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Lyapunov Approach in Global Asymptotic Stability of an Epidemic Model of Computer Viruses

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ملخص

الهدف من هذا العمل هو دراسة نظام المعادلات التفاضلية العادية من الدرجة الأولى لتحليل ديناميكيات فيروسات الكمبيوتر من خلال نموذج رياضي مقترح. يتم إجراء تحليل الاستقرار الكلي للنموذج الموسع من خلال دالة ليابونوف المناسبة، حيث نهتم فقط بأجهزة الكمبيوتر الداخلية المتصلة بالإنترنت والتي تصنف إلى أجهزة غير مصابة (أي أجهزة كمبيوتر خالية من الفيروسات)، أجهزة الكمبيوتر المصابة الكامنة حاليًا وأجهزة الكمبيوتر المصابة التي ستخترق . يمكن أن يلعب رقم التكاثر الأساسي R_0 دورًا في تحديد ما إذا كان الفيروس سينقرض أو يستمر، إذا كان $R_0 < 1$ فإن التوازن الخالي من الفيروس يكون مستقرًا كليا وغير مستقر اذا كان $R_0 > 1$.

ومن ثم ، فإننا نقترح بعض الحالات للبحث المستقبلي ، وهي: عندما $R0 = 1$.هل التوازن الخالي من الأمراض والمتوطن مستقر بشكل مقارب؟

الكلمات المفتاحية: نماذج ديناميكية، انتشار فيروس الحاسوب ، استقرار كلي، فيروس الحاسوب، دالة ليابونوف، استقرار فولتيرا-ليابونوف.

Résumé

Le but de ce travail est d'étudier un système d'équations différentielles ordinaires de premier ordre est utilisé pour analyser la dynamique des virus ordinateurs via un modèle mathématique proposé. L'analyse de la stabilité globale est menée pour le modèle étendu par la fonction appropriée Lyapunov, intéressant seulement sur les ordinateurs internes connectés à Internet qu'ils sont classés à des ordinateurs non infectés (c.-à-d., ordinateurs sans virus), les ordinateurs infectés qui sont actuellement latents et les ordinateurs infectés qui sont entrain de se piraté. Le numéro de reproduction de base R_0 peut être utilisé pour déterminer si le virus va disparaître ou persister, si $R_0 < 1$, alors l'équilibre exempt de virus est globalement asymptotiquement stable et instable lorsque $R_0 > 1$.

Par conséquent, nous suggérons quelques aspects pour des recherches futures, qui sont : lorsque $R_0 =$. 1 Les équilibres sans maladie et endémiques sont-ils asymptotiquement stables ?

Mots clés: Modèles dynamiques , Propagation virale d'ordinateur , Stabilité globale , Virus d'ordinateur, Fonction de Lyapunov , Stabilité de Volterra–Lyapunov

Abstract.

The aim of this work is to study a system of first order ordinary differential equations is used to analyse the dynamics of computer viruses via a mathematical model proposed. The global stability analysis is conducted for the extended model by suitable Lyapunov function, interesting only on internal computers connected to the internet which they are classified to uninfected computers (i.e., virus-free computers), infected computers that are currently latent and infected computers that are currently breaking. The basic reproduction number R_0 can be played role in determining whether the virus will extinct or persist, if $R_0 < 1$ then the virus-free equilibrium is globally asymptotically stable and unstable when $R_0 > 1$.

Hence, we suggest some aspects for future research, which are : when $R_0 = 1$. Are the disease-free and endemic equilibrium asymptotically stable?

Key words: Dynamical models, Computer viral propagation, Global stability, Computer virus, Lyapunov function, Volterra–Lyapunov stability.

Dedication

I devote this modest work to all, who from near and far have given me their moral and physical support for the realization of this work.

To my parents Abdallah and Souhaila Hichour , my faithful brother Aimen and sisters Loudjain and Mira thank you for your everlasting love and warm encouragement throughout my wok. Without you , I couldn’t overcome my difficulties and concentrate on my studies.

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for agreeing to review this work.

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List of abbreviations and symbols

- The set of the real numbers is denoted by $\mathbb{R}$.
- The set of the real numbers of the n-elements, is denoted by $\mathbb{R}^n$.
- The determinant of real and complex martices is denoted by $\det(A)$.
- The trace of real and complex matrices is denoted by $tr(A)$.
- The invers of real and complex matrices is denoted by A^{-1}.
- The transpose of matrix A is denoted by A^T.
- The diagonal of real and complex matrices is denoted by $diag(A)$.
- The real part of a complex number is denoted by $\mathrm{Re}(A)$.
- The spectral radius of A, is denoted by $\rho(A)$.
- R_0 Basic Reproduction Number.
- The space of continuous and derivative functions is denoted by C^1.
- The disease-free equilibrium is denoted by (**DFE)**.

Introduction

By development of computer technologies and network applications, the Internet has become a powerful mechanism for propagating computer virus. Because of this, computers connected to the Internet become much vulnerable to digital threats. Computer viruses, including the narrowly defined viruses and network worms, are loosely defined as malicious codes that can replicate themselves and spread among computers. In this scenarioge number of existing computer viruses and their high level of destructivity appear as an important risk factor for corporations and individuals.

Developing a mathematical model for the computer viral propagation is of critical importance not only for under standing better the behavior of computer virus but also for stopping the spread of the virus.

The study of the global stability is not only mathematically important, but also essential in predicting the evolution of the virus in the long run, so that prevention and intervention strategies can be effectively designed.

In our work, we focus on the study of a dyn, the laramical model characterizing the spread of computer viruses over the Internet.

This work is divided into three chapter:

Chapter1: An Introduction to Computer Viruses

Presents an ovreview of computer viruses .

Chapter2: Notions of global stability and preliminary concepts

Introducing some definitions and concepts that we will used later.

Chapter3: Global stability of equilibrium points

Is devoted to the global stability of computer viruses

CHAPTER 1

AN INTRODUCTION TO COMPUTER VIRUSES

1.1 Introduction

A computer virus is a manmade destructive computer program or code that is generally loaded onto a computer system without the knowledge of a user and causes unauthorized and unwanted changes to the information stored on the computer. The term computer virus may be applied to software, code, code blocks and code segments which perform illegal damaging functions.

It is important to note that all software that causes damage is not virus. Popular legal software may cause damages because of certain unfixed bugs. But viruses are made for infecting other programs and troubling others, which these software are not.

Computer viruses are called viruses because they share some of the traits of biological viruses. A computer virus transmits from computer to computer like a biological virus transmits from person to person.

There are many who consider computer viruses as the offspring of Dr. Frederick B. Cohen. He created a virus, as part of his doctoral thesis, in an effort to find ways to defend computer systems from self-replicating programsThere are others who claim that computer viruses existed well before 1984 when Dr. Cohen did his research. The debate about the appearance of the first virus will probably continue far into the future. Currently it does not appear likely that computer scientists will agree upon an 'official' definition of the term.([13]

1.2 History and evolution

The viruses started to grow during mid 1980's till mid 1990's, when the use of PCs started to grow for businesses and homes. During that period computer games were very important and applications like word processors and spreadsheets were very popular. The viruses typically attached themselves to games or other bona fide computer programs. Here , we present a brief chronology of computer viruses :

1986 , First PC virus was created and termed the Brain virus. The virus was created in Pakistan and is a boot sector virus, i.e., it affects only the boot records. It falls under the stealth virus category.

1987, First memory resident file infector was discovered in Lehigh University and named Lehigh. Attacks executable files. Jerusalem virus first appeared at the Hebrew University, Jerusalem. It's another memory resident file infector.

1988, First anti-virus was Den Zuk created in Indonesia. It was designed to detect and remove the Brain virus and immunize disks against a Brain infection. Cascade Virus is found in Germany. It is an encrypted virus, meaning it was coded so that it could not be analyzed easily

1989, Data Crime virus is on the loose and strikes on Friday the 13th. Dark Avenger virus, attacks slowly, so that it goes unnoticed. It is the first full-stealth file infector.

1990, Many anti-virus products are introduced including IBM's McAfee, Digital Dispatch and Iris. Viruses combining various characteristics spring up like the Polymorphism and Multipartite.

1991, Symantec releases Norton antivirus software. Tequlia, a stealth, polymorphic and multi-partite virus is found.

1992, Media mayhem greeted the virus Michelangelo that March. Predictions of massive disruptions were made, and anti-virus software sales soared.

1994, A virus called Kaos4 was posted on a pornography news group file. It was encoded as text and downloaded by a number of users. Virus Pathogen appeared in England; the writer was tracked down by Scotland Yard's computer crime unit.

1994, The SatanBug virus appears; The anti-virus industry helps the FBI find the person who wrote it-a kid. Cruncher was considered a good virus as it compressed infected programs.

1995, Anti-virus companies worry about staying profitable with the emergence of Windows 95 because the boot viruses cannot infect it, hence the possible catastrophic loss of business. But the macro viruses that do infect the Windows-95 applications soon appear keeping the anti-virus companies happy and in the green.

1996, Concept, a macro-virus, becomes the most common virus in the world. Laroux

is the first virus to infect Microsoft® Excel spreadsheets.

1997, The Anti-Virus Research Center in Cupertino develops an exclusive technology called Bloodhound-Macro to address the growing number of new and unknown macro viruses.

1998, Posing as an anti-virus software, an e-mail attachment virus infects computers worldwide.

1999, The Melissa virus, a macro,causes worldwide destruction. Primarily infecting Microsoft(r) Word and Outlook, it automatically sends mail to everyone in the user's address book.

2000, I Love You virus causes havoc; it is transmitted via e-mail and, when opened, it automatically sends mail to everyone in the user's address book.

2001, Code Red virus exploits a security hole in Microsoft Internet Information Server (IIS) to spread. It disables the system file checker (SFC) in Windows. It probes random IP addresses to spread to other hosts.

2002 Nimda is a complex virus with a mass mailing worm component, which spreads itself in attachments named README. EXE. Nimda affects EXE files on local machines, locates email addresses and spreads itself. It also locates Web servers and infects them[16].

1.3 A Computer Virus

A computer 'virus' is defined as a program that can 'infect' other programs by modifying them to include a possibly evolved copy of itself. With the infection property, a virus can spread throughout a computer system or network using the authorizations of every user using it to infect their programs. Every program that gets infected may also act as a virus and thus the infection grows[15].

The following pseudo-program shows how a virus might be written in a pseudo-computer language. The ' := ' symbol is used for definition, the ':' symbol labels a statement, the ';' separates statements, the ' = ' symbol is used for assignment or comparison, the ' − ' symbol stands for not, the '{'*and*'}' symbols group sequences of statements together, and the ' ... ' symbol is used to indicate that an irrelevant portion of code has been left implicit.

1. This example virus (V) (Fig. 1) searches for an uninfected executable file (E) by looking for executable files without the "1234567" in the beginning, and prepends V to E, turning it into an infected file (I). V then checks to see if some triggering condition is true, and does damage. Finally, V executes the rest of the program it was prepended ' to. When the user attempts to execute E, I is executed in its place;

it infects another file and then executes as if it were E. With the exception of a slight delay for infection, I appears to be E until the triggering condition causes damage. We note that viruses need not prepend themselves nor must they be restricted to single infection per use[15].

```
program virus :=
{1234567;

subroutine infect-executable :=
  {loop: file = random-executable;
  if first-line-of-file = 1234567
        then goto loop;
  prepend virus to file;
  }

subroutine do-damage :=
  {whatever damage is desired}

subroutine trigger-pulled :=
  {return true on desired conditions}

main-program :=
  {infect-executable;
  if trigger-pulled then do-damage;
  goto next;
  }

next:}
```

Fig.1 Simple virus 'V'

(fig.1)

2. This program (C) finds an uninfected executable (E), compresses it and prepends C to form an infected executable (I). It then uncompresses the rest of itself into a temporary file and executes normally. When I is run, it will seek out and compress another executable before decompressing E into a temporary file and executing it. The effect is to spread through the system compressing executable files, decompressing them as they are to be executed. Users will experience significant delays as their executables are decompressed before being run[15].

```
program compression-virus :=
{01234567;
subroutine infect-executable :=
  {loop: file = random-executable;
  if first-line-of-file = 01234567
        then goto loop;
  compress file;
  prepend compression-virus to file;
  }
main-program :=
  {if ask-permission
        then infect-executable;
  uncompress the-rest-of-this-file
        into tmpfile;
  run tmpfile;
  }
}
```

Fig.2 Compression virus 'C'

(fig.2)

1. As a more threatening example, let us suppose that we modify the program V by specifying trigger-pulled as true after a given date and time and specifying do-damage as an infinite loop. With the level of sharing in most modern systems, the entire system would likely become unusable as of the specified date and time. A great deal of work might be required to undo the damage of such a virus. This modification is shown in Fig. 3[15].

```
...
subroutine do-damage :=
  {loop: goto loop;}
subroutine trigger-pulled :=
  {if year > 1984 then return(true)
        otherwise return(false);
...
```

Fig.3 A denial of services virus

(fig.3)

As an analogy to a computer virus, consider a biological disease that is 100% infectious, spreads whenever animals communicate, kills all infected animals instantly at a given moment, and has no detectable side effects until that moment. If a delay of even one week were used between the introduction of the disease and its effect, it would be very likely

to leave only a few remote villages alive, and would certainly wipe out the vast majority of modem society. If a computer virus of this type could spread through the computers of the world, it would likely stop most computer use for a significant period of time and wreak havoc on modern government, financial, business and academic institutions[15].

1.4 Computer viruses characteristics

There are thousands of viruses developed by different people at different times. Their operating platform, modes of operation, level of nuisance are all different. But all of them are unlawfully developed with malefic intentions. Some of the common characteristics of the viruses are as follows([14].

- Virus programs are illegitimate and secretly developed. No software company publicly declares to be developing viruses.
- One fundamental characteristic of a computer virus is that it is executable. Either the virus executes itself or attaches its code to another file to get executed. Without execution capability it can do no harm and will gain no importance.
- Another fundamental characteristic of a computer virus is that it replicates. A virus infects other programs generally by attaching its copies to those programs. Without replication the viruses cannot grow or survive.
- Virus can transmit from one computer to another through an infected file or an infected disk. Some viruses are capable of transmitting themselves across networks even bypassing security systems.
- Different types of viruses have different capabilities and limitations. For example, a boot sector virus infects boot sector and not the data files, a macro virus infects the documents and not executable files and so on.
- Skilled virus writers don't want their viruses to be detected. So they adopt stealth techniques. Thus many viruses use encryption mechanism and even change the encryption key as they travel from one host to another.
- The viruses are harmful whether intentionally or unintentionally. The socalled harmless viruses may appear to be harmless but they at least replicate themselves and utilize some memory of the system. This by itself is a harmful behavior.
- Every virus does not act immediately. Some viruses, like Trojan horses, wait patiently as benign programs until their events are triggered to become active.

- One of the weaknesses of viruses is that they generally don't do too much calculation without drawing attention to themselves. Hence although a virus is aware of the original checksum of a file it would be difficult for the virus to add itself to the file without changing the checksum.

1.5 How does the virus function

As the function of a computer system is very complex, there are many possible ways for a virus to get into a computer system[14].

- A boot sector virus enters through infected floppies. When a user transports an infected floppy disk to a second computer the virus infects the second computer and so on. The boot sector virus changes the master boot record of the hard disk and permanently resides in the computer system.

- A file virus works by copying itself to each executable program file it infects. When the executable program executes, the virus gains control of the computer and attempts to infect other files.

- An email virus takes the help of emails to be carried to other computers. Some email viruses create emails by using the address book in the infected system. Some viruses attach themselves to the outgoing emails and get transferred to the destination computer.

- A worm is a small piece of software that scans the network for another machine that has a specific security hole. When it finds the desired security hole it copies itself to the new machine by using the security hole and then starts replicating from there as well.

- A virus generally wants to get executed first before the host program is executed. Hence the virus typically adds its executable codes to the beginning of the program or replaces its first instructions, causing the program to be invoked with the first instructions of the virus instead of the first instructions of the program.

- Since a virus is a software code it can be transmitted along with any legitimate software that enters the computer. For example, a virus can spread from a LAN/ WAN based file server to any of the client systems attached to the server. Some viruses can rapidly spread through the Internet.

- In most cases the offending user spreads the virus without his knowledge. For example, a person might download an infected program and run it. In this case the virus loads itself into memory and infects other programs on the disk. Although the virus goes on infecting one file after other the user has no way to know even that a virus had ever ran.

1.6 Viruses infection patternes

There are several patterns of virus infections. Some viruses add themselves at the beginning of the host program. Some viruses append themselves to the host program and modify the header of the original host so that the execution will begin at the virus rather than the host. In these two patterns the original code of the host program remains in one single block.([14]

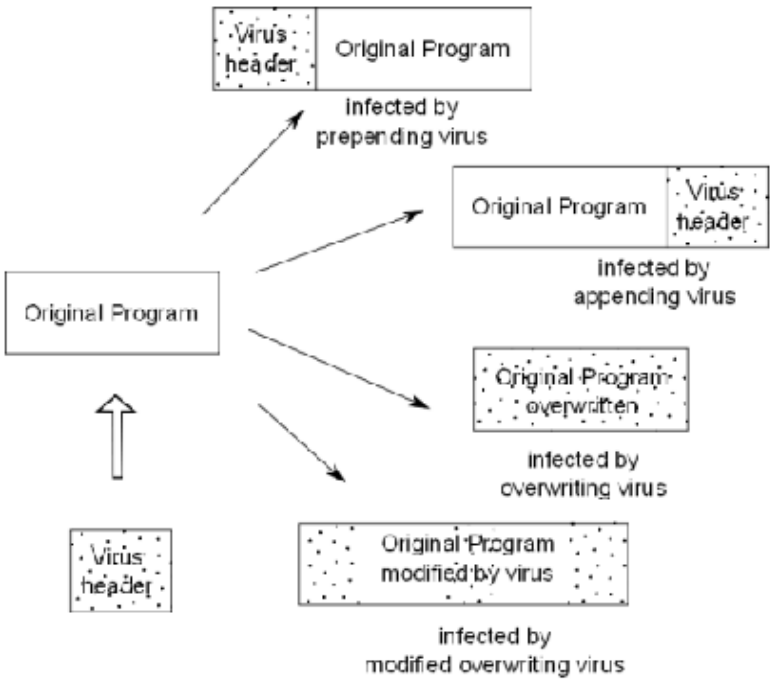

Fig.4 Virus Infection Paterns

(fig.4)

However there are many more complicated patterns too. The overwriting viruses overwrite a portion of the host and modify the header of the host so as to begin execution inside the virus. This pattern is dangerous as the original program is likely to suffer permanent damage. Modified overwriting viruses may write themselves at the beginning, end and other places in the original program. There are some complicated modified

overwriting viruses who scramble and overwrite the original host in various different ways and make them totally useless.

1.7 The result of viruses infection

Viruses may cause various kinds of damages to a computer system. The damage caused by a virus depends upon the type of virus causing the damage. Some viruses may cause fewer problems like displaying notorious messages on the screen, whereas some may cause serious dangers like formatting hard disk, causing permanent destruction of data and thereby making the system completely unusable and irreparable. The following are some of the common problems caused by the viruses([14].

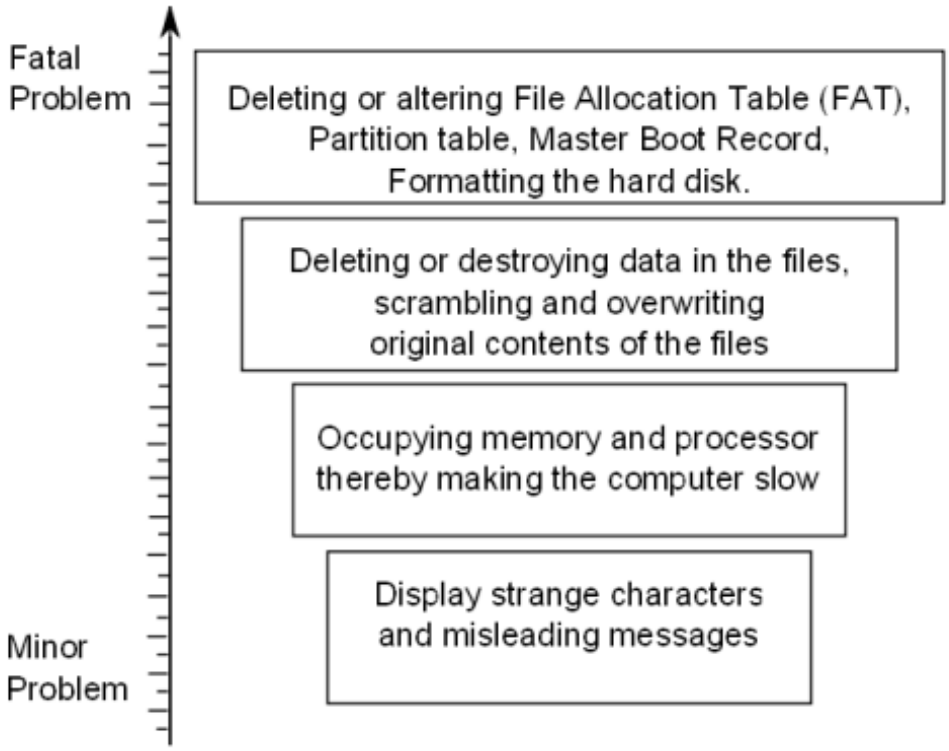

Fig.5 Different levels of harms made by computers viruses

(fig.5)

- Some viruses interfere into the display system by scrambling characters on the video display screens, displaying misleading text, video or audio messages to users. However these viruses are less dangerous.
- The so-called benign viruses even create problems for the computer user because

they typically take up computer memory used by legitimate programs. As a result, they often slow down the system or even result in system crashes. Apart from that many viruses are bug-ridden, and the bugs may lead to system crashes and data loss.

- Most viruses intend to alter or destroy data stored on the disks. They damage the computer by corrupting programs, erasing files, scrambling data on the hard disk, attacking FAT (File Allocation Table), attacking partition table, and even by formatting the hard disk. Some viruses operate to destroy the contents of the system hard disk thereby causing the system to completely inoperable.

- Many viruses like. Trojan horses, have a payload, which is a trigger such as a date or an action done by the user. When the payload is triggered the virus does its intended damages which can be mild to severe.

- Some viruses consume excessive amount of computer resources thereby preventing users from performing the desired tasks or leading to hang the computer. Even a simple virus can be dangerous as the virus ca quickly use a large portion of the available memory and possibly bring the computer system down.

- The viruses may attack client computers, network servers, network traffic or even the whole network. If an infected computer system is part of a network then viruses can be transmitted to other systems in the network causing widespread damage. Some viruses may use excessive amount of network resources or choke the network traffic.

- Another type of virus is a so-called stealth virus, which can take over some actions of the operating system, such as opening a file, in order to hide the infection of that file, before other programs can read that file (Conventional scanning programs cannot detect these viruses).

- Generally viruses try to exploit the loopholes of the operating system. Some viruses are carefully written to exploit the loopholes of anti-virus programs. As the users blindly believe on the anti-virus program intelligent viruses can exploit the loopholes of such programs.

1.8 Analogy Between Biological and Computer Viruses

Von Neumann's works aimed at finding a model to describe biological evolution process, and particularly self-reproduction. Later on, it was no accident when the term virus was chosen by Fred Cohen, since it perfectly matched phenomena already present in the wild. Gradually, a parallel between these two fields was naturally drawn in researchers's minds. There are many historical examples showing that scientific researchers have always drawn their inspiration from nature and have always tried to reproduce it.

As a matter of fact, each viral biological mechanism has an equivalent in the world of computer viruses. The following table summarizes the main features which are shared by both fields (further details about biological viruses are available in [18]).

Biological Viruses	**Computer viruses**
Attack on specific cells	Attack on specific file formats
Infected cells producenew viral off-springs	Infected programs produce viral codes
Modification of cell's genome	Modification of program's functions
Viruses use cell's structures to replicate	Viruses use format tructures for copy mechanisms
Viral interactions	Combined or antiviruses
Viruses replicates only in living cells	Execution is required to spread
Already infected cells are not reinfected	Viruses use infection marker to prevent over infection
Viral mutation	Viral polymorphism
Healthy virus carriers	Latent or dormant viruses

Analogy Between Biological Viruses and ComputerViruses

As a basic but powerful comparison, a cell's genetic material (DNA or Desoxyribonucleic Acid and RNA or RiboNucleic Acid) can be compared with program's codes (respectively source code and binary code; indeed in the same way DNA is the blueprint for RNA, source code is the blueprint for the executable code).As an example, a biological virus like the Ebola virus is very close to a computer worm such as Sapphire/Slammer insofar as in both cases, the virus quickly overcomes the carriers who consequently are unable to propagate the infection for very long. Similarly, a parallel could be drawn between the hiv and any polymorphic computer virus. In 1997, some researchers belonging to the Computer Departement of New Mexico University in Albuquerque, defined the

computer immunology concept by studying existing analogies between computer viruses and biological viruses with respect to the human immune system.

1.9 Conclusion

The viruses generally try to exploit the loopholes of the operating system application programs, windows sockets and even anti-virus programs. Som viruses are so dangerous that they can make the system completely unusabl and irreparable.

The large number of existing computer viruses and their high level of destructivity appear as an important risk factor for corporations and individuals, so developing a mathematical model (who lead to a better understanding and prediction of the scale and speed of computer virus propagation) for the computer viral propagation is of critical importance not only for under standing better the behavior of computer virus but also for stopping the spread of the virus.

CHAPTER 2

NOTIONS OF GLOBAL STABILITY AND PRELIMINARY CONCEPTS

In this chapter, we define and introduce the basic functional tools necessary to the global stability

2.1 Spectral radius

Definition 1 *[19]Let T be an operator in a finite dimensional Banach space X (i.e T is a square matrix). Then the set $\sigma(T)$ is compact and it is composed of the eigenvalues of T.*

Definition 2 *Let M be a square matrix with complex coefficients, we call spectral radius of M, and we denote by $\rho(M)$ the greatest modulus of the eigenvalues of M*

Definition 3 *[19] The spectral radius $\rho(M)$ of matrix M is the number*

$$\rho(M) = \max\{|\lambda, \lambda \in \sigma(M)|\}$$

2.2 Ordinary Differential Equations

2.2.1 Definitions

Let $\Omega \subset \mathbb{R}^{1+n}$ be qn open connected set. We will note the points in Ω by (t, x) where $t \in \mathbb{R}$ and $x \in \mathbb{R}^n$. Let $f : \Omega \to \mathbb{R}^n$ a continuous function .

From an initial point $(t_0, x_0) \in \Omega$, we want to build a unique solution to the initial value problem:

$$\begin{cases} x' = f(t,x), \\ x(t_0) = x_0, \end{cases} \tag{2.1}$$

For it , $x(t)$ must be a function of classe C^1 in some interval $I \subset \mathbb{R}$ containing the initial instant t_0 with values in $\mathbb{R}^n$ such that the solution curve satisfies

$$\{(t, x(t)) : t \in I\} \subset \Omega$$

Such a solution is called a local solution when $I \neq \mathbb{R}$. When $I = \mathbb{R}$,the solution is called global.

The Cauchy-Peano Existence Theorem

Theorem 4 *((Cauchy-Peano)[8]) If $f : \Omega \to \mathbb{R}^n$ is continuous, then for every point $(t_0, x_0) \in \Omega$ the initial value problem (2.1) has local solution.*

The Picard Existence Theorem

The failure of uniqueness can be rectified by placing an additional restriction on the vector field. The next definitions introduce this key property.

Definition 5 *([8]) Let $S \subset \mathbb{R}^m$. Suppose $x \mapsto f(x)$ is a function from S to $\mathbb{R}^n$.*
The function f is said to be Lipschitz continuous on S if there exists a constant $C > 0$ such that

$$\|f(x_1) - f(x_2)\|_{\mathbb{R}^n} \leq C \|x_1 - x_2\|_{\mathbb{R}^m}$$

for all $x_1, x_2 \in S$.

Definition 6 *([8]) Let $\Omega \subset \mathbb{R}^{n+1}$ be an open set. A continuous function $(t,x) \mapsto f(t,x)$ from Ω to $\mathbb{R}^n$ is said to be locally Lipschitz continuous in x if for every compact set $K \subset \Omega$, there is a constant $C_K > 0$ such that*

$$\|f(t, x_1) - f(t, x_2)\| \leq C_K \|x_1 - x_2\|,$$

for every $(t,x_1),(t,x_2)\in K$. *If there is a constant for which the inequality holds for all* $(t,x_1),(t,x_2)\in\Omega$, *then* f *is said to be Lipschitz continuous in* x.

Lemma 7 *([8]) If* $f:\Omega\to\mathbb{R}^n$ *is* C^1, *then it is locally Lipschitz continuous in* x.

Theorem 8 *(Picard [8]). Let* $\Omega\subset\mathbb{R}^{n+1}$ *be open. Assume that* $f:\Omega\to\mathbb{R}^n$ *is continuous and that* $f(t,x)$ *is locally Lipschitz continuous in* x. *Let* $K\subset\Omega$ *be any compact set. Then there is a* $\delta>0$ *such that for every* $(t_0,x_0)\in K$, *the initial value problem (2.1) has a unique local solution defined on the interval* $|t-t_0|<\delta$.

Theorem 9 *(Uniqueness). Suppose that* $f:\Omega\to\mathbb{R}^n$ *satisifes the hypotheses of the Picard Theorem. For* $j=1,2$, *let* $x_j(t)$ *be solutions of* $x'(t)=f(t,x(t))$ *on the interval* I_j. *If there is a point* $t_0\in I_1\cap I_2$ *such that* $x_1(t_0)=x_2(t_0)$, *then* $x_1(t)=x_2(t)$ *on the interval* $I_1\cap I_2$. *Moneover, the function*

$$x(t)=\begin{cases}x_1(t), & t\in I_1\\ x_2(t), & t\in I_2\end{cases}$$

defines a solution on the interval $I_1\cup I_2$.

Lemma 10 *(Gronwall([8])). Let* $f(t),\varphi(t)$ *be nonnegative continuous functions on an open interval* $J=(\alpha,\beta)$ *containing the point* t_0. *Let* $c_0\geq 0$. *If*

$$f(t)\leq c_0+\left|\int_{t_0}^{t}\varphi(s)f(s)ds\right|$$

for all $t\in J$, *then*

$$f(t)\leq c_0\exp\left|\int_{t_0}^{t}\varphi(s)ds\right|$$

for $t\in J$.

Proof 11 *Suppose first that $t \in [t_0, \beta)$. Define*

$$F(t) = c_0 + \int_{t_0}^{t} \varphi(s) f(s) ds.$$

Then F is C^1 and

$$F'(t) = \varphi(t) f(t) \leq \varphi(t) F(t)$$

for $t \in [t_0, \beta)$, since $f(t) \leq F(t)$. This implies that

$$\frac{d}{dt}\left[\exp\left(-\int_{t_0}^{t} \varphi(s) ds\right) F(t)\right] \leq 0,$$

for $t \in [t_0, \beta)$. Integrate this over the interval $[t_0, \tau)$ to get

$$f(\tau) \leq F(\tau) \leq c_0 \exp \int_{t_0}^{\tau} \varphi(s) ds$$

for $\tau \in [t_0, \beta)$.
On the interval $(\alpha, t_0]$, perform the analogous argument to the function

$$G(t) = c_0 + \int_{t}^{t_0} \varphi(s) f(s) ds$$

2.2.2 Stability of equiliberia

Let $\Omega = \mathbb{R} \times O$ for some open set $O \subset \mathbb{R}^n$ and the suppose that $f : \Omega \to \mathbb{R}^n$ satisfy the Picard theorem hypothesis .

Definition 12 *A point $\overline{x} \in O$ is called an equiliberium point if $f(t\ , \overline{x}) = 0$ for each $t \in \mathbb{R}$.*

Let the differential equation

$$\dot{x} = f(x), \tag{2.2}$$

where $f : \Omega \subset \mathbb{R}^n \rightarrow \mathbb{R}^n$ is a function of class C^1. Let x^* an equiliberium point of the $eq(2.2)$

Definition 13 *([9]) The equiliberium point x^* of (2.2) is stable if for all $\varepsilon > 0$, there exist $\eta > 0$ for every solution $x(t)$ of (2.2) we have*

$$\|x(0) - x^*\| < \eta \Rightarrow \forall t \geq 0, \quad \|x(t) - x^*\| < \varepsilon.$$

Definition 14 *([9]) The equiliberium point x^* of (2.2) is stable if for all $\varepsilon > 0$, there exist $\eta > 0$, such there exist a solution $x(t)$ of (2.2) verify*

$$\|x(0) - x^*\| < \eta \Rightarrow \forall t \geq 0, \quad \|x(t) - x^*\| \geq \varepsilon.$$

Definition 15 *([9]) The equiliberium point x^* of (2.2) is asymptotically stable if it is stable and there is a $r > 0$ such that for every solution $x(t)$ of (2.2) we have*

$$\|x(0) - x^*\| < r \Rightarrow \lim_{t \to \infty} \|x(t) - x^*\| = 0.$$

Case of linear system

Consider the linear system

$$\dot{x} = Ax \tag{2.3}$$

where A is a square matrix of order n . $\lambda_1, ..., \lambda_s$ (*with* $s = 1, ..., n$) the eigenvalues of the matrix A and x^* the equilibrium point of linear system (2.3) .

Theorem 16 *([9]*

1) If the eigenvalues of the matrix A have a zeros or negatives real parts then the equilibrium x^* is stable .

2) If the eigenvalues of the matrix A have strictly negative real part then the equilibrium x^* is asymptotically stable .

3) If the real part of the matrix A have at least one eigenvalue is positive then the equilibrium x^* is unstable

Consider the system (2.2), we denote by $J_f(x^*) := \frac{\partial f}{\partial x}(x^*)$ the jaccobian matrix of f evaluated at the point x^*.

The linear system

$$\dot{x} = Ax,$$

where $A = J_f(x^*)$, is called the linearized or the linear approximation of the nonlinear system in x^*.

The study of the stability of the origin for the linearized allows in certain cases to characterize the stability of the equilibrium $x^*$of (2.2) . More precisely we have:

Theorem 17 *([9]*

1) If $x = 0$ is asymptotically stable ((i.e. if all the eigenvalues have a strictly negative real part)for (2.3) then x^* is asymptotically stable for (2.3).

2) If $x = 0$ is unstable (i.e there exist at least one eigenvalue which his real part is strictly positive) for (2.3) then x^* is unstable for (2.3).

3) In all other cases nothing can be said about the stability of x^* for(2.3).

2.3 Lyapunov stability

2.3.1 Stability of equilibria

Let the differential equation

$$\dot{x} = f(x), \tag{2.4}$$

where $f : \Omega \subset \mathbb{R}^n$ is a given function , and Ω is an open set from $\mathbb{R}^n$such that $0 \in \Omega$, and $f(0) = 0$.

Let $x^* = 0$ an equilibrium point of (2.5), and $V : \Omega \to \mathbb{R}^n$ a function defined in a neighborhood Ω of the origin and admitting continuous partial derivatives.

Lemma 18 *([10]) Let V be a nonnegative function defined in a neighborhood $V \subset U$ of the origin. Suppose that $\dot{V}(x) = (d/dt)\left(V\left(X_t(x)\right)\right)|_{t=0} \leq 0$ for all $x \in V$, then M_0 is positively invariant and $M_0 \subset M^*$.*

Proof 19 *Since the function V decreases along the trajectories of (2.1) , we have*

$$0 \leq V\left(X_t(x)\right) \leq V(x).$$

So if $x \in M_0$, then $0 \leq V\left(X_t(x)\right) \leq V(x) = 0$ for all nonnegative t, which proves that M_0 is positively invariant. Moreover,

$$\dot{V}(x) = \frac{d}{dt}\left(V\left(X_t(x)\right)\right)\Big|_{t=0} = 0.$$

So $M_0 \subset M$.

Theorem 20 *(Stability in the sense of Lyapunov [10]) If there exist a neighborhood* $V \subset U$ *of the origin and a function* $V \in C^1(V, \mathbb{R})$ *such that*
(1) $V(x) \geq 0$ *for all* $x \in V$ *and* $V(0) = 0$,
(2) $\dot{V}(x) = X \cdot V(x) \leq 0$ *for all* $x \in V$, *and*
(3) there exists $\Delta > 0$ *such that* $B_\Delta \cap \{x \in V : V(x) = 0\}$ *does not contain any negative orbit except the trivial one* $x \equiv 0$,
then the origin is Lyapunov stable.

Proof 21 *Suppose that the origin is not stable. Then there exist* $\varepsilon > 0$, *a sequence*

$$(x_{0n})_{n\in\mathbb{N}} \subset B_\varepsilon, \lim_{n\to\infty} \|x_{0n}\| = 0,$$

and a sequence $(t_n)_{n\in\mathbb{N}} \subset \mathbb{R}^+$*in such a way that*

$$\begin{cases} \|X_t(x_{0n})\| < \varepsilon & \text{for} \quad 0 \leq t < t_n, \\ \|X_{t_n}(x_{0n})\| = \varepsilon, & \forall n \in \mathbb{N}. \end{cases}$$

S_ε *is compact so without loss of generality we can assume that the sequence* $y_n = X_{t_n}(x_{0_n})$ *tends to* $y \in S_\varepsilon$. *On the other hand, because of the continuity of solutions of (1),* $t_n \to +\infty$ *as* $n \to +\infty$.
First we show that $\gamma^-(y) \subset \overline{B_\varepsilon}\backslash\{0\}$. *Ad absurdum, suppose that there exists* $\tau < 0$ *such that* $\|X_\tau(y)\| > \varepsilon$. *We choose* $\mu > 0$ *in such a way* $0 < \mu < \frac{1}{2}(\|X_\tau(y)\| - \varepsilon)$. *The solutions of (1) are continuous functions of the initial conditions so we can find a positive number* $v = v(\mu) > 0$ *such that*

$$\|y - z\| < v \Rightarrow \|X_\tau(y) - X_\tau(z)\| < \mu.$$

The sequence $y_n = X_{t_n}(x_{0n})$ *tends to* y *so there exists* $N \subset \mathbb{N}$ *such that*

$$\|y - y_n\| < v, \quad \forall n \geq N.$$

Thus we have

$$\|X_\tau(y) - X_\tau(y_n)\| < \mu, \quad \forall n \geq N.$$

We can choose the integer N *sufficiently large in such a way that* $0 < t_n + \tau$, $\forall n \geq N$

(since $t_n \to +\infty$).

$$d\left(X_\tau\left(y_n\right), \overline{B_\varepsilon}\right) \geq d\left(X_\tau(y), \overline{B_\varepsilon}\right) - d\left(X_\tau\left(y_n\right), X_\tau(y)\right) \geq d\left(X_\tau(y), \overline{B_\varepsilon}\right) - \mu, \quad \forall n \geq N$$

so

$$d\left(X_\tau\left(y_n\right), \overline{B_\varepsilon}\right) = d\left(X_{t_n+\tau}\left(x_{0_n}\right), \overline{B_\varepsilon}\right) > 0, \quad \forall n \geq N,$$

which means $\|X_{t_n+\tau}(x_{0n})\| > \varepsilon$ for all $n \geq N$ but this contradicts (2) because $0 < t_n + \tau < t_n$. So we have proved that $\gamma^-(y) \subset \overline{B_\varepsilon}\backslash\{0\}$ and $\|y\| = \varepsilon$.
Now we show that $V(\gamma^-(y)) = 0$; let τ be any negative number, there exists $n \in \mathbb{N}$ such that $t_n + \tau \geq 0\,(t_n \to +\infty)$. So, using hypothesis (1) and (2) of theorem , we have

$$0 \leq V\left(X_{t_n+\tau}\left(x_{0n}\right)\right) \leq V\left(x_{0n}\right).$$

Moreover, $\lim_{n\to\infty} x_{0n} = 0$, so by continuity of V and by (1) of theorem (2.6) we have

$$\lim_{n\to\infty} V\left(X_{t_n+\tau}\left(x_{0n}\right)\right) = \lim_{n\to\infty} V\left(X_\tau\left(X_{t_n}\left(x_0\right)\right)\right) = V\left(X_\tau(y)\right) = 0.$$

Thus we have proved that $V(\gamma^-(y)) = 0$ which ends the proof of Theorem (2.6).

Theorem 22 *(Asymptotic stability in the sense of Lyapunov[10])If there exist a neighborhood $V \subset U$ of the origin and a function $V \in C^1(V, \mathbb{R})$ such that*
(1) $V(x) \geq 0$ for all $x \in V$ and $V(0) = 0$,
(2) $\dot{V}(x) = X \cdot V(x) \leq 0$ for all $x \in V$, and
(3) there exists $\Delta > 0$ such that $B_\Delta \cap \{x \in V : \dot{V}(x) = 0\}$ does not contain any negative orbit except the trivial one $x \equiv 0$,
then the origin is asymptotically stable.

Proof 23 *By Lemma 1 the set $M_0 = \{x \in V : V(x) = 0\}$ is contained in $M = \{x \in V : \dot{V}(x) = 0\}$, so by Theorem (2.6) the origin is stable, that is, for any positive ε there exists a positive number δ such that any solution of (1) which starts in B_δ remains in B_ε for all positive t. Suppose the origin is not attractive, that is, there exists $x_0 \in B_\delta$ in such a way that $L^+(x_0) \neq \{0\}$. Let $y \in L^+(x_0)$ with $y \neq 0$, because of the invariance of $L^+(x_0), \gamma^-(y)$ is contained in $L^+(x_0)$ which is contained in B_ε. On the other hand,*

LaSalle's invariance principle implies that $L^+(x_0) \subset M$, *so* $\gamma^-(y) \subset M \cap B_\varepsilon$, *which contradicts hypothesis (3) of Theorem (2.7).*

Theorem 24 *(Lyapunov[11]) Let* $\dot{\mathbf{x}} = \mathbf{f}(\mathbf{x})$ *be a time-independent ODE defined on some subset* G *of* $\mathbb{R}^n$. *Let* $V : G \to \mathbb{R}$ *be continuously differentiable. If for some solution* $\mathbf{x}(t)$, *the derivative* $\dot{V}$ *of the map* $t \to V(\mathbf{x}(t))$ *satisfies the inequality* $\dot{V} \geq 0$ *(or* $\dot{V} \leq 0$ *), then* $\omega(\mathbf{x}) \bigcap G$ *is contained in the set* $\{\mathbf{x} \in G : \dot{V}(\mathbf{x}) = 0\}$ *(and so is* $\alpha(\mathbf{x}) \cap G$*).*

Proof 25 *If* $\mathbf{y} \in \omega(\mathbf{x}) \cap G$, *there is a sequence* $t_k \to +\infty$ *with* $\mathbf{x}(t_k) \to \mathbf{y}$. *Since* $\dot{V} \geq 0$ *along the orbit of* $\mathbf{x}$, *one has* $\dot{V}(\mathbf{y}) \geq 0$ *by continuity. Suppose that* $\dot{V}(\mathbf{y}) = 0$ *does not hold. Then* $\dot{V}(\mathbf{y}) > 0$. *Since the value of* V *can never decrease along an orbit, this implies*

$$V(\mathbf{y}(t)) > V(\mathbf{y}_0), \tag{2.5}$$

for $t > 0$. *The function* $V(x(t))$ *is also monotonically increasing. Since* V *is continuous,* $V(\mathbf{x}(t_k))$ *converges to* $V(\mathbf{y})$, *and hence*

$$V(\mathbf{x}(t)) \leq V(\mathbf{y}), \tag{2.6}$$

for every $t \in \mathbb{R}$. *From* $\mathbf{x}(t_k) \to \mathbf{y}$ *it follows that* $\mathbf{x}(t_k + t) \to \mathbf{y}(t)$ *and hence*

$$V(\mathbf{x}(t_k + t)) \to V(\mathbf{y}(t))$$

so that by (2.5)

$$V(\mathbf{x}(t_k + t)) > V(\mathbf{y})$$

for k *sufficiently large. This contradicts* (2.6)

2.3.2 Stability of matrix

We write a matrix $A > 0 (< 0)$ if A is symmetric positive (negative) definite. The following fundamental result on matrix stability was originally proved by Lyapunov.

Lemma 26 *[4]. Let* A *be an* $n \times n$ *real matrix. Then, all the eigenvalues of* A *have negative (positive) real parts if and only if there exists a matrix* $H > 0$, *such that*

$HA + A^T H^T < 0 (> 0)$.

Definition 27 *We say a nonsingular $n \times n$ matrix A is $Volterra - Lyapunov$ stable if there exists a positive diagonal $n \times n$ matrix M, such that $MA + A^T M^T < 0$.*

The following lemma determines all 2×2 $Volterra - Lyapunov$ stable matrices.

Lemma 28 *[5]. Let $D = \begin{bmatrix} d_{11} & d_{12} \\ d_{21} & d_{22} \end{bmatrix}$ be a 2×2 matrix. Then D is $Volterra - Lyapunov$ stable if and only if $d_{11} < 0, d_{22} < 0$, and $\det(D) = d_{11}d_{22} - d_{12}d_{21} > 0$.*

The characterization of $Volterra - Lyapunov$ stable matrices of higher dimensions, however, is much more difficult. We need the following definition.

Definition 29 *We say a nonsingular $n \times n$ matrix A is diagonally stable (or positive stable) if there exists a positive diagonal $n \times n$ matrix M, such that $MA + A^T M^T > 0$.*

From Definitions (2.10) and (2.11), it is clear that a matrix A is $Volterra - Lyapunov$ stable if and only if its negative matrix, $-A$, is diagonally stable.

Notation 30 *For any $n \times n$ matrix A, let $\widetilde{A}$ denote the $(n-1) \times (n-1)$ matrix obtained from A by deleting its last row and last column.*

The following generalized result was obtained by Redheffer (1985a, b) which will be frequently used in our global stability analysis. For simplicity, we only state the sufficient condition below .

Lemma 31 *[7] Let $D = [d_{ij}]$ be a nonsingular $n\times n$ matrix $(n \geq 2)$ and $M = \operatorname{diag}(m_1, \ldots, m_n)$ be a positive diagonal $n \times n$ matrix. Let $E = D^{-1}$. Then, if $d_{nn} > 0, \widetilde{ME} + (\widetilde{ME})^T > 0$, and $\widetilde{MD} + (\widetilde{MD})^T > 0$, it is possible to choose $m_n > 0$, such that $MD + D^T M^T > 0$.*

Lemma 32 *[6]. Consider a disease model system written in the form:*

$$\begin{cases} \frac{dX_1}{dt} = F(X_1, X_2) \\ \frac{dX_1}{dt} = G(X_1, X_2) \\ G(X_1, 0) = 0, \end{cases} \tag{2.7}$$

where $X_1 \in \mathbb{R}^m$ denotes (by its components) the uninfected populations and $X_2 \in \mathbb{R}^n$ denotes (by its components) the infectious populations; $X_0 = \left(X_1^E, 0\right)$ denotes the diseasefree equilibrium of the system.

In addition, assume the conditions (C_1) and (C_2) below:

- C_1 : For $\frac{dX_1}{dt} = F\left(X_1, 0\right), X_1^E$ is globally asymptotically stable;

- $C_2 : G\left(X_1, X_2\right) = AX_2 - \hat{G}\left(X_1, X_2\right)$, with $\hat{G}\left(X_1, X_2\right) \geq 0$ for $\left(X_1, X_2\right) \in \Omega$, where the Jacobian matrix $A = \frac{\partial G}{\partial X_2}\left(X_1^E, 0\right)$ has all non-negative off-diagonal elements and X is the region where the model makes biological sense.

CHAPTER 3

STABILITY OF COMPUTER VIRUSES MODEL

3.1 Introduction

In this chapter, we are interested in a mathematical model of computer virus proposed by Yang et al.[12] . A computer is classified as internal and external depending on weather it is connected to internet or not. In this model, assumes that only internal computers are concerned, and all internal computers are categorized into three classes: uninfected computers (i.e., virus-free computers), infected computers that are currently latent (latent computers, for short), and infected computers that are currently breaking out (seizing computers, for short). Due to the fact that in the future, the total amount of computers in the world would tend to saturation, it is reasonable to suppose that this total number is constant. Let $S(t)$, $L(t)$, and $B(t)$ denote, at time t, the percentages of uninfected, latent, and seizing computers in all internal computers, respectively. Then, $S(t)+L(t)+B(t) = 1$ Unless otherwise stated, let S, L, and B stand for $S(t)$, $L(t)$, and $B(t)$, respectively.

The mathematical model of the transmission of Computer Virus is described by the following system of differential equations :

$$\begin{cases} \frac{dS}{dt} = \delta - \beta S(L+B) + \gamma_1 L + \gamma_2 B - \delta S, \\ \frac{dL}{dt} = \beta S(L+B) - \gamma_1 L - \alpha L - \delta L, \\ \frac{dB}{dt} = \alpha L - \gamma_2 B - \delta B, \end{cases} \tag{3.1}$$

with the initial conditions

$$S(0) > 0, L(0) > 0, B(0) > 0. \tag{3.2}$$

The coefficients $\delta, \alpha, \beta, \gamma_1, \gamma_2$ are positives and they are explained in the following :

δ : the rate of a computer leaving the internet

α : the breaking rate

β : the incidence rate

γ_1 : the recovred rate

γ_2 : the rate of an infected computers reinstalling the operating system

3.2 Existence , positivity and boundedness

3.2.1 Existence

Let $x = (S, L, B)^T$. The system (3.1) become

$$\begin{cases} x^{'}(t) = g(x(t)), \\ x(0) = (S_0, L_0, B_0)^T, \end{cases}$$

where

$$g(x) = \begin{pmatrix} \delta - \beta S(L+B) + \gamma_1 L + \gamma_2 B - \delta S \\ \beta S(L+B) - \gamma_1 L - \alpha L - \delta L \\ \alpha L - \gamma_2 B - \delta B \end{pmatrix},$$

the function g is globally Lipschitz , according to the theorem of Cauchy–Lipschitz, the global existence of the solutions is ensured.

3.2.2 Positivity

Since the second member of the equations of the system (3.1) are polynomes , then the system (3.1) is quasipositive, if the condition

$$x \geq 0, x_k = 0 \Rightarrow g_k(x) \geq 0,$$

∞

where $g = (g_1, ..., g_k)$ for every $k = 1, 2, 3$. So the solutions of (3.1), (3.2) with initial condition $x(0) \in \mathbb{R}^3_+$ stay in $\mathbb{R}^3_+$ for all t positive.

3.2.3 Boundedness

We will show that the solution of system (3.1) is borned .

Let $(S(t), L(t), B(t))$ the solution of (3.1) with the initial conditions (3.2) , and $(0, T)$ the maximal interval of existence of the solution.

We pose

$$N(t) = S(t) + L(t) + B(t),$$

then

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dL}{dt} + \frac{dB}{dt}.$$

we obtain

$$\frac{dN}{dt} = \delta\left(1 - B - L - S\right).$$

Hence

$$\limsup_{t\to\infty} N \leq \delta.$$

3.3 Basic repoduction number

In this mathematical model , the basic reproduction number, is defined as the number of previously uninfected computers that are infected by a single infected computer during its life cycle, can be calculated as follow :

Let

$$\begin{cases} F_1 = \delta - \beta S(L+B) + \gamma_1 L + \gamma_2 B - \delta S, \\ F_2 = \beta S(L+B) - \gamma_1 L - \alpha L - \delta L, \\ F_3 = \alpha L - \gamma_2 B - \delta B. \end{cases} . \tag{3.3}$$

We extract the Jacobian Matrix where

$$J = \begin{pmatrix} \frac{dF_1}{dS} & \frac{dF_1}{dL} & \frac{dF_1}{dB} \\ \frac{dF_2}{dS} & \frac{dF_2}{dL} & \frac{dF_2}{dB} \\ \frac{dF_3}{dS} & \frac{dF_3}{dL} & \frac{dF_3}{dB} \end{pmatrix}, \tag{3.4}$$

by compensation we find

$$J = \begin{pmatrix} -\delta - B\beta - L\beta & \gamma_1 - S\beta & \gamma_2 - S\beta \\ \beta(B+L) & S\beta - \delta - \gamma_1 - \alpha & S\beta \\ 0 & \alpha & -\delta - \gamma_2 \end{pmatrix}, \tag{3.5}$$

Evaluating the Jacobian matrix (3.5) at E_0 yields

$$J(E_0) = \begin{pmatrix} -\delta & \gamma_1 - \beta & \gamma_2 - \beta \\ 0 & \beta - \delta - \gamma_1 - \alpha & \beta \\ 0 & \alpha & -\delta - \gamma_2 \end{pmatrix},$$

where

$$w = \begin{pmatrix} -\alpha - \gamma_1 & \beta \\ \alpha & -\delta - \gamma_2 \end{pmatrix}, \tag{3.6}$$

and

$W = F - V = \begin{pmatrix} \beta & \beta \\ 0 & 0 \end{pmatrix} - \begin{pmatrix} \gamma_1 + \alpha + \beta & 0 \\ -\alpha & \gamma_2 + \delta \end{pmatrix},$

where F and V are the 2×2 matrices defined by

$$F(E_0) = \begin{pmatrix} \beta & \beta \\ 0 & 0 \end{pmatrix}$$

and

$$V(E_0) = \begin{pmatrix} \gamma_1 + \alpha + \beta & 0 \\ -\alpha & \gamma_2 + \delta \end{pmatrix}$$

The conditions listed above allow us to partition the matrix $J(E_0)$ as shown by the following

$$R_0 = \rho(FV^{-1}) = \max(|\boldsymbol{\lambda}_1|, |\boldsymbol{\lambda}_2|) \tag{3.7}$$

$$V^{-1} = \frac{1}{\det(V)} \left(\tilde{V}\right)^t, \tag{3.8}$$

we count $\left(\tilde{V}\right)^t$:

$$\left(\tilde{V}\right)^t = \begin{pmatrix} \delta + \gamma_2 & 0 \\ \alpha & \alpha + \beta + \gamma_1 \end{pmatrix}, \tag{3.9}$$

which has determinant

$$\det(V) = (\delta + \gamma_2)(\alpha + \beta + \gamma_1). \tag{3.10}$$

Using (3.9) and (3.10), this can be rewritten as

$$V^{-1} = \frac{1}{(\delta + \gamma_2)(\alpha + \beta + \gamma_1)} \begin{pmatrix} \delta + \gamma_2 & 0 \\ \alpha & \alpha + \beta + \gamma_1 \end{pmatrix}, \tag{3.11}$$

then

$$\begin{aligned} FV^{-1} &= \frac{1}{(\delta + \gamma_2)(\alpha + \beta + \gamma_1)} \begin{pmatrix} \beta & \beta \\ 0 & 0 \end{pmatrix} \begin{pmatrix} \delta + \gamma_2 & 0 \\ \alpha & \alpha + \beta + \gamma_1 \end{pmatrix} \\ &= \begin{pmatrix} \frac{\beta}{\alpha+\beta+\gamma_1} + \alpha\frac{\beta}{(\delta+\gamma_2)(\alpha+\beta+\gamma_1)} & \frac{\beta}{\delta+\gamma_2} \\ 0 & 0 \end{pmatrix} \end{aligned}$$

$$= \begin{pmatrix} \beta\frac{\alpha+\delta+\gamma_2}{(\delta+\gamma_2)(\alpha+\beta+\gamma_1)} & \frac{\beta}{\delta+\gamma_2} \\ 0 & 0 \end{pmatrix}, \tag{3.12}$$

Using (3.12), we obtain

$$det(FV^{-1} - \lambda I_2) = \begin{pmatrix} \beta\frac{\alpha+\delta+\gamma_2}{(\delta+\gamma_2)(\alpha+\beta+\gamma_1)} - \lambda & \frac{\beta}{\delta+\gamma_2} \\ 0 & -\lambda \end{pmatrix},$$

$\boldsymbol{\lambda}_{i,i=\overline{1,2}}$ are the eignvalue

$$\begin{cases} \lambda_1 = \beta\frac{\alpha+\delta+\gamma_2}{(\delta+\gamma_2)(\alpha+\beta+\gamma_1)} \\ \lambda_2 = 0 \end{cases}. \tag{3.13}$$

Then, the basic reproduction number is defined, as the spectral radius of the next generation matrix, FV^{-1} :

$$R_0 = \rho(FV^{-1}) = \max(|\lambda_1|, |\lambda_2|) = |\lambda_1|$$
$$R_0 = \beta\frac{\alpha+\delta+\gamma_2}{(\delta+\gamma_2)(\alpha+\beta+\gamma_1)}.$$

3.4 Equilibrium Points

The endemic equilibrium point $E_* = (S^*, L^*, B^*)$ the system (3.1) as follows:

$$\begin{cases} \delta - \beta S^* \left(L^* + B^*\right) + \gamma_1 L^* + \gamma_2 B^* - \delta S^* = 0 \\ \beta\delta \left(L^* + B^*\right) - \gamma_1 L^* - \alpha L^* - \delta L^* = 0 \\ \alpha L^* - \gamma_2 B^* - \delta B^* = 0 \end{cases}, \tag{3.14}$$

by adding the seconde equation to the third one ze get

$$\delta - L^*\alpha - L^*\delta - S^*\delta + B^*\gamma_2 = 0, \tag{3.15}$$

from the seconde eauqtion we get

$$\beta S^*(L^* + B^*) = (\gamma_1 + \alpha + \delta)L^*, \tag{3.16}$$

from the third equation we get

$$L^* = \frac{(\gamma_2 + \delta)}{\alpha} B^*, \tag{3.17}$$

substitute L^* in (3.15)

$$\delta - \left(\frac{(\gamma_2 + \delta)}{\alpha} B^*\right) \alpha - \left(\frac{(\gamma_2 + \delta)}{\alpha} B^*\right) \delta - S^*\delta + B^*\gamma_2 = 0,$$

we get

$$\delta - \delta B^* - \delta S^* - B^*\delta \frac{\delta + \gamma_2}{\alpha} = 0 \tag{3.18}$$

substitute L^* in (3.16)

$$\beta S^* \left(\frac{(\gamma_2 + \delta)}{\alpha} B^*\right) + B^*) = (\gamma_1 + \alpha + \delta) \left(\frac{(\gamma_2 + \delta)}{\alpha} B^*\right),$$

we get

$$S^* = \frac{(\delta + \gamma_2)(\alpha + \delta + \gamma_1)}{\beta(\alpha + \delta + \gamma_2)} = \frac{1}{R_0}. \tag{3.19}$$

From (3.18) we have

$$B^* = \frac{\alpha(1 - S^*)}{(\alpha + \delta + \gamma_2)}, \tag{3.20}$$

substitute S^* in (3.20)

$$B^* = \frac{\alpha\left(1 - \frac{1}{R_0}\right)}{(\alpha + \delta + \gamma_2)},$$

we get

$$B^* = \frac{\alpha\,(R_0 - 1)}{R_0\,(\alpha + \delta + \gamma_2)}. \tag{3.21}$$

Substitute B^* in (3.17)

$$L^* = \frac{(\gamma_2 + \delta)}{\alpha}\left(\frac{\alpha\,(R_0 - 1)}{R_0\,(\alpha + \delta + \gamma_2)}\right),$$

we get

$$L^* = \frac{(\delta + \gamma_2)\,(R_0 - 1)}{R_0\,(\alpha + \delta + \gamma_2)}. \tag{3.22}$$

So the value of endemic equilibrium point $E_*{=}\,(S^*, L^*, B^*)$ is

$$\begin{cases} \mathrm{S}^*{=}\dfrac{1}{R_0}{=}\dfrac{(\delta + \gamma_2)\,(\alpha + \delta + \gamma_1)}{\beta\,(\alpha + \delta + \gamma_2)} \\ \mathrm{B}^*{=}\dfrac{\alpha\,(R_0 - 1)}{R_0\,(\alpha + \delta + \gamma_2)} \\ \mathrm{L}^*{=}\dfrac{(\delta + \gamma_2)\,(R_0 - 1)}{R_0\,(\alpha + \delta + \gamma_2)} \end{cases}. \tag{3.23}$$

If $B^* = 0$, then from (3.17) we get

$$L^* = 0,$$

and we get from the first equation of the system (3.1)

$$S^* = 1.$$

So the value of disease-free equilibrium is

$$E_0{=}\,(1, 0, 0)$$

3.5 Global stability of equilibrium points

3.5.1 Global stability of the DFE

In this section we will study the global stability of the disease-free equilibrium of the model (3.1).

Theorem 33 *If $R_0 < 1$, then the disease-free equilibrium f system (3.1) $E_0 = (1, 0, 0)$ is*

a globally asymptotically stable

Proof 34 *We will use the theorem by Castillo-Chavez [6] to prove the global stability result.*

Applying Lemma 2.7 to system(3.1) , consider $X_1 = S, X_2 = \begin{bmatrix} L \\ B \end{bmatrix}$.
When $L = B = 0$, *the uninfected subsystem (i.e., the equation for* S *) becomes*

$$\frac{dS}{dt} = \delta - \delta S = \delta (1 - S)$$

integrate the both side

$$\int \frac{1}{1-S} dS = \int \delta dt$$

we pose $I_1 = \int \frac{1}{1-S} dS$ *and* $I_2 = \int \delta dt$
starting by integrating I_1*:*

$$I_1 = \int \frac{1}{1-S} dS,$$

apply u-substitution

$$I_1 = \int -\frac{1}{u} dS,$$

take the constante out

$$I_1 = -\int \frac{1}{u} dS,$$

use the common integral : $\int \frac{1}{u} dS = \ln(|u|)$

$$I_1 = -\ln(u),$$

substitute back $u = 1 - S$

$$I_1 = -\ln|1 - S|,$$

add constante to solution

$$I_1 = -\ln|1 - S| + c_1.$$

Now integration $I_2 = \int \delta dt$

$$\begin{aligned} I_2 &= \int \delta dt, \\ &= \delta t, \end{aligned}$$

add constante to solution

$$I_2 = \delta t + c_{2,}$$

now we take $I_1 = I_2$

$$\begin{aligned} \ln|S-1| + c_1 &= \delta t + c_2 \\ \ln|S-1| &= \delta t + c_2 - c_1, \end{aligned}$$

we pose $c = c_2 - c_1$

$$\ln|S-1| = \delta t + c,$$

by exponents both side

$$\begin{aligned} e^{\ln|S-1|} &= e^{\delta t + c}, \\ S-1 &= e^{\delta t} e^{c}, \end{aligned}$$

we pose $C = e^C$

$$S - 1 = e^{\delta t} C,$$

for $t = 0$

$$S(0) - 1 = e^{\delta(0)} C,$$

we get

$$C = S(0) - 1,$$

finally we

$$S(t) = e^{-\delta t}\ (S(0) - 1) + 1$$

obviously, $S(t) \to 1$ *as* $t \to \infty$ *regardless of the initial value* $S(0)$*. Therefore, it shows that condition* (C_1) *in Lemma* 3.2 *holds for our model.*
Next, the right-hand side of the infectious subsystem (i.e., the equations for L *and* B *) can be written as*

$$
\begin{aligned}
\frac{dX2}{dt} &= G(X1, X2) \\
&= \begin{bmatrix} \beta BS - L\left(-\alpha - \delta - \gamma_1 + \beta S\right) \\ \alpha L - B\left(\delta + \gamma_2\right) \end{bmatrix} \\
&= \begin{bmatrix} \beta BS - L\left(-\alpha - \delta - \gamma_1 + \beta S\right) \\ \alpha L - B\left(\delta + \gamma_2\right) \end{bmatrix} \\
&= \begin{bmatrix} \beta\left(-\alpha - \delta - \gamma_1\right) & \beta \\ \alpha & -\left(\delta + \gamma_2\right) \end{bmatrix} \begin{bmatrix} L \\ B \end{bmatrix} - \begin{bmatrix} \beta SL + \beta L - \beta SB + \beta B \\ 0 \end{bmatrix} \\
&= AX_2 - \hat{G}(X1, X2)
\end{aligned}
$$

where

$$
A = \begin{bmatrix} \beta\left(-\alpha - \delta - \gamma_1\right) & \beta \\ \alpha & -\left(\delta + \gamma_2\right) \end{bmatrix} \text{ and } \hat{G}(X1, X2) = \begin{bmatrix} \beta SL + \beta L - \beta SB + \beta B \\ 0 \end{bmatrix}
$$

It is obvious that, $S \leq 1$*, hence, it is clear that condition* (C_2) *holds for our model. We also notice that the matrix* A *is an* M *matrix, since all its off-diagonal elements are non-negative.*

Hence, this proves the global stability of the DFE (E_0).

3.5.2 Global stability of endemic equiliberium

In this section we will study the global stability of the equilibrium point E_1 of the model (3.1).

Theorem 35 *Assume* $R_0 > 1$ *Then, the endemic equilibrium* $E_1 = (S^*, L^*, B^*)$ *is globally asymptotically stable.*

Proof 36 *To prove global stability result, we propose the following Lyapunov function:*

$$
V = w_1\left(S - S^*\right)^2 + w_2\left(L - L^*\right)^2 + w_3\left(B - B^*\right)^2, \tag{3.24}
$$

where w_1, w_2*, and* w_3 *are positive constants. Calculating the time derivative of* V *along*

the trajectories of the system (3.1), we obtain

$$\begin{aligned}\dot{V} =&2w_1\left(S-S^*\right)\dot{S}+2w_2\left(L-L^*\right)\dot{L}+2w_3\left(B-B^*\right)\dot{B}\\ \\ =&2w_1\left(S-S^*\right)\left[-\beta S(L+B)+\beta S^*\left(L^*+B^*\right)\right.\\ &\left.+\gamma_1\left(L-L^*\right)+\gamma_2\left(B-B^*\right)-\delta\left(S-S^*\right)\right]\\ &+2w_2\left(L-L^*\right)\left[\beta S(L+B)-\beta S^*\left(L^*+B^*\right)\right.\\ &\left.-\gamma_1\left(L-L^*\right)-\alpha\left(L-L^*\right)-\delta\left(L-L^*\right)\right]\\ &+2w_3\left(B-B^*\right)\left[\alpha\left(L-L^*\right)-\gamma_2\left(B-B^*\right)-\delta\left(B-B^*\right)\right]\end{aligned}$$

Then, we add the expression $\beta S'L$ and $\beta S'B$ into the first and second square bracket. As a result, we obtain

$$\begin{aligned}\dot{V} =&2w_1\left(S-S^*\right)\left[-\beta SL-\beta SB+\beta S^*L^*+\beta S^*B^*+\beta S^*L\right.\\ &\left.-\beta S^*L+\beta S^*B-\beta S^*B+\gamma_1\left(L-L^*\right)+\gamma_2\left(B-B^*\right)\right.\\ &\left.-\delta\left(S-S^*\right)\right]+2w_2\left(L-L^*\right)\left[\beta SL+\beta SB-\beta S^*L^*\right.\\ &\left.-\beta S^*B^*+\beta S^*L-\beta S^*L+\beta S^*B-\beta S^*B-\gamma_1\left(L-L^*\right)\right.\\ &\left.-\alpha\left(L-L^*\right)-\delta\left(L-L^*\right)\right]+2w_3\left(B-B^*\right)\left[\alpha\left(L-L^*\right)\right.\\ &\left.-\gamma_2\left(B-B^*\right)-\delta\left(B-B^*\right)\right],\end{aligned}$$

therefore, we have

$$\begin{aligned}\dot{V} =&2w_1\left(S-S^*\right)\left[\left(-\beta L-\beta B-\delta\right)\left(S-S^*\right)+\left(-\beta S^*+\gamma_1\right)\right.\\ &\left.\left(L-L^*\right)+\left(-\beta S^*+\gamma_2\right)\left(B-B^*\right)\right]+2w_2\left(L-L^*\right)\\ &\left[\left(\beta L+\beta B\right)\left(S-S^*\right)+\left(\beta S^*-\gamma_1-\alpha-\delta\right)\left(L-L^*\right)\right.\\ &\left.+\beta S^*\left(B-B^*\right)\right]+2w_3\left(B-B^*\right)\left[\alpha\left(L-L^*\right)-\left(\gamma_2+\delta\right)\right.\\ &\left.\left(B-B^*\right)\right]=2w_1(-\beta L-\beta B)\left(S-S^*\right)^2\\ &+2w_1\left(-\beta S^*+\gamma_1\right)\left(S-S^*\right)\left(L-L^*\right)\\ &+2w_1\left(-\beta S^*+\gamma_2\right)\left(S-S^*\right)\left(B-B^*\right)]+2w_2(\beta L+\beta B)\\ &\left(L-L^*\right)\left(S-S^*\right)+2w_2\left(\beta S^*-\gamma_1-\alpha-\delta\right)\left(L-L^*\right)^2\\ &+2w_2\beta S^*\left(L-L^*\right)\left(B-B^*\right)+2w_3\alpha\left(L-L^*\right)\left(B-B^*\right)\\ &-2w_3\left(\gamma_2+\delta\right)\left(B-B^*\right)^2=Y\left(WP+P^TW^T\right)Y^T,\end{aligned}\tag{3.25}$$

where $Y = [S - S^*, L - L^*, B - B^*]$, $W = \text{diag}(w_1, w_2, w_3)$, *and*

$$P = \begin{bmatrix} -\beta L - \beta B - \delta & -\beta S^* + \gamma_1 & -\beta S^* + \gamma_2 \\ \beta L + \beta B & \beta S^* - \gamma_1 - \alpha - \delta & \beta S^* \\ 0 & \alpha & -\gamma_2 - \alpha \end{bmatrix}. \tag{3.26}$$

Theorem 37 *The matrix* P *defined in Eq (Equation 3.25) is* $Volterra - Lyapunov$ *stable*

To discuss the global asymptotic stability of $E_1 = (S^*, L^*, B^*)$ we proceed to show that the matrix P defined in Eq (3.26) is $Volterra - Lyapunov$ stable or $-P$ is diagonal stable. For this goal, we prove the following lemmas.

Lemma 38 *For the matrix* P *defined in eq. (3.25)* $-P$ *is diagonal stable.*

Proof 39 *To prove the diagonal stability of* $-P$ *and based on Lemma* 3.3 *, we need to show that the following three conditions are satisfied:*

- *Condition 1:*$-P_{33} > 0$
- *Condition 2:*$D = -\bar{P}$ *is diagonal stable*
- *Condition 3:*$E = -\widetilde{P^{-1}}$ *is diagonal stable*

1. *clearly* $-P_{33} > 0$
2. *Let us delete the last row and last column of matrix*$-P$ *and call it matrix*$-\bar{P}$ *It follows that*

$$D = -\tilde{P} = \begin{bmatrix} \beta L + \beta B + \delta & \beta S^* - \gamma_1 \\ -\beta L - \beta B & -\beta S^* + \gamma_1 + \alpha + \delta \end{bmatrix}$$

For this purpose, it is necessary to show that $-D$ *is* $Volterra - Lyapunov$ *stable:*

$$-D = \begin{bmatrix} -\beta L - \beta B - \delta & -\beta S^* + \gamma_1 \\ \beta L + \beta B & \beta S^* - \gamma_1 - \alpha - \delta \end{bmatrix}$$

Clearly, $-D_{11} < 0$. *Next, we show* $-D_{22} < 0$*; according to (3.14), we have*

$$\beta S^* (L^* + B^*) = (\gamma_1 + \alpha + \delta) L^*,$$

and it is obvious that

$$\beta S^* L^* \leq (\gamma_1 + \alpha + \delta) L^*,$$

hence, $-D_{22} < 0$. *Now, we show* $-D_{12} < 0$, *that is*

$$-\beta S^* + \gamma_1 < 0,$$

using (3.14), (3.21), we can see that

$$\beta S^* \left(L^* + B^*\right) - \left(\gamma_1 + \delta\right) L^* - \left(\gamma_2 + \delta\right) B^* = 0$$

since $0 < \gamma_1 < \gamma_2$, *we have*

$$\begin{aligned} \beta S^* \left(L^* + B^*\right) - \left(\gamma_1 + \delta\right) L^* - \left(\gamma_2 + \delta\right) B^* < \beta S^* \left(L^* + B^*\right) \\ - \left(\gamma_1 + \delta\right) L^* - \left(\gamma_1 + \delta\right) B^* \end{aligned}$$

therefore

$$\beta S^* \left(L^* + B^*\right) > \left(\gamma_1 + \delta\right) \left(L^* + B^*\right),$$

hence, $-D_{12} < 0$. *It is easy to see* $-D_{21} > 0$. *Therefore,* $-D$ *is* $Volterra - Lyapunov$ *stable based on Lemma* 2.5.

3. *We show that the matrix* $E = -\widetilde{P^{-1}}$ *is diagonal stable. In fact, we show that* $-E$ *is* $Volterra - Lyapunov$ *stable:*

$$-E = -\left(-\widetilde{P^{-1}}\right) = \frac{1}{\det(-P)} \begin{bmatrix} -E_{11} & -E_{12} \\ -E_{21} & -E_{22} \end{bmatrix}$$

where

$$\begin{aligned} -E_{11} &= -\left(\gamma_2 + \delta\right)\left(-\beta S^* + \gamma_1 + \alpha + \delta\right) + \alpha\beta S^*, \\ -E_{12} &= -\left(\gamma_2 + \delta\right)\left(-\beta S^* + \gamma_1\right) + \alpha\left(\beta S^* - \gamma_2\right), \\ -E_{21} &= -(\beta L + \beta B)\left(\gamma_2 + \delta\right). \\ -E_{22} &= -(\beta L + \beta B + \delta)\left(\gamma_2 + \delta\right). \end{aligned}$$

It is obvious that $-E_{21} < 0$ *and* $-E_{22} < 0$. *Below, we show* $-E_{11} = 0$ *and* $-E_{12} > 0$. *The* $(1,1)$ *entrie of this* $-E$ *is writen as*

$$-E_{11} = -\left(\gamma_2 + \delta\right)\left(-\beta S^* + \gamma_1 + \alpha + \delta\right) + \alpha\beta S^*,$$

multiplying the (3.14) by α, *and using (**??**) we have*

$$\beta S^* \alpha L^* + \beta S^* \alpha B^* - \left(\gamma_1 + \alpha + \delta\right) \alpha L^* = 0. \tag{3.27}$$

Therefore

$$\beta S^* (\gamma_2 + \delta) B^* + \beta S^* \alpha \cdot B^* - (\gamma_1 + \alpha + \delta)(\gamma_2 + \delta) B^* = 0 \tag{3.28}$$

from where

$$\beta S^* (\gamma_2 + \alpha + \delta) = (\gamma_1 + \alpha + \delta)(\gamma_2 + \delta), \tag{3.29}$$

You sent hence, $-E_{11} = 0$. *It is easy to see* $\det(-E) > 0$, *see the Appendix of this paper. Therefore,* $-E$ *is* $Volterra - Lyapunov$ *stable based on Lemma* 2.5.

So based on Lemma 2.6 *and Lemma* 3.1, *there exists a positive diagonal matrix* W , *such that* $W(-P) + (-P)^T W^T > 0$. *Thus* $W(P) + (P)^T W^T > 0$.

We conclude that the matrix P *defined in (3.25) is* $Volterra - Lyapunov$ *stable* .

We result the global asymptotic stability of $E_1 = (S^*, L^*, B^*)$.

3.6 Numerical results

Consider system (3.1) with $\alpha = 0.6, \beta = 0.3, \delta = 0.1, \gamma_1 = 0.1, \gamma_2 = 0.3$. We plot the phase plane portrait of L vs. S and B vs. S in Figs. (*Fig.6*),(*Fig.7*) for $R_0 = 0.9375$, a typical case of $R_0 < 1$, where the DFE is globally asymptotically stable. This is evidenced in these figures by the fact that all the five orbits converge to the DFE at $S = 1$ and $L = B = 0$.

Remark 40 *The figure (Fig.6) and (Fig.7) represent the five curves correspond to different initial conditions with* $L(0) = 0.1, 0.3, 0.5, 0.7, 0.9$, *respectively*

In addition, consider system (3.1) with $\alpha = 0.3, \beta = 0.4, \delta = 0.1, \gamma_1 = 0.1, \gamma_2 = 0.3$. Then, $R_0 = 1.4$ in this case, and the unique positive endemic equilibrium is located at $S^* = 0.71, L^* = 0.1$ and $B^* = 0.1$. We pick five different initial conditions, and plot these five solution curves by the phase plane portrait of L vs. S and B vs. S in Figs. (*Fig.8*), (**??**). From which one can see all these five orbits converge to the endemic equilibrium, showing the global asymptotic stability of the endemic equilibrium.([17])

Remark 41 *the figure(Fig.8) and* (**??**) *represent the five curves correspond to different initial conditions with* $L(0) = 0.1, 0.3, 0.5, 0.7, 0.9$, *respectively.*

- $R_0 \prec 1$

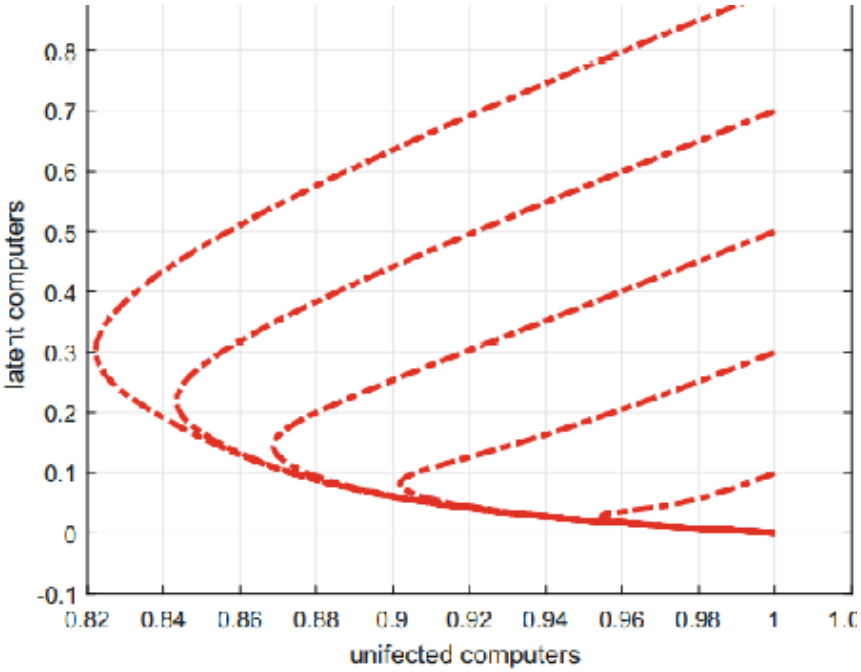

Fig. 6 Phase plane portraits of L vs. S for system (3.1).

(Fig.6)

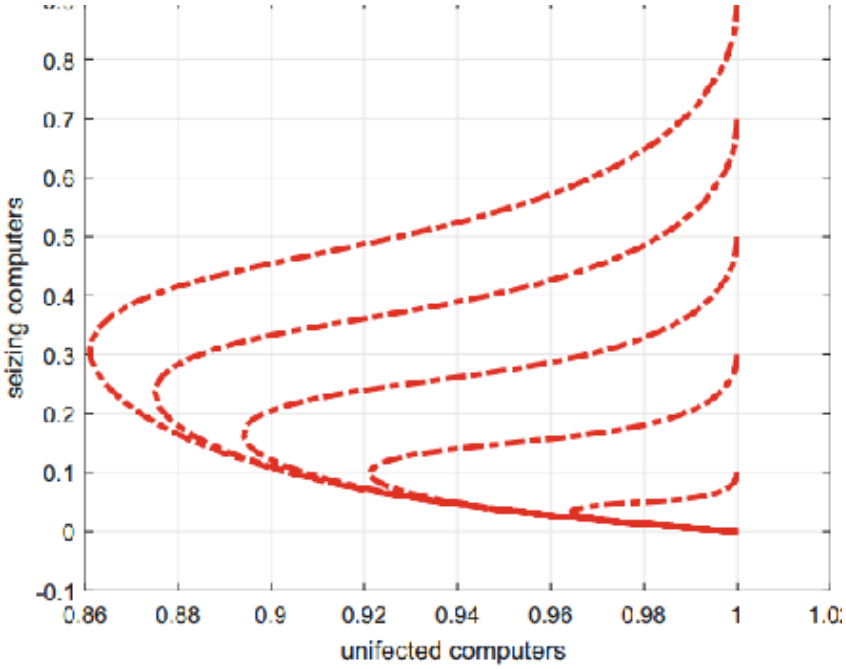

Fig.7 Phase plane portraits of B vs.S for system(3.1).

(Fig.7)

- $R_0 \succ 1$

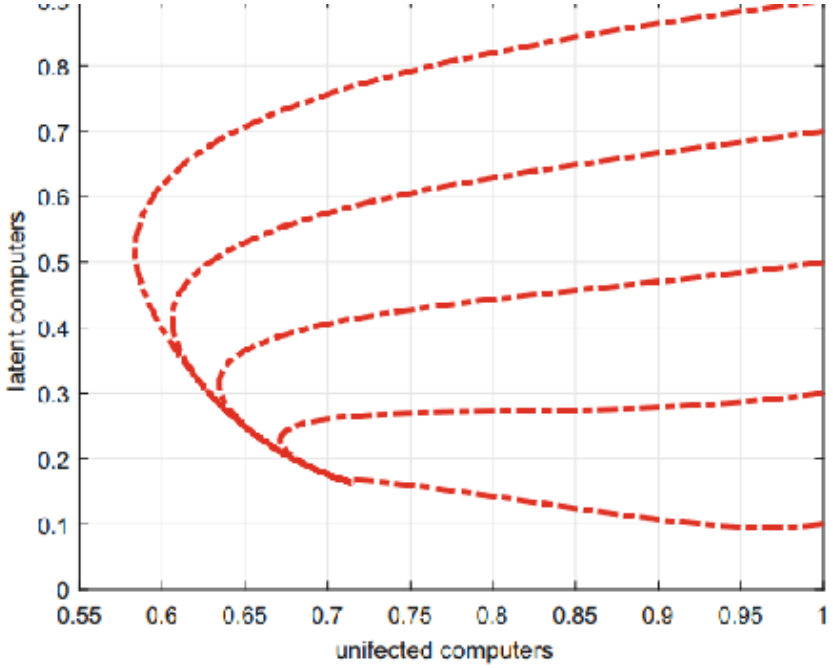

Fig. 8 Phase plane portraits of L vs. S for system (3.1).

(Fig.8)

-

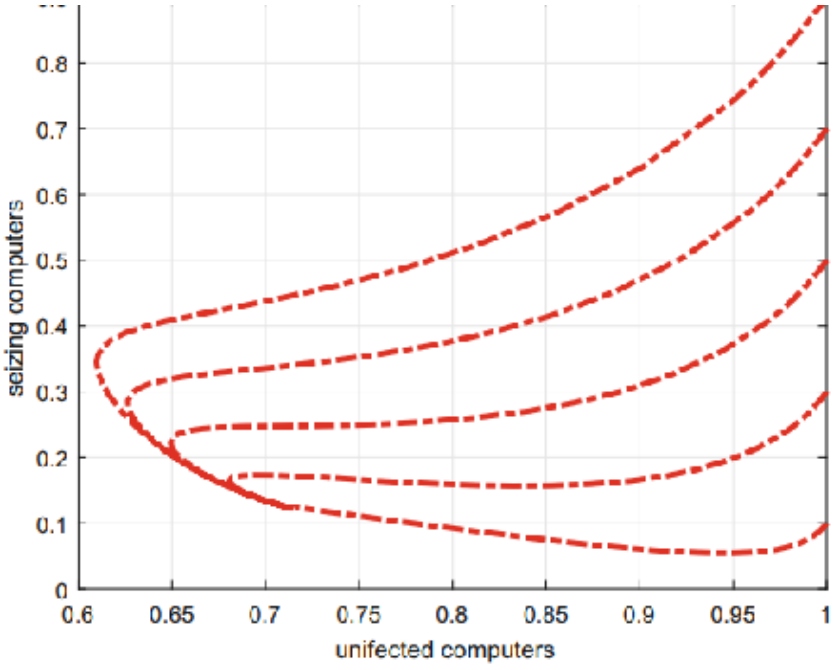

Fig. 9 Phase plane portraits of B vs. S for system (3.1).

(fig.9)

Numerical reuslts doing by matlab

Here is another numerical results for system (3.1) doing with matlab .

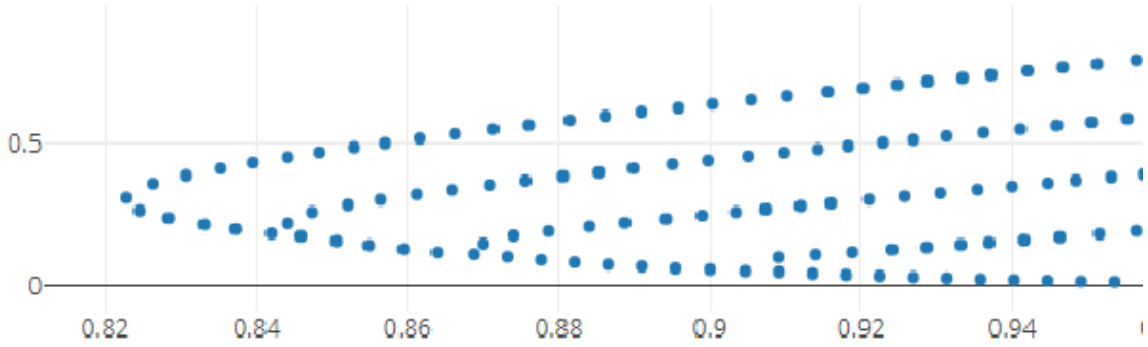

Fig. 10 Phase plane portraits of L vs. S for system (3.1).

(Fig.10)

The figure (*Fig.*10) represent the five curves correspond to different initial conditions with $L(0) = 0.1, 0.3, 0.5, 0.7, 0.9$, respectively.

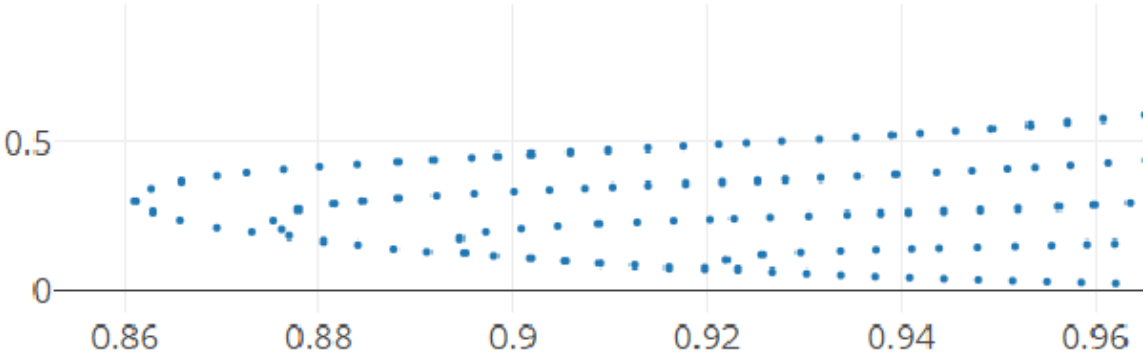

Fig.11 Phase plane portraits of B vs.S for system(3.1).

(Fig.11)

the figure (*Fig.*11) represent the five curves correspond to different initial conditions with $B(0) = 0.1, 0.3, 0.5, 0.7, 0.9$, respectively.

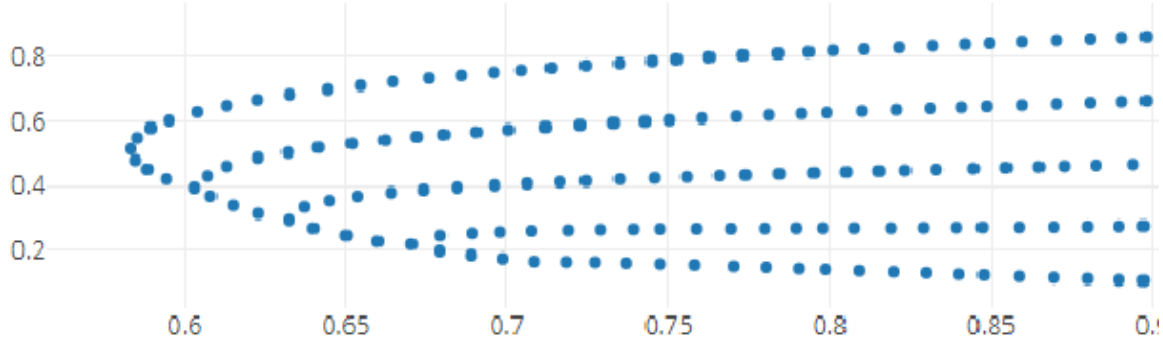

Fig. 12 Phase plane portraits of L vs. S for system (3.1).

(Fig.12)

the figure(*Fig.8*) represent the five curves correspond to different initial conditions with $L(0) = 0.1, 0.3, 0.5, 0.7, 0.9$, respectively .

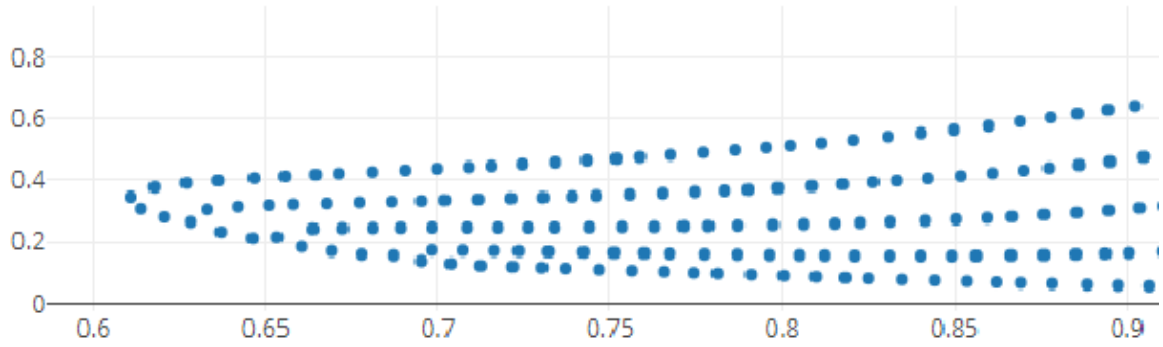

Fig. 13 Phase plane portraits of B vs. S for system (3.1).

(Fig.3)

the figure(*Fig.3*) represent the five curves correspond to different initial conditions with $B(0) = 0.1, 0.3, 0.5, 0.7, 0.9$ respectively.

Conclusion

We study a dynamical model characterizing the spread of computer viruses over the Internet. It is assumed that all infected computers possess infectivity, and latent comput ers have a lower cure rate than seizing computers. As we know, a computer user might try to clear viruses sponta neously even if he is not sure that viruses are staying in his computer possibly because:

1. he is accustomed to running antivirus program regu larly, or
2. he is informed that viruses are spreading over the Internet.

The global stability of a computer virus propagation model, which incorporates the two features mentioned above, is investigated. One major difficulty in studying the qualitative properties of this model lies in the construction of suitable Lyapunov functions, so that its success largely depends on trial and error as well as on specific problems. By combining this classical approach with the Volterra–Lyapunov matrix analysis, we have leveraged the difficulty of determining specific coefficient values, and as such, wider application of Lyapunov functions to dynamical systems could be promoted. The method Volterra–Lyapunov stability in this work is applied for a model of a computer virus propagation model. The analytical expressions of the stability analysis are provided and their numerical implementation is discussed .

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