br_1}{K_1} \end{pmatrix}.$$

Let us investigate the stability of each equilibrium points.

1. Equilibrium Point $E_0 = (\frac{\Lambda_1}{d}, 0, \frac{\Lambda_2}{q_0}, 0, 0)$

   This equilibrium point contains only human and susceptible tick populations $N = \frac{\Lambda_1}{d}$ and $T = \frac{\Lambda_2}{q_0}$ respectively.

   Then let us do the stability analysis by substituting the disease-free equilibrium point E_0 in the Jacobian matrix,

$$J(E_0) = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ 0 & -d-\alpha-\gamma & 0 & \frac{\beta_1\Lambda_1}{d} & 0 \\ 0 & 0 & -q_0 & 0 & -\frac{q_1\Lambda_2}{q_0} \\ 0 & 0 & 0 & -q_0+\frac{\beta_2\Lambda_2}{q_0} & \frac{\beta_3\Lambda_2}{q_0} \\ 0 & 0 & 0 & 0 & r \end{pmatrix}.$$

   Eigenvalues of this matrix are

$$\begin{aligned} \lambda_1 &= -d, \\ \lambda_2 &= -q_0, \\ \lambda_3 &= r, \\ \lambda_4 &= -d-\alpha-\gamma, \\ \lambda_5 &= -\frac{{q_0}^2-\beta_2\Lambda_2}{q_0} \end{aligned}$$

   It is clear that the eigenvalues $\lambda_1, \lambda_2, \lambda_3$ are non positive and the eigenvalue λ_3 is grater than zero. Therefore, we can say that E_0 equilibrium point is an unstable equilibrium point.

2. Equilibrium Point $E_1 = (\bar{N}, \bar{I}, \bar{T}, \bar{T}_i, 0)$

   Components of the equilibrium point E_1 are given;

$$\bar{N} = \frac{q_0(q_0\beta_1 - \beta_2(d+\alpha+\gamma))\Lambda_1 - \beta_1\beta_2\Lambda_1\Lambda_2}{q_0(d+\alpha)(q_0\beta_1 - d\beta_2) - dq_0\beta_2\gamma - (d+\alpha)\beta_1\beta_2\Lambda_2},$$
$$\bar{I} = \frac{\beta_1\Lambda_1(\beta_2\Lambda_2) - {q_0}^2}{q_0(d+\alpha)(q_0\beta_1 - d\beta_2) - dq_0\beta_2\gamma - (d+\alpha)\beta_1\beta_2\Lambda_2},$$
$$\bar{T} = \frac{\Lambda_2}{q_0},$$
$$\bar{T}_i = \frac{\Lambda_2}{q_0} - \frac{q_0}{\beta_2}.$$

   There are some conditions for the existence of the equilibrium point E_1. If these conditions for existence of equilibrium point E_1 are satisfied, one of the eigenvalues which are obtained by substituting in the Jacobian matrix is found positive. Therefore, this equilibrium point is also unstable.

3. Equilibrium Point $E^* = (N^*, I^*, T^*, T_i^*, K_1)$

   Components of the equilibrium point E^* are given;

$$N^* = \frac{(d+\alpha+T_i^*\beta_1+\gamma)\Lambda_1}{(d+\alpha)(d+T_i^*\beta_1)d\gamma},$$
$$I^* = \frac{T_i^*\beta_1\Lambda_1}{(d+\alpha)(d+T_i^*\beta_1)+d\gamma},$$
$$T^* = \frac{\Lambda_2}{q_0 +_1 Kq_1},$$
$$T_i^* = -\frac{1}{2(q_0+K_1q_1)\beta_2}\left[(q_0+K_1q_1)(q_0K_1(q_1+\beta_3)) - \beta_2\Lambda_2\right.$$
$$\left. -\sqrt{(q_0Kq_1)^2(q_0+K_1(q_1+\beta_3))^2 - 2(q_0+K_1q_1)\beta_2(q_0+K_1(q_1-\beta_3))\Lambda_2 + {\beta_2}^2{\Lambda_2}^2}\right].$$

   If we select all the parameters for the components of the equilibrium point E^* as non-negative, we see that T_i^* is positive. N^* and I^* contain T_i^*. Therefore, I^* and T_i^* are also positive. Besides, it is clear that T^* is positive. Let us substitute the equilibrium point in the Jacobian matrix;

$$J(E^*) = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ T_i^*\beta_1 & -d-\alpha-T_i^*\beta_1-\gamma & 0 & (-I^*+N^*)\beta_1 & 0 \\ 0 & 0 & -q_0-K_1q_1 & 0 & -q_1T^* \\ 0 & 0 & T_i^*\beta_2+K_1\beta_3 & (T^*-2T_i^*)\beta_2-K_1(q_1+\beta_3)-q_0 & T^*\beta_3-T_i^*(q_1+\beta_3) \\ 0 & 0 & 0 & 0 & -r_1 \end{pmatrix}.$$

   The matrix $J(E^*)$ is can be written as block matrix,

$$J(E^*) = \left(\begin{array}{cc|ccc} -d & -\alpha & 0 & 0 & 0 \\ T_i^*\beta_1 & -d-\alpha-T_i^*\beta_1-\gamma & 0 & (N^*-I^*)\beta_1 & 0 \\ \hline 0 & 0 & -q_0-K_1q_1 & 0 & -q_1T^* \\ 0 & 0 & T_i^*\beta_2+K_1\beta_3 & (T^*-2T_i^*)\beta_2-K_1(q_1+\beta_3)-q_0 & T^*\beta_3-T_i^*(q_1+\beta_3) \\ 0 & 0 & 0 & 0 & -r_1 \end{array}\right).$$

We can write

$$J_1(E^*) = \begin{pmatrix} -d & -\alpha \\ T_i^*\beta_1 & -d-\alpha-T_i^*\beta_1-\gamma \end{pmatrix},$$

$$J_2(E^*) = \begin{pmatrix} -q_0K_1q_1 & 0 & -q_1T^* \\ T_i^*\beta_2+K_1\beta_3 & (T^*-2T_i^*)\beta_2-K_1(q_1+\beta_3)-q_0 & T^*\beta_3-T_i^*(q_1+\beta_3) \\ 0 & 0 & -r_1 \end{pmatrix}.$$

Since

$$Trace(J_1(E_1)) = -2d-\alpha-T_i^*\beta_1-\gamma < 0$$

and

$$det(J_1(E_1)) = d^2+T_i^*\alpha\beta_1+d(\alpha+T_i^*\beta_1+\gamma) > 0,$$

according to the theorem (3.2.1) both eigenvalues of the matrix $J_1(E^*)$ have negative real parts. Let's write the characteristic polynomial of $J_2(E^*)$

$$P(\lambda) = (\lambda+q_0+K_1q_1)(\lambda+r_1)(\lambda+q_0+K_1(q_1+\beta_3)+\beta_2(2T_i^*-T^*)).$$

Eigenvalues of this matrix are

$$\begin{aligned} \lambda_1 &= -q_0-K_1q_1, \\ \lambda_2 &= -r_1, \\ \lambda_3 &= -q_0-K_1(q_1+\beta_3)-\beta_2(2T_i^*-T^*). \end{aligned}$$

It is clear that the eigenvalues λ_1 and λ_2 are negative. If $2T_i^* > T^*$ is provided, all eigenvalues are negative. Then, the equilibrium point E^* is called stable equilibrium point.

Otherwise, if $2T_i^* < T^*$ and $q_0+K_1(q_1+\beta_3) > \beta_2(2T_i^*-T^*)$, all eigenvalues will be negative again and equilibrium point E^* is called stable. And if $2T_i^* < T^*$ and $q_0+K_1(q_1+\beta_3) < \beta_2(2T_i^*-T^*)$, equilibrium point is a unstable.

3.2.3 Tick logistic growth - chicken constant model

Here we consider the problem within the framework of the numbers of entering to the ticks and chicken populations as constants.

The diagram of the tick logistic growth - chicken constant model is shown in the figure (3.12)

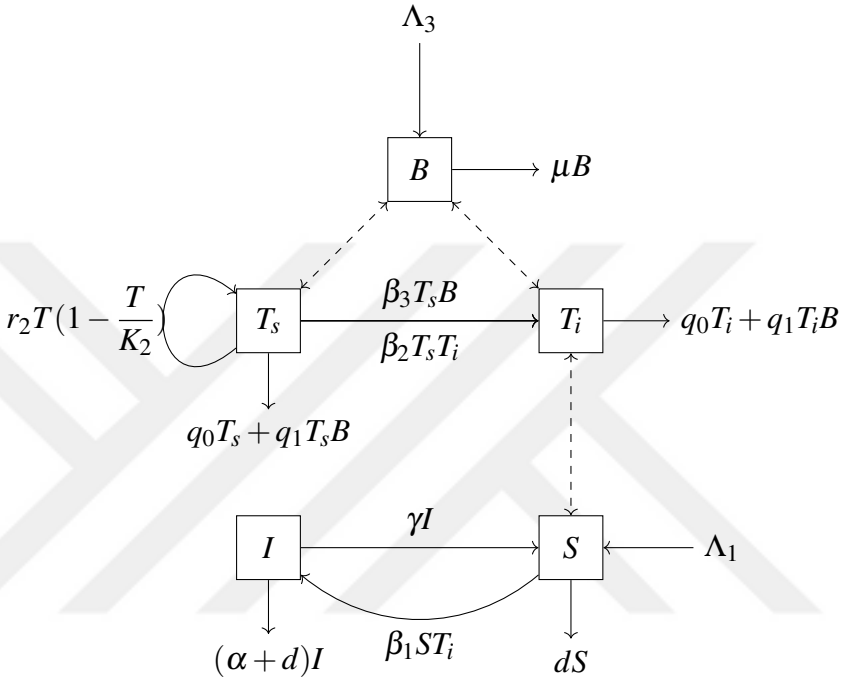

Figure 3.12 : Tick Logistic Growth - Chicken Constant Model Chart.

Let us write the system of non-linear equation according to figure 3.12;

$$\frac{dS}{dt} = \Lambda_1 + \gamma I - \beta_1 S T_i - dS, \tag{3.34}$$

$$\frac{dI}{dt} = \beta_1 S T_i - (\alpha + d + \gamma) I, \tag{3.35}$$

$$\frac{dT_s}{dt} = r_2 T(1 - \frac{T}{K_2}) - \beta_2 T_s T_i - \beta_3 T_s B - (q_0 + q_1 B) T_s, \tag{3.36}$$

$$\frac{dT_i}{dt} = \beta_2 T_s T_i + \beta_3 T_s B - (q_0 + q_1 B) T_i, \tag{3.37}$$

$$\frac{dB}{dt} = \Lambda_3 - \mu B. \tag{3.38}$$

The variation of the susceptible human population, S: Equation (3.34) represents the susceptible human population dynamics. The increments of the susceptible population are shown by Λ_1 and γI. Here, Λ_1 represents the number of individuals entering the environment. γ represents the recovery rate of infected individuals. Terms that

cause decrements of the susceptible human population are shown by $\beta_1 ST_i$ and dS. β_1 represents the transmission rate of disease between susceptible and infected individuals and d represents the natural death rate.

The variation of the infected human population, I : Equation (3.35) represents the dynamics of the infected human population. The source term of infected people compartment is the term $\beta_1 ST_i$. Individuals leave the compartment of infected people either with the healing condition which is represented by γI, or leave with disease-related death or natural death which are represented by αI and dI, respectively.

The variation of the susceptible tick population, T_s: Equation (3.36) represents the dynamics of the susceptible tick population. In this model, we write the source term of the susceptible tick population with the logistic equation because the tick population varies depending on the temperature of the environment, the season and the number of nutrients in the environment. The parameters growth rate and carrying capacity are represented by r_2 and K_2, respectively in this logistic equation. Terms that cause decrements of susceptible tick population are represented by $\beta_3 T_s B$, $\beta_2 T_s T_i$, $q_0 T_s$ and $q_1 T_s B$ where the parameter β_3 is the transmission rate between ticks and chickens. Transmission rate between ticks is denoted by β_2.

The variation of the infected tick population, T_i: Equation (3.37) represents the dynamics of the infected tick compartment. The source terms of the infected tick population are given by $\beta_3 T_s B$ and $\beta_2 T_s T_i$. Terms that cause decrements of infected tick population are similar to the susceptible tick population.

The variation of the chicken population, B: Eq.(3.38) represents the dynamics of the chicken population. In the model, we represent the source term of the chicken population with a constant term Λ_3. Loss due to death of the chicken population is represented by the term μB.

Since $S+I=N$ and $T_s+T_i=T$, to analyse more easily, we can write the following equations (3.34)-(3.38)

$$\frac{dN}{dt} = \Lambda_1 - dN - \alpha I, \tag{3.39}$$
$$\frac{dI}{dt} = \beta_1(N-I)T_i - (\alpha+d+\gamma)I, \tag{3.40}$$
$$\frac{dT}{dt} = r_2T(1-\frac{T}{K_2}) - (q_0+q_1B)T, \tag{3.41}$$
$$\frac{dT_i}{dt} = \beta_2(T-T_i)T_i + \beta_3(T-T_i)B - (q_0+q_1B)T_i, \tag{3.42}$$
$$\frac{dB}{dt} = \Lambda_3 - \mu B. \tag{3.43}$$

All parameters in this non-linear system of equations are non-negative constants. Since, this system of equations is a non-linear system, we will again use the Hartman Grobman theorem and to use the theorem similarly what have been done in the previous cases, we examine the behaviours of the linearised system in the neighbourhood of the equilibrium points. so that we shall investigate the local stability of the problem. Equilibrium points of the system are found by solving the following equations:

$$\frac{dN}{dt} = 0 \Rightarrow \Lambda_1 - dN - \alpha I = 0,$$
$$\frac{dI}{dt} = 0 \Rightarrow \beta_1(N-I)T_i - (\alpha+d+\gamma)I = 0,$$
$$\frac{dT}{dt} = 0 \Rightarrow r_2T(1-\frac{T}{K_2}) - (q_0+q_1B)T = 0,$$
$$\frac{dT_i}{dt} = 0 \Rightarrow \beta_2(T-T_i)T_i + \beta_3(T-T_i)B - (q_0+q_1B)T_i = 0,$$
$$\frac{dB}{dt} = 0 \Rightarrow \Lambda_3 - \mu B = 0.$$

Equilibrium Points / (N,I,T,T_i,B)	**Description**
$E_0 = (\frac{\Lambda_1}{d},0,0,0,\frac{\Lambda_3}{\mu})$	Disease free equilibrium point.
$E^* = (N^*,I^*,T^*,T_i^*,B^*)$	Endemic Equilibrium point.

Table 3.3 : Equilibrium Points of Tick Logistic Growth - Chicken Constant Model.

Equilibrium points are given in the Table (3.3).

Stability Analysis

Let us write the Jacobian matrix,

$$J = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ T_i\beta_1 & -d-\alpha-T_i\beta_1-\gamma & 0 & (N-I)\beta_1 & 0 \\ 0 & 0 & r_2 - Bq_1 - q_0 - 2\frac{r_2 T}{K_2} & 0 & -q_1 T \\ 0 & 0 & T_i\beta_2 + B\beta_3 & (T-T_i)\beta_2 - q_0 - B(q_1+\beta_3) & (T-T_i)\beta_3 - q_1 T_i \\ 0 & 0 & 0 & 0 & -\mu \end{pmatrix}.$$

Let us investigate the stability of the equilibrium points.

1. Equilibrium point $E_0 = (\frac{\Lambda_1}{d}, 0, 0, 0, \frac{\Lambda_3}{\mu})$

   This equilibrium point is disease-free equilibrium point because, it contains only human population and chicken population $N = \frac{\Lambda_1}{d}$ and $B = \frac{\Lambda_3}{\mu}$ respectively.

   Then let us do the stability analysis by substituting the disease-free equilibrium point E_0 in the Jacobian matrix,

$$J(E_0) = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ 0 & -d-\alpha-\gamma & 0 & \frac{\beta_1\Lambda_1}{d} & 0 \\ 0 & 0 & r_2 - q_0 - \frac{q_1\Lambda_3}{\mu} & 0 & 0 \\ 0 & 0 & \frac{\beta_3\Lambda_3}{\mu} & -\frac{(q_1+\beta_3)\Lambda_3 + q_0\mu}{\mu} & 0 \\ 0 & 0 & 0 & 0 & -\mu \end{pmatrix}.$$

   Eigenvalues of $J(E_0)$ are

$$\begin{aligned} \lambda_1 &= -d, \\ \lambda_2 &= -d-q-\gamma, \\ \lambda_3 &= -\mu, \\ \lambda_4 &= -\frac{q_1\Lambda_3 + q_0\mu + \beta_3\Lambda_3}{\mu}, \\ \lambda_5 &= -q_0 + r_2 - \frac{q_1\Lambda_3}{\mu}. \end{aligned}$$

   It is obvious that eigenvalues $\lambda_1, \lambda_2, \lambda_3$ and λ_4 are negative. But the eigenvalue λ_5 becomes negative when

$$\frac{r_2\mu}{q_0\mu + q_1\Lambda_3} < 1$$

   is satisfied. In this case, all eigenvalues will have negative real parts then we can say that the disease-free equilibrium point E_0 is called locally asymptotically

stable according to theorem 1.2.1. Hence, the disease dies out and tick population decrease. If

$$\frac{r_2\mu}{q_0\mu + q_1\Lambda_3} > 1$$

then λ_5 is positive. Then disease-free equilibrium point E_0 is a unstable equilibrium point. So, we can say that the threshold value of this model is

$$R_0 = \frac{r_2\mu}{q_0\mu + q_1\Lambda_3}. \tag{3.44}$$

As a result, when $R_0 < 1$, the equilibrium point E_0 is called locally asymptotically stable according to the theorem (1.2.1). In this case, the flow gets closer to the equilibrium point over time. If $R_0 > 1$ the equilibrium point is called unstable and flow goes away from the equilibrium point E_0. Besides if $R_0 = 1$ bifurcation occurs.

2. Equilibrium point $E^* = (N^*, I^*, T^*, T_i^*, B^*)$

The components of this endemic equilibrium point are given;

$$\begin{aligned}
N^* &= \frac{(d+\alpha+T_i^*\beta_1+\gamma)\Lambda_1}{(d+\alpha)(d+T_i^*\beta)+d\gamma}, \\
I^* &= \frac{T_i^*\beta_1\Lambda_1}{(d+\alpha)(d+T_i^*\beta_1)+d\gamma}, \\
T^* &= \frac{K_2((r_2-q_0)\mu - q_1\Lambda_3)}{r_2\mu}, \\
T_i^* &= -\frac{1}{2r_2\beta_2\mu^2}\Big[(q_1(r_2+K_2\beta_2)+r_2\beta_3)\Lambda_3\mu + (q_0r_2+K_2(q_0-r_2)\beta_2)\mu^2 \\
&\quad -\{\mu^2(-4K_2r_2\beta_2\beta_3\Lambda_3(q_1\Lambda_3+(q_0-r_2)\mu) + [q_1(r_2+K_2\beta_2)\Lambda_3 + K_2q_0\beta_2\mu \\
&\quad +r_2(\beta_3\Lambda_3+q_0\mu-K_2\beta_2\mu)]^2)\}^{1/2}\Big], \\
B^* &= \frac{\Lambda_3}{\mu}.
\end{aligned}$$

The endemic equilibrium point E^* exists when the conditions

- $r_2\mu > q_1\Lambda_3$,
- $\dfrac{r_2\mu}{q_0\mu + q_1\Lambda_3} > 1$

are provided. Note that last condition refers $R_0 > 1$(see (3.44)). When we substitute endemic equilibrium in Jacobian matrix,

$$J(E^*) = \begin{pmatrix}
-d & -\alpha & 0 & 0 & 0 \\
T_i^*\beta_1 & -d-\alpha-T_i^*\beta_1-\gamma & 0 & (N^*-I^*)\beta_1 & 0 \\
0 & 0 & r_2-q_0-\dfrac{2r_2T^*}{K_2}-\dfrac{q_1\Lambda_3}{\mu} & 0 & -q_1T^* \\
0 & 0 & T_i^*\beta_2+\dfrac{\beta_3\Lambda_3}{\mu} & T^*\beta_2-q_0-2T_i^*\beta_2-\dfrac{(q_1+\beta_3)\Lambda_3}{\mu} & T^*\beta_3-T_i^*(q_1+\beta_3) \\
0 & 0 & 0 & 0 & -\mu
\end{pmatrix}$$

is obtained. The matrix $J(E^*)$ is can be written as a block matrix;

$$J(E^*)=\left(\begin{array}{cc|ccc} -d & -\alpha & 0 & 0 & 0 \\ T_i^*\beta_1 & -d-\alpha-T_i^*\beta_1-\gamma & 0 & (N^*-I^*)\beta_1 & 0 \\ \hline 0 & 0 & r_2-q_0-\frac{2r_2T^*}{K_2}-\frac{q_1\Lambda_3}{\mu} & 0 & -q_1T^* \\ 0 & 0 & T_i^*\beta_2+\frac{\beta_3\Lambda_3}{\mu} & T^*\beta_2-q_0-2T_i^*\beta_2-\frac{(q_1+\beta_3)\Lambda_3}{\mu} & T^*\beta_3-T_i^*(q_1+\beta_3) \\ 0 & 0 & 0 & 0 & -\mu \end{array}\right).$$

We cab write

$$J_1(E^*)=\begin{pmatrix} -d & -\alpha \\ T_i^*\beta_1 & -d-\alpha-T_i^*\beta_1-\gamma \end{pmatrix}$$

and

$$J_2(E^*)=\begin{pmatrix} r_2-q_0-\frac{2r_2T^*}{K_2}-\frac{q_1\Lambda_3}{\mu} & 0 & -q_1T^* \\ T_i^*\beta_2+\frac{\beta_3\Lambda_3}{\mu} & T^*\beta_2-q_0-2T_i^*\beta_2-\frac{(q_1+\beta_3)\Lambda_3}{\mu} & T^*\beta_3-T_i^*(q_1+\beta_3) \\ 0 & 0 & -\mu \end{pmatrix}.$$

Since

$$Trace(J_1(E^*))=-2d-\alpha-T_i^*\beta_1-\gamma<0,$$

and

$$det(J_1(E^*))=d^2+d\alpha+dT_i^*\beta_1+T_i^*\alpha\beta_1+d\gamma>0$$

according to theorem (3.2.1) both eigenvalues of matrix $J_1(E^*)$ have negative real parts. Now, let us investigate sign of the eigenvalues of matrix $J_2(E^*)$. Eigenvalues of this matrix are

$$\begin{aligned} \lambda_1&=-\mu, \\ \lambda_2&=r_2-q_0-\frac{2r_2T^*}{K_2}-\frac{q_1\Lambda_3}{\mu}, \\ \lambda_3&=\beta_2(T^*-T_i^*)-q_0-\frac{(q_1+\beta_3)\Lambda_3}{\mu}. \end{aligned}$$

It is clear that the eigenvalue λ_1 is negative. If we substitute T^*, T_i^* and R_0 in eigenvalues λ_2 and λ_3, we obtain

$$\begin{aligned} \lambda_2&=-\frac{(R_0-1)(q_1\Lambda_3+q_0\mu)}{\mu}, \\ \lambda_3&=-\frac{1}{R_0\mu(q_1\Lambda_3+q_0\mu)}\{(q_1\Lambda_3+q_0\mu)^2[4K_2(R_0-1)R_0\beta_2\beta_3\Lambda_3\mu \\ &\quad+(q_1R_0\Lambda3+K_2\beta_2\mu+R_0(\beta_3\Lambda_3+q_0\mu-K_2\beta_2\mu))^2]\}^{1/2}. \end{aligned}$$

Recall, the equilibrium point E^* exists, when $R_0 > 1$. So we can say that eigenvalues λ_2 and λ_3 are negative. As a result, all eigenvalues of matrix $J_2(E^*)$ are negative. So, this equilibrium point is a locally asymptotically stable according to the theorem 1.2.1.

As a result, equilibrium points of the system has two non-negative equilibria. E_0 is a disease-free equilibrium point and E^* is an endemic equilibrium point. Equilibrium point E_0 exists without any conditions, whereas E^* exists when $R_0 > 1$.

Sensitivity Analysis of R_0

To analyse the sensitivity of basic reproduction number R_0 to each of its parameters, we use method of Arriola and Hyman [25]. The normalised forward sensitivity index with respect to each of the parameters is calculated as,

$$A_{r_2} = \frac{\frac{\partial R_0}{R_0}}{\frac{\partial r_2}{r_2}} = \frac{r_2}{R_0}\frac{\partial R_0}{\partial r_2} = r_2\left(\frac{q_0\mu + q_1\Lambda_3}{r_2\mu}\right)\left(\frac{\mu}{q_0\mu + q_1\Lambda_3}\right) = 1, \tag{3.45}$$

$$\begin{aligned}
A_\mu &= \frac{\frac{\partial R_0}{R_0}}{\frac{\partial \mu}{\mu}} = \frac{\mu}{R_0}\frac{\partial R_0}{\partial \mu} = \mu\left(\frac{q_0\mu + q_1\Lambda_3}{r_2\mu}\right)\left(\frac{q_1 r_2\Lambda_3}{(q_1\Lambda_3 + q_0\mu)^2}\right) \\
&= \frac{q_1\Lambda_3}{q_1\Lambda_3 + q_0\mu} > 0, \\
A_{q_1} &= \frac{\frac{\partial R_0}{R_0}}{\frac{\partial q_1}{q_1}} = \frac{q_1}{R_0}\frac{\partial R_0}{\partial q_1} = q_1\left(\frac{q_0\mu + q_1\Lambda_3}{r_2\mu}\right)\left(-\frac{r_2\Lambda_3\mu}{(q_1\Lambda_3 + q_0\mu)^2}\right) \\
&= -\left(\frac{q_1\Lambda_3}{q_1\Lambda_3 + q_0\mu}\right) < 0, \\
A_{\Lambda_3} &= \frac{\frac{\partial R_0}{R_0}}{\frac{\partial \Lambda_3}{\Lambda_3}} = \frac{\Lambda_3}{R_0}\frac{\partial R_0}{\partial \Lambda_3} = \Lambda_3\left(\frac{q_0\mu + q_1\Lambda_3}{r_2\mu}\right)\left(-\frac{q_1 r_2\mu}{(q_1\Lambda_3 + q_0\mu)^2}\right) \\
&= -\left(\frac{q_1\Lambda_3}{q_1\Lambda_3 + q_0\mu}\right) < 0, \\
A_{q_0} &= \frac{\frac{\partial R_0}{R_0}}{\frac{\partial q_0}{q_0}} = \frac{q_0}{R_0}\frac{\partial R_0}{\partial q_0} = q_0\left(\frac{q_0\mu + q_1\Lambda_3}{r_2\mu}\right)\left(-\frac{r_2\mu^2}{(q_1\Lambda_3 + q_0\mu)^2}\right) \\
&= -\left(\frac{q_0\mu}{q_1\Lambda_3 + q_0\mu}\right) < 0.
\end{aligned}$$

We can see that, among these six parameters, basic reproduction number R_0 is most sensitive to change in r_2 and μ. A decrease or increase in r_2 causes a decrease or

increase in R_0 with the same proportion. And also an increase or decrease in the value of μ leads to a corresponding increase or decrease in R_0. Conversely, the other three parameters have an inversely proportional relationship with R_0, so an increase in q_1, Λ_3 and q_0 will bring about a decrease in R_0. Recall that the parameter μ is the death rate of chickens. Increase in μ is not preferred. Moreover, the parameters q_1, Λ_3, β_3 and q_0 may have directly or inversely proportional relationship with the reproduction number. As a result R_0 is the most sensitive to changes in r_1 because, although $A_\mu > 0$, it is also seen that $A_\mu < 1$.

Bifurcation Analysis

In this model we shall examine bifurcation analysis for this model. It is known that, bifurcation may occur when basic reproduction number $R_0 = 1$. We use the Castillo-Chavez and Song bifurcation theorem (2.2.1).

We set $S = x_1$, $I = x_2$, $T_s = x_3$, $T_i = x_4$ and $B = x_5$. Therefore it can be said that $N = x_1 + x_2$ and $T = x_3 + x_4$. System (3.34)-(3.38) is written in therms of the notation $\dot{x} = f(x)$ as follows:

$$\begin{aligned} f_1 &= \Lambda_1 - d(x_1 + x_2) - \alpha x_2, \\ f_2 &= \beta_1 x_1 x_4 - (\gamma + d + \alpha)x_2, \\ f_3 &= r_2(x_3 + x_4)(1 - \frac{x_3 + x_4}{K_2}) - (q_0 + q_1 x_5)(x_3 + x_4), \\ f_4 &= \beta_2 x_3 x_4 + \beta_3 x_3 x_5 - (q_0 + q_1 x_5)x_4, \\ f_5 &= \Lambda_3 - \mu x_5. \end{aligned} \tag{3.46}$$

Let us consider the parameter ϕ which is given in the theorem (2.2.1) is represented by r_2. Note that r_2 is the obvious choice of the bifurcation parameter because, as it has been shown in (3.45) that basic reproduction number R_0 is more sensitive to changes in r_2. So, we can write,

$$\tilde{r_2} = \frac{q_0\mu + q_1\Lambda_3}{\mu}.$$

So, the disease free equilibrium point E_0 of this model is $(\tilde{x_1} = \frac{\Lambda_1}{d}, \tilde{x_2} = 0, \tilde{x_3} = 0, \tilde{x_4} = 0, \tilde{x_5} = \frac{\Lambda_3}{\mu})$. The linearisation around the disease free equilibrium evaluated at $\tilde{r_2}$ is

given by

$$J = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ 0 & -d-\alpha-\gamma & 0 & \frac{\beta_1\Lambda_1}{d} & 0 \\ 0 & 0 & -0 & 0 & 0 \\ 0 & 0 & \frac{\beta_3\Lambda_3}{\mu} & -\frac{(q_1+\beta_3)\Lambda_3+q_0\mu}{\mu} & 0 \\ 0 & 0 & 0 & 0 & -\mu \end{pmatrix}. \tag{3.47}$$

Eigenvalues of this matrix are

$$\begin{aligned} \lambda_1 &= 0, \\ \lambda_2 &= -d, \\ \lambda_3 &= -d-\alpha-\gamma, \\ \lambda_4 &= -\mu, \\ \lambda_5 &= -\frac{q_1\Lambda_3+\beta_3\Lambda_3+q_0\mu}{\mu}. \end{aligned}$$

It is clear that λ_1 is a simple eigenvalue of $D_x f$. So we can find right eigenvector and left eigenvector according to the theorem (2.2.1).

The right eigenvector w corresponding to the zero eigenvalue is

$$w = (-\frac{\alpha\beta_1\Lambda_1}{d^2(d+\alpha+\gamma)}, \frac{\beta_1\Lambda_1}{d(d+\alpha+\gamma)}, \frac{(q_1+\beta_3)\Lambda_3+q_0\mu}{\beta_3\Lambda_3}, 1, 0)^T$$

and left eigenvector v corresponding to the zero eigenvalue is

$$v = (0,0,1,0,0)$$

.

The second derivatives in formulas (3.18) are evaluated at the disease free equilibrium point $E_0 = (\frac{\Lambda_1}{d}, 0, 0, 0, \frac{\Lambda_3}{\mu})$, and $r_2 = \tilde{r_2}$. The non-zero derivatives are given as follows,

$$\begin{aligned} &\frac{\partial^2 f_2}{\partial x_1 \partial x_4} = \frac{\partial^2 f_2}{\partial x_4 \partial x_1} = \beta_1, && \frac{\partial^2 f_3}{\partial x_3 \partial x_4} = \frac{\partial^2 f_3}{\partial x_4 \partial x_3} = -q_1 - 2\frac{r_2}{K_2}, \\ &\frac{\partial^2 f_3}{\partial {x_3}^2} = -2\frac{r_2}{K_2}, && \frac{\partial^2 f_3}{\partial {x_4}^2} = -2q_1 - 2\frac{r_2}{K_2}, \\ &\frac{\partial^2 f_4}{\partial x_3 \partial x_4} = \frac{\partial^2 f_4}{\partial x_4 \partial x_3} = \beta_2, && \frac{\partial^2 f_4}{\partial x_3 \partial x_5} = \frac{\partial^2 f_4}{\partial x_5 \partial x_3} = \beta_3, \\ &\frac{\partial^2 f_4}{\partial x_4 \partial x_5} = \frac{\partial^2 f_4}{\partial x_5 \partial x_4} = -q_1, \end{aligned}$$

$$\frac{\partial^2 f_3}{\partial x_3 \partial r_2} = 1, \qquad \frac{\partial^2 f_3}{\partial x_4 \partial r_2} = 1.$$

If these derivatives are substituted in a and b,

$$\begin{aligned}
a &= v_2 w_1 w_4 \frac{\partial^2 f_2}{\partial x_1 \partial x_4} + v_2 w_4 w_1 \frac{\partial^2 f_2}{\partial x_4 \partial x_1} + 2 v_3 {w_3}^2 \frac{\partial^2 f_3}{\partial {x_3}^2} + v_3 w_3 w_4 \frac{\partial^2 f_3}{\partial x_3 \partial x_4} \\
&+ v_3 w_4 w_3 \frac{\partial^2 f_3}{\partial x_4 \partial x_3} + v_3 {w_4}^2 \frac{\partial^2 f_3}{\partial {x_4}^2} + v_4 w_3 w_4 \frac{\partial^2 f_4}{\partial x_3 \partial x_4} + v_4 w_4 w_3 \frac{\partial^2 f_4}{\partial x_4 \partial x_3} \\
&+ v_4 w_3 w_5 \frac{\partial^2 f_4}{\partial x_3 \partial x_5} + v_4 w_5 w_3 \frac{\partial^2 f_4}{\partial x_5 \partial x_3} + v_4 w_4 w_5 \frac{\partial^2 f_4}{\partial x_4 \partial x_5} + v_4 w_5 w_4 \frac{\partial^2 f_4}{\partial x_5 \partial x_4} \\
&= -2q_1 - 2\frac{r_2}{K_2} - \frac{2(K_2 q_1 + 2r_2)((q_1+\beta_3)\Lambda_3) + q_0\mu}{K_2 \beta_3 \Lambda_3} - \frac{4r_2((q_1+\beta_3)\Lambda_3 + q_0\mu)^2}{K_2 {\beta_3}^2 {\Lambda_3}^2}, \\
b &= v_3 w_3 \frac{\partial^2 f_3}{\partial x_3 \partial r_2} + v_3 w_4 \frac{\partial^2 f_3}{\partial x_4 \partial r_2} = 2 + \frac{q_1 \Lambda_3 + q_0 \mu}{\beta_3 \Lambda_3}
\end{aligned}$$

are obtained. It is clear that $a < 0$ and $b > 0$. This shows, according to the result of *iv.* in the theorem (2.2.1), the stability of the equilibrium point E_0 is changed from stable to unstable.

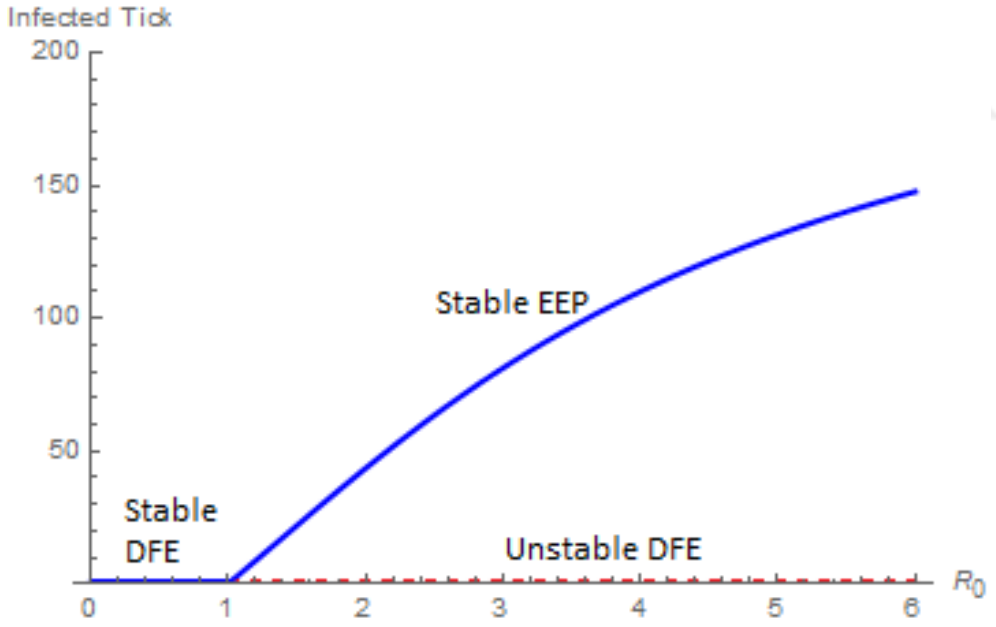

Figure 3.13 : Bifurcation diagram of the Infected Tick Population. Where $K_2 = 1000$, $q_0 = 0.015$, $q_1 = 0.033$, $\beta_3 = 0.0015$, $\Lambda_3 = 3$, $\beta_2 = 0.0023$, $\mu = 0.05$.

When $R_0 < 1$, equilibrium point E_1 is stable and components of the equilibrium point E^* are negative. It is mean, when $R_0 < 1$, the equilibrium point E^* does not exist. Therefore, when $R_0 < 1$, there only exists the disease-free equilibrium point. When $R_0 > 1$, the disease-free equilibrium point becomes unstable. And also when $R_0 > 1$

and $r_2\mu > q_1\Lambda_3$ provide endemic equilibrium point E^* becomes positive and stable. As a result, we can say that forward bifurcation occurs.

Simulations

Here, we simulate the model to understand the stability analysis of the system better. Let us fix the following parameters and total human population in the system (3.39) - (3.43) as follows:

$$N = 5000, \quad r_2 = 0.95, \quad K_2 = 1000, \quad r_2 = 0.95$$
$$q_0 = 0.015, \quad \beta_2 = 0.0023, \quad \beta_3 = 0.0015, \quad \beta_1 = 0.0027,$$
$$\alpha = 0.0018, \quad d = 0.0064, \quad \gamma = 0.037.$$

We choose the initial conditions as,

$$B(0) = 20, \quad T(0) = 100, \quad T_i(0) = 0, \quad I(0) = 0.$$

1. *Case 1*

   In this case, we assume that the chicken population is increased over time. When $\Lambda_3 = 5$ and $\Lambda_3 = 4$, the increment in chicken population over time is shown in figure (3.14). Also, how this increment in the chicken population affects the number of ticks is shown in figures (3.15) and (3.16).

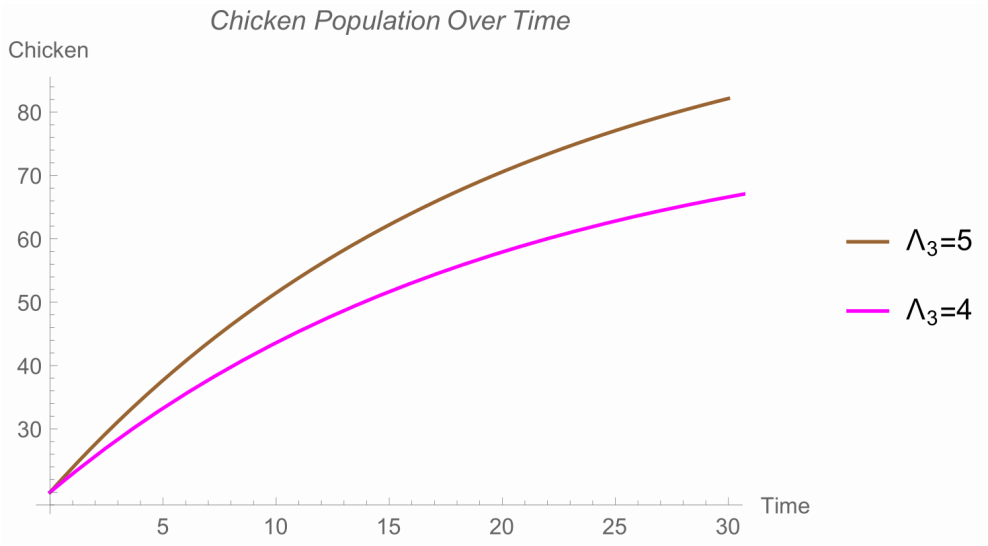

Figure 3.14 : Chicken Population Over Time. We choose $\Lambda_3 = 4$ and $\Lambda_3 = 5$, so that $R_0 < 1$.

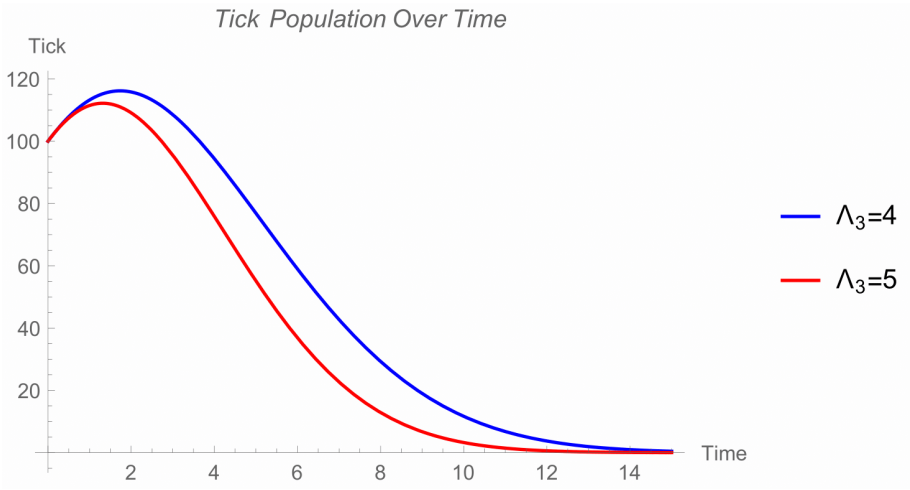

Figure 3.15 : Tick Population Over Time.

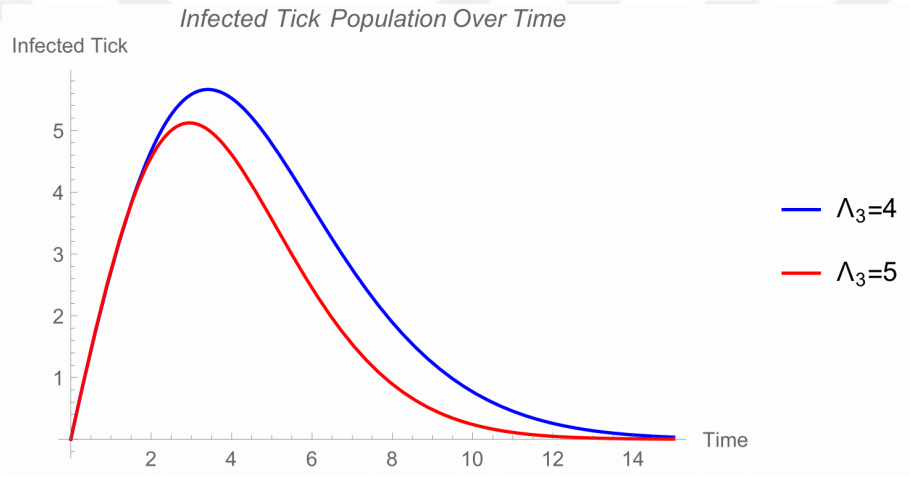

Figure 3.16 : Infected Tick Population Over Time.

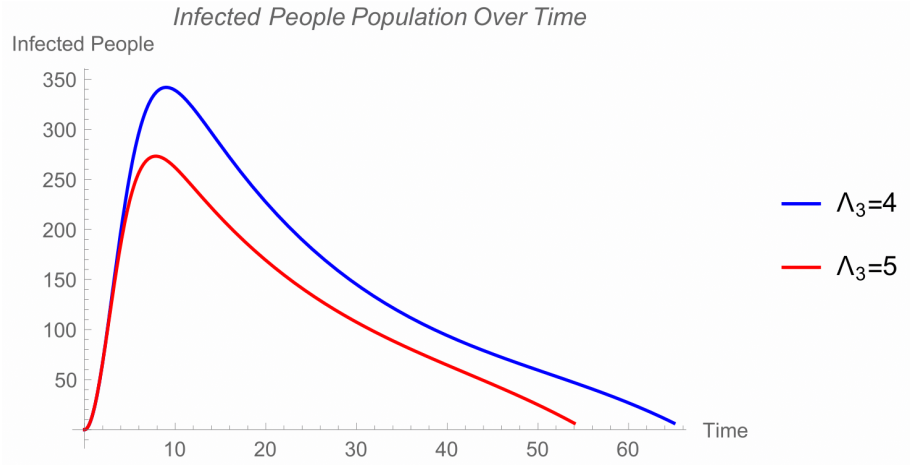

Figure 3.17 : Infected People Population Over Time.

If we choose the parameter $\Lambda_3 = 4$, then $R_0 = 0.3578 < 1$. And if we choose the parameter $\Lambda_3 = 5$, then $R_0 = 0.2865 < 1$. As we can see in the figures (3.15), (3.16) and (3.17), tick population, infected tick population decreases and eventually vanishes. Also the disease eventually dies out.

Recall that, Λ_3 is population growth of chickens. According to figures (3.14) an increase in Λ_3 causes the tick population and infected tick population increase less and vanish faster. In conclusion, when the number of daily added chickens in to the environment increase, tick population and infected tick population vanish faster in the environment.

2. *Case 2*

   In this case, we assume that the chicken population is decreased over time. When $\Lambda_3 = 0.9$ the decrement in chicken population over time is shown in figure (3.19). Also, how this decrement in the chicken population affects the number of ticks and infected people population is shown in Figures (3.19), (3.20) and (3.21).

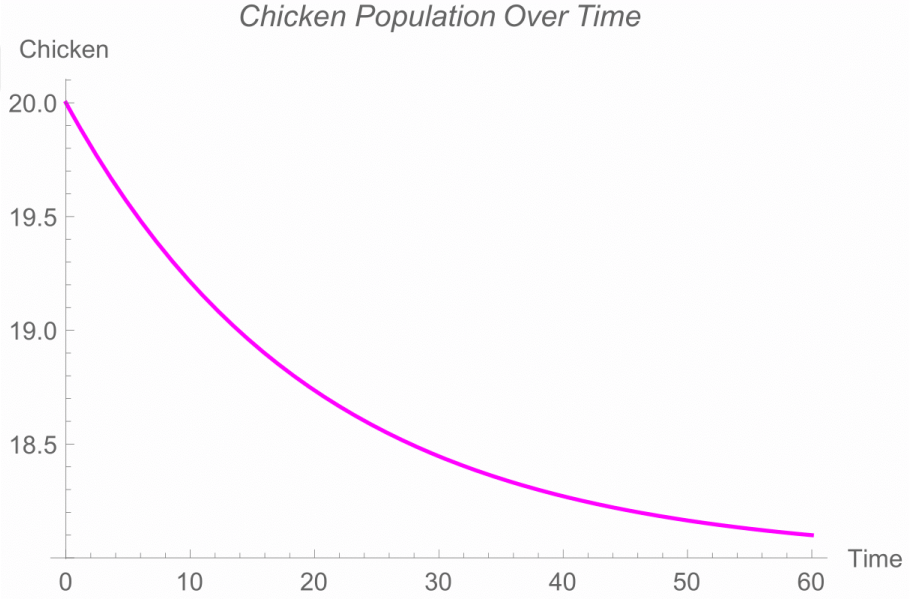

Figure 3.18 : Chicken Population Over Time. We choose $\Lambda_3 = 0.9$, so that $R_0 > 1$.

If we choose parameter $\Lambda_3 = 0.9$, then $R_0 = 1.5599$. As we can see in the figures (3.19), (3.20) and (3.21), we can say that the tick population, the infected tick population and the infected people population increase. So, the disease spreads and becomes endemic.

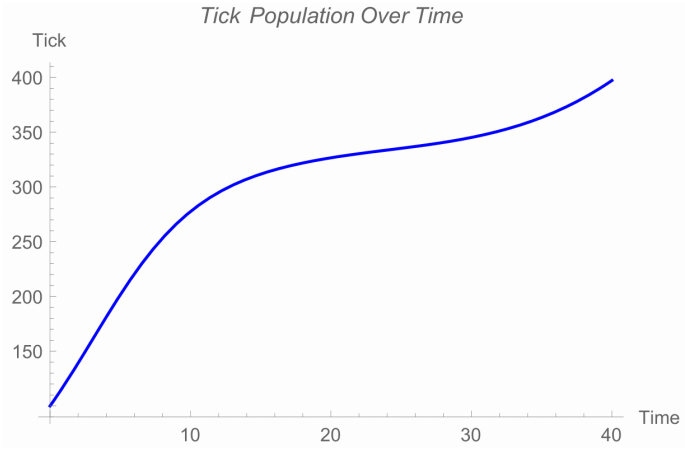

Figure 3.19 : Tick Population Over Time.

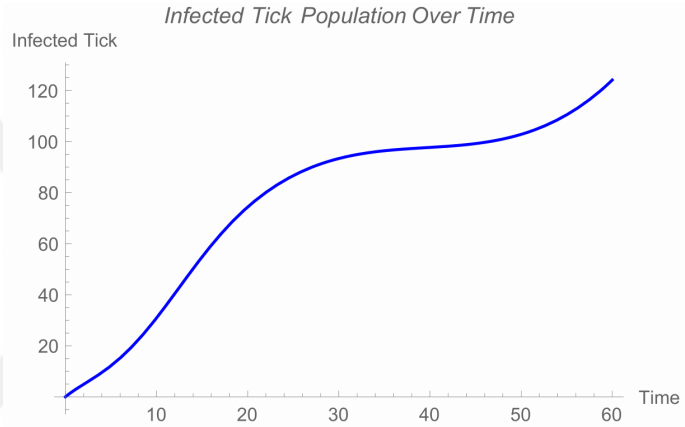

Figure 3.20 : Infected Tick Population Over Time.

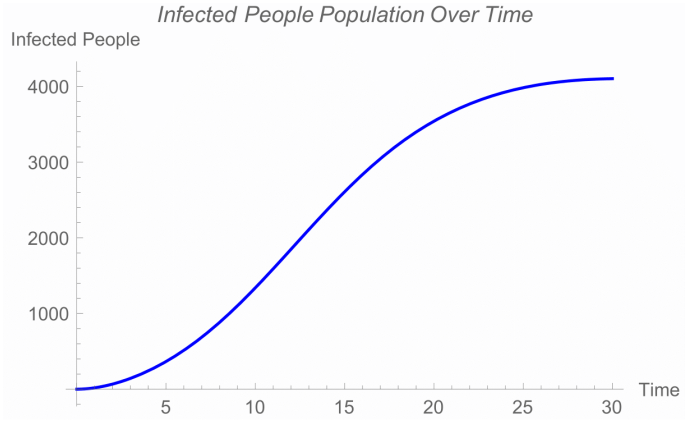

Figure 3.21 : Infected People Population Over Time.

As a result, if the number of chickens daily added in the environment increases, the number of tick and infected tick population can be decreased. Thus, the spread of the disease can be prevented in the environment.

4. CONCLUSIONS AND RECOMMENDATIONS

In this thesis, we have studied the spread of a tick bone disease into human with the effect of chicken-bird existence in a mathematical perspective. We considered every dependent variable as a separated compartment and transmission between these components are assumed to be continuous. There are three basic dependent variables; human and ticks are taken into consideration in two main groups, susceptible and infected whereas birds are assumed to be a single group. In addition to the interactions between two subgroups of human and ticks, we have also investigated the interaction between birds-ticks and human-tick.

We have examined three different cases by depending on the assumptions to the entrance to bird and tick compartments. In one, the entrance of both are considered with logistic growth model and in the other two, when one is taken into account with logistic growth, the other is assumed to be constant. We have considered in all these cases the virus might be transmitted to human by a tick bite. Tick might get virus either by a contact to an infected tick or to the birds which carry the virus. The relation between ticks and bird compartments is not only limited to this transmission, but the decrement of ticks has been also taken into consideration as they are natural preys for birds-chicken. We have also taken into consideration the natural and disease-related deaths.

The problem, as it is expressing many various possible cases, represents a quite general study which has not been done before in the literature. But as mentioned in the thesis before, normally ticks are at three stages and the diseases might be transmitted by not only by the adult ticks but also by larvae and nymph. Here we have only considered ticks as adult ticks, for a more general future study ticks might be taken into consideration at their three different stages. Because the problem with its present form is hard enough, considering ticks in their 3 stage will be for sure much harder. To handle such a problem some contacts between compartments should be given up.

In the thesis, each problem is examined as application of Hartman Grobman Theorem. That is to say we have investigated the local stability of the equilibrium points. The basic Reproduction numbers are determined and with respect to the reproduction numbers, the parameter regimes are studied. The bifurcation analysis has also been done by considering Castillo Chavez Bifurcation Theorem. Finally, for each case, some simulations for particular parametric regimes has been done. The graphs of solution curves are depicted and the physical expressions of the results have been discussed.

As a future problem, even though it would be really hard, the global stability analysis might be studied by taking some simplifications.

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CURRICULUM VITAE

Name Surname: Miray ALIN

Place and Date of Birth: 16.10.1993, ISTANBUL

E-Mail: mirayalinn@gmail.com

EDUCATION:

- **B.Sc.:** 2015, Mimar Sinan Fine Arts University, Faculty of Science and Letters, Mathematics
- **M.Sc.:** 2020, Istanbul Technical University, Faculty of Science and Letters, Mathematical Engineering