Annals of Optimization Theory and Practice

Volume 2, Number 2, 35-45

August 2019

DOI: 10.22121/aotp.2020.213803.1025

# Mathematical simulation of contagious virus spread

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(Received: June 05, 2019 / Accepted: July 20, 2019)

**Abstract** In this article we study the prevalence of contagious disease in a community. To this end, we view society as a dynamic system and apply mathematical equations and relationships to it. First, we present the history of mathematical modeling in the field of contagious diseases and the work done in this field, then find out the probability of the virus spreading in a population with a number of people with contagious disease, and finally the differential equation of disease spread. Obtain the environment and then calculate the time it took to get the disease. In addition, we also plan to provide a model to describe how a virus is transmitted. In this model, we have four boxes called susceptible individuals, virus carriers, treated individuals, and improved individuals. We obtain the differential equations of growth and decline of each of these boxes and examine the stability condition of the system.

**Keywords** contagious disease; susceptible; treated; improved

## 1. Introduction

The virus is a small infectious agent that can only replicate within the living cells of an organism. Viruses are found in almost all the ecosystems of the earth and are known to transmit infections to many species, including bacteria, fungi, plants, insects, vertebrates and more. Large number of viruses cause disease in humans, pets or crops

Mathematical models and computer simulations are useful laboratory tools for testing past theories, making new theories, making quantitative guesses, and finding the right answers to complex questions. These models can replace the previous parameters with the specified parameters and compare the results with the previous models.

Considering ways of transmitting contagious diseases in communities, regions, and countries has helped scientists find appropriate ways to prevent or reduce transmission. There are many uses to these models. These models are used extensively in benchmarking, designing, implementing, and evaluating programs, identifying

optimization techniques, finding ways of prevention, drug therapy, and finally disease control.

Programs to improve health, use of antibiotics, and vaccination had a major impact on the popular belief of the 1960s that contagious diseases would eventually be eliminated. As a result, chronic diseases such as cardiovascular disease and cancer were attracted to industrialized countries, especially the United States, but contagious diseases were still the cause of human suffering and death in developed countries. In addition, agents of contagious diseases were constantly adapting and evolving, so that new contagious diseases arising from the diseases existing at the time emerged.

Newly diagnosed diseases at that time included Legionnaires' Disease (1976), Toxic Syndrome (1978), Hepatitis C (1989), and Hepatitis E (1990). Human Immunodeficiency Virus (HIV), the cause of Acquired Immune Deficiency Syndrome (AIDS), appeared worldwide in 1981, and the most important way of transmitting it was to have sex.

In the same years, diseases such as tuberculosis and pneumonia formed resistant chains against antibiotics, and the same antibiotic resistance led to the transformation of the agents of these diseases and their evolution. Diseases like yellow fever and malaria have reappeared and spread to different climates. One of the reasons for their re-emergence was climate change in different regions of the globe. Diseases such as plague and cholera have emerged as new diseases such as bovine madness and mumps fever. Surprisingly, the agents of these emerging diseases all came from previous disease agents such as bacteria, viruses, protozoa, and worms (such as pumpkin worms).

Since clinical trials involve both high cost and time-consuming time for their results to be known and some diseases cannot be studied in vitro, scientists have been thinking of modeling them using mathematical science, and in particular using it. They fell out of the differential equation system, so that mathematical models became increasingly important tools in analyzing and controlling outbreaks, especially contagious diseases.

The first mathematical model for smallpox epidemic was written in 1760 by Daniel Bernoulli. The model analyzed people's health in preventing the spread of smallpox, but serious work began on the mathematical modeling of epidemic diseases in the 20th century, and then diseases were modeled one after another by mathematicians and biologists. These models have been perfected over the years and data analysis has been performed on them. Simulators were also developed for the computers as they came on. Here are two mathematical models that have been very effective in recent advances in medical science in the field of infectious diseases.

In recent years, a mathematical model has been introduced to diagnose epidemics of the most common infectious diseases based on climatic parameters. Researchers at Tufts University School of Medicine in Boston have developed a mathematical model that assesses the probability of outbreaks based on environmental parameters in each season by daily infectious disease surveys. The scientists tested their mathematical model based on data collected by the University of Massachusetts on six diseases, according to the

Medical News ToD. The six diseases are: Jardia and Cryptosporidium (two intestinal infectious diseases), Salmonella and Campylobacter (two common intestinal diseases that are caused by the entry of Salmonella and Campylobacter bacteria and are very common in Europe), Shigellosis Tropical (caused by Shigella infection) and HIV caused by hepatitis A virus infection. Then, using climatic data collected between 1992 and 2001, the scientists studied the prevalence of each disease in Massachusetts based on the average daily temperature grade, time, and duration of each disease. Preliminary results of this model showed that the onset of these diseases was associated with a heat peak other than hepatitis A. The researchers then developed a mathematical algorithmic model based on daily, seasonal, and monthly information that analyzes the epidemic of these infectious diseases.

Another mathematical model that revolutionized medical science is the model proposed for AIDS. Infectionists and other physicians have long had a clear theory about AIDS, which is that the AIDS virus can stick to specific cells and infect them. These infected cells, most of which are white blood cells, either die by themselves, or they kill and kill their own cells instead of strangers. There were various biological evidences to support this hypothesis, but a group of mathematical scientists questioned the hypothesis that was popular in the medical world. These mathematicians presented a mathematical model for AIDS and based on this model showed that this hypothesis did not justify the slow course of the disease over the years, and that if the proposed hypothesis were correct, the disease would have to be recovered within a few months. It makes sense. These calculations challenged all previous and accepted assumptions among scientists. Of course, the researchers said in their report in Plus Madison that this study is only a mathematical model and cannot say what is really going on in the patient's body infected with the virus, and therefore more extensive physio pathological research is needed than the course of development.

Most of these mathematical models studied in the field of infectious and infectious diseases are composed of continuous time equations. For the first time in 1906, Hummer introduced a discrete time model and examined it thoroughly. In this model, the number of patients per unit of time was analyzed in terms of disease distribution density. Subsequently, differential equations were used to control diseases such as malaria and human-animal diseases. Among the scientists who have been involved in this field for many centuries have been Bonhoeffer and Novak whose proposed models have been the basis of many studies. Most of the information obtained from these models is the rate of disease growth, the parameters of the virus's initial proliferation, the conditions of disease growth decline, and the conditions to achieve infection status. Mathematical modeling and the study of the dynamics of their equation systems grew well into the mid-twentieth century, with the first major book published in this field by Bali in 1957 being one of the most prominent and important sources of modeling and dynamic study of infectious diseases.

In recent years, various branches of mathematics have been used in most sciences, including medicine, biology, environment, economics, engineering and meteorology, so that mathematics has become an integral part of science. It has always been one of the

human mental dreams in life to prevent and control unwanted and undesirable factors such as diseases. The regular repetition of the epidemics and the similar form of the epidemics of a disease for a long time has led mathematical researchers to develop a model for their interpretation and ultimately to control the spread by careful and precise control.

## 2. Diseases caused by the virus

Common diseases in humans, such as the common cold, influenza, smallpox, and herpes blistering, are caused by viruses. There are currently 21 families of viruses known to cause human disease. Figure 1.1 shows important diseases caused by various viruses in humans. Some of these diseases, such as AIDS, hepatitis, herpes simplex, measles, avian influenza, SARS, etc., are highly contagious (47) and viruses are also important contributors to disease transmission. The plague is on the human body. Mortality due to infectious diseases in 1998 and 2007 is shown in Figure 1.2. Approximately 1.6 and 4.4 million deaths from acute respiratory diseases occurred in 1998 and 2007 respectively, many of which were caused by viruses. AIDS in 1998 and 2007 killed 1.5 million and 1 million people worldwide, and measles is still a major killer in developing countries. Some viruses, such as HIV and AIDS, influenza, insect diseases, yellow fever and dengue are highly contagious. According to the World Health Organization (WHO) report on the global status of HIV / AIDS, approximately 70 million people have been infected with the virus, and about 35 million have died of AIDS since its spread. Although the burden of the disease varies considerably between countries and regions, the epidemic persists, and by the end of 2011, it was estimated that 34.0 million people (6.5–7.5 million) And 1.5% of adults between the ages of 15-49 lived with HIV worldwide.

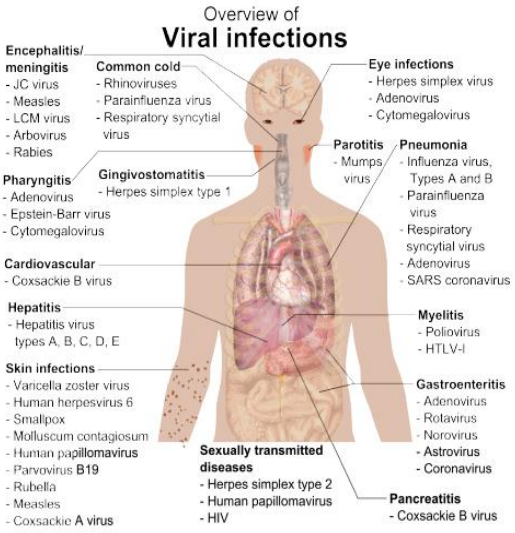


Figure 1. Overview of the major types of viral infection and the most important species associated with it.

Many dangerous viruses are effectively controlled by vaccination. There are useful vaccines for disease-causing viruses, such as smallpox, measles, rubella, epilepsy, varicella zoster, hepatitis A and B. The spread of some of these diseases, such as measles, has been significantly reduced in some developed countries by the use of vaccines.

However, these diseases still lead to pathogenicity in many developing countries. In addition, many vaccines that cause major diseases, such as hepatitis C, hepatitis D, and HIV, are not yet vaccinated.

Viruses have different mechanisms of pathogenicity in an organism that are highly dependent on the type of virus. Viruses usually damage the host through cell analysis, production of toxins, and cell deformation (3). When a virus enters the cell and completes its natural proliferation cycle, the host cell breaks down due to the internal physical stress caused by the virus replication or the defensive immune response. In addition, during the period of virus replication, many toxic viral components as well as byproducts due to cell proliferation accumulate in the cell. Cell degradation and toxic compounds cause cell death. In multicellular organisms, if sufficient numbers of cells are lost, the entire organism will suffer from its effects. Some viruses, if they continue to proliferate in the body, despite host defense mechanisms, can lead to lifelong infection or chronic infection. This is the case with hepatitis B and C viruses.

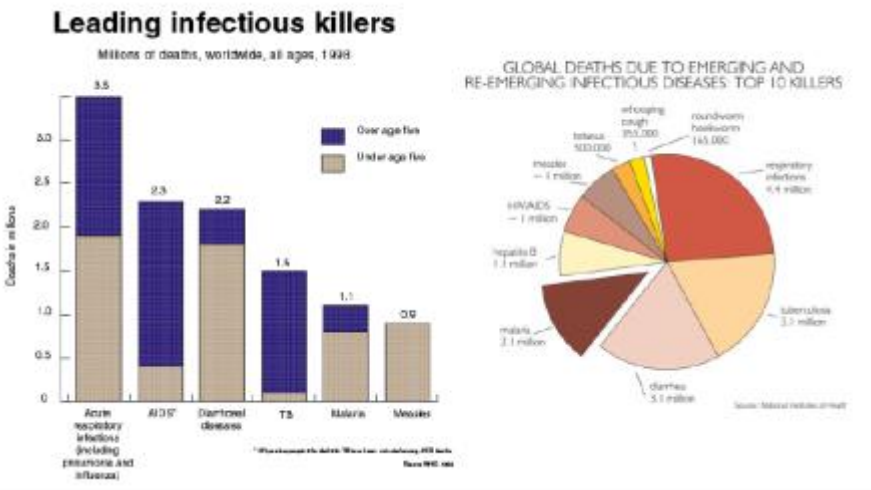


Figure 2: Infectious lethal agents in 1998 and 2007.

## 3. Mathematical modeling

We consider a community of $n$ population. Suppose a virus is spread by $k$ people. So $n-1$ people in this community are exposed to the virus. We choose a random sample randomly from this community. If the random variable $x$ represents the number of virus transmitters in a unit of time, then the probability function for this random variable is the same as the geometrical distribution function, as follows:

$$P(x)=\frac{\binom{k}{x}\binom{n-k}{k-x}}{\binom{n}{k}};\ 0\le x\le \min\{n-k,k\} \tag{1}$$

For example, in a 200-member community with 5 virus carriers, the probability of 3 people transmitting the virus is equal to:

$$P(x)=\frac{\binom{5}{3}\binom{195}{2}}{\binom{200}{5}}\cong 0.000075$$

Our goal is to obtain the expected length of time that all members of the population will be affected by the disease, so we expect the mathematical expectation of the preceding geometrical function.

$$E(x)=\frac{k(n-k)}{n}=k-\frac{k^2}{n} \tag{2}$$

Now, in order to obtain the equation for the growth of the number of virus carriers and also to calculate the time of infection of the whole population, we write the following differential equation based on the above mathematical expectation.

Suppose $d_1$ the number of carriers expected to be affected at t-th . Then we have:

$$d_{t+1}=d_t+d_t-\frac{d_t^{\,2}}{n}=d_t+\frac{1}{n}d_t(n-d_t);d_1=1,t=1,...,m \tag{3}$$

Here $m$ are the number of days to $d_1$ come to $n$. The strategic differential equation of this system is as follows:

$$\frac{dy}{dt}=y(1-\frac{y}{n}),\ \ y(0)=1 \tag{4}$$

The above differential equation is a separable differential equation that we solve:

$$dy=y(1-\frac{y}{n})dt\rightarrow\frac{dy}{y(1-\frac{y}{n})}=dt \tag{5}$$

We have a relationship with integrating parties:

$$\begin{aligned}&\int\left(\frac{1}{y}+\frac{1/n}{1-y/n}\right)dy=\int dt\ \rightarrow \ln y-\ln(1-y/n)=t\ \rightarrow ln\frac{y}{1-y_n}=t\\&\rightarrow y(t)=\frac{n}{1+(n-1)e^{-t}};\ t>o.\end{aligned} \tag{6}$$

We are now using the exact answer to this equation to estimate $m$, that is, how many days do the whole community become infected with the virus?

We calculate the time $t_n$ it takes to get $y(t)$ very close to the number $n$ . Suppose $\varepsilon > 0$ ,we solve the equation $y(t_n) = n - \varepsilon$ in terms of $t_n$ :

$$\frac{n}{1+(n-1)e^{-t_n}} = n - \varepsilon \rightarrow e^{t_n} = \frac{(n-\varepsilon)(n-1)}{\varepsilon} \rightarrow t_n = \ln(\frac{(n-\varepsilon)(n-1)}{\varepsilon}) \quad (7)$$

Now we use the Taylor series $\ln(1-x) = -x + {}^{x^2}/_2 + O(x^3)$ to approximate $t_n$ .

$$\begin{aligned} t_n &= \ln(\frac{(n-\varepsilon)(n-1)}{\varepsilon}) = \ln[n(1-{}^{\varepsilon}/_n)] + \ln[n(1-{}^{\varepsilon}/_n)] + \ln({}^{1}/_{\varepsilon}) \\ &= 2\ln n + [-{}^{\varepsilon}/_n + O(\frac{\varepsilon^2}{n^2})] + [-{}^{1}/_n + O(\frac{1}{n^2})] + \ln({}^{1}/_{\varepsilon}) \\ &= c\, log_2\, n + \ln({}^{1}/_{\varepsilon}) - (\frac{1+\varepsilon}{n}) + O(\frac{1}{n^2}) \end{aligned} \quad (8)$$

Where is $x = \frac{1}{n}$ or $x = \frac{\varepsilon}{n}$ and $c = 2ln2 \approx 1.386$. For the big ones $n$, $t_n = c \log_2 n$

That is, the time it takes for the whole community to become infected is: $t_n = 1.386 \log_2 n$.

For example, the length of time a person is infected with a virus is equal to:

$t_{1000} \approx 1.386 \log_2 1000 \approx 13.81$

Next, we are going to provide a model for describing how a virus is transmitted in general.

In this four-box model, we consider people susceptible(S) to the virus, contagious(I), treated(T), and improved(R).

In this paper, we focus on an epidemiological model, dividing the population into four categories of susceptibility, contagion, contamination, treatment and improvement. In short, we use SITR. In the model SITR, a susceptible person is exposed to a contamination prior to infection, which is after the infection has spread.

## 4. Model formulation

According to the previous section, the total population size N(t) in time t for the virus in question is as follows:

$$N(t) = S(t) + I(t) + R(t) + T(t) \quad (9)$$

We publish the rate (per capita) by $\beta$ and the mortality rate by $\mu$. We show the rate of people being treated with $\delta$ and $\omega$ the rate of their recovery.

The fraction $\gamma$ represents the infected individuals selected for treatment at a time. In addition, we hypothesized that treatment would reduce the proportion of infections $\delta$.

How can we write a differential system?

Suppose there is an average population of people $\beta N(t)$ who infect others at the time $t$, which $N(t)$ is the size of the whole population. Since $\frac{S(t)}{N(t)}$ is the likelihood of accidental exposure by an infected person to a susceptible person is equal to $\beta \frac{S(t)}{N(t)}$ the number of new infected individuals per unit time $t$, the rate of new infections at exposure is as follows:

$$\beta N(t)\frac{S(t)}{N(t)}(I(t)+\delta T(t)) = \beta S(t)(I(t)+\delta T(t)) \tag{10}$$

So $\beta S(t)(I(t) + \delta T(t))$ is the rate of people who have left the class $S(t)$ per unit of time t.

The number of susceptible viruses is reduced at a reduced rate $\beta$ and the amount $-\beta SI$ of susceptible is reduced. On the other hand, because the growth rate of people $\pi$ has improved and these people may be exposed to the virus again after recovery, so they are added to the $\pi R$ amount of talent, so we have:

$$S' = -\beta SI + \pi R \tag{11}$$

People who are susceptible to the disease are treated at the same rate $\delta$, and patients may also die at the rate $\mu$ before the treatment phase.

$$I' = \beta SI - \delta T - \mu I \tag{12}$$

$\omega T(t)$ is the rate of people receiving treatment that improve in time $t$ and die in proportion $\mu$. As a result:

$$T' = \delta T - (\mu + \omega)T \tag{13}$$

The equation for growth or decline of the number of people improved is as follows:

$$R' = \omega T - \pi R \tag{14}$$

And finally the population reduction equation will be:

$$N' = \text{-}\mu\text{N} \tag{15}$$

But since the death rates are taken into account in all the differential equations of the previous factors, we do not mention the latter equation in our equation system, which represents the dynamics of virus propagation.

$$\begin{cases} \frac{dS(t)}{dt} = -\beta S(t)I(t) + \pi R(t) \\ \frac{dI(t)}{dt} = \beta S(t)I(t) - \delta T(t) - \mu I(t) \\ \frac{dT(t)}{dt} = \delta T(t) - (\mu + \omega)T(t) \\ \frac{dR(t)}{dt} = \omega T(t) - \pi R(t) \\ \frac{dD(t)}{dt} = \mu I(t) + \mu T(t) \end{cases} \quad (16)$$

We are now looking for a system balance point, that is, where we do not have the patient or the person infected with the virus. Mathematically, when the number of contagious and treated people is zero. In this case the Jacobian matrix corresponds to said device at equilibrium point $E^* = (S^*.0.0.R^*.D^*)$

It is as follows:

$$\begin{bmatrix} 0 & -\beta S^* & 0 & \pi & 0 \\ 0 & \beta S^* - \mu & -\delta & 0 & 0 \\ 0 & 0 & \delta - \mu - \omega & 0 & 0 \\ 0 & 0 & \omega & -\pi & 0 \\ 0 & \mu & \mu & 0 & 0 \end{bmatrix} \quad (17)$$

So

$$p(\lambda) = \lambda^2(\lambda + \pi)(\lambda - \beta S^* + \mu)(\lambda - \delta + \mu + \omega) \quad (18)$$

By placing

$$a \coloneqq \pi \ . \ b \coloneqq -\beta S^* + \mu \ . \ c \coloneqq -\delta + \mu + \omega \quad (19)$$

We have:

$$p(\lambda) = \lambda^2(\lambda^3 + (a + b + c)\lambda^2 + (ab + ac + bc)\lambda + abc) \quad (20)$$

Thus, according to the Horowitz theorem, the prerequisite for local asymptotic stability is:

$$\begin{array}{l} (a + b + c) > 0 \implies \pi - \beta S^* + 2\mu - \delta + \omega > 0 \\ abc > 0 \implies \pi(-\beta S^* + \mu)(\mu - \delta + \omega) > 0 \end{array} \quad (21)$$

And the third condition is $(a + b + c)(ab + ac + bc) > abc$. Given the two preconditions, just have:

$$\begin{cases} (a + b) > 0 \implies \pi - \beta S^* + \mu > 0 \\ (a + c) > 0 \implies \pi + \mu - \delta + \omega > 0 \\ (b + c) > 0 \implies -\beta S^* + 2\mu - \delta + \omega > 0 \end{cases} \quad (22)$$

Given the above relationships and also by referring to the definitions of the variables above, we can obtain the conditions for the persistence of the virus in a community.

## 5. Conclusions and Suggestions

The basic purpose of mathematical modeling of communicable diseases is to study the prevalence and spread of diseases both in space and time. So that the mechanism of disease transmission and its affective features enable decision makers to make predictions about the disease and thus to design disease control strategies. Understanding the type of infectious disease in the region or country can lead us to approach reducing disease transmission.

The process of model selection and its formulation clarifies assumptions, values, and parameters; the parameters used in an epidemic model must be clearly interpreted. Mathematical models must be formulated in such a way that they are simple enough to be able to answer the questions.

Having an accurate understanding of the predicted parameters in the model and knowing the dominant relationship between the various parameters involved in the mathematical expression of the prevalence model of a particular disease has an important role in analyzing the model.

Many mathematical models used in the analysis of the prevalence of communicable diseases often appear in the form of several differential equations. Since it is often difficult and in some cases impossible to obtain the analytical solution of ordinary differential equations, so numerical methods can be employed as approximate tools by approximating the differential equation.

Mathematically simulating the prevalence of diseases, the following questions are now considered as future goals and tasks.

How can mathematical modeling prevent specific outbreaks of measles, measles, smallpox, AIDS, etc. depending on local and regional conditions?

How can a suitable mathematical model be presented for a contagious disease according to local and regional conditions?

How can the epidemic of the disease be diagnosed in the target population according to the mathematical model of a contagious disease in a target population?

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