Team Control Number
23578992
Problem Chosen
B
2023
IMMC
Summary Sheet

Investigations about disease has long proved effective for stopping pandemics from expanding. It is necessary to build a model and predict the origin, the spreading speed and its future.

After carefully observing the phylogenetic tree, we first conduct data processing on this tree by calculating the average velocity through nenerations. During data processing, we were surprised to find that the curve of the virus is generally linear, so we suppose that the mutation-lapse pattern fits in a expontional function and draw the final conclusion. We find out that the average mutation speed of this pathogan is 1.092 mutation per day, and the origin can date back to March the 8^{th}, 2020.

We also managed to find other factors influencing the spread of the virus to make it a linear pattern rather than an exponential function. So we introduced the epidemiological model SIR model to simulate the virus. By describing relationships between susceptible, infected and removed confluences and tranformation between these tribes, we can effectively explain the spread of virus and its mutations. Solution for differential equations make our model precise and reasonable.

Considering that the mutated virus will be more infectious than the previous generation virus to a certain extent, we introduced the concepts of strong and weak disease strains. When the strong disease strains will completely replace the weak disease strains after a certain period of time, we set the influence function of disease strains, and combined with the differential equation of SIR to calculate and solve. Finally, we found that under such a background, our model conforms to the linear relationship of a linear function.

Finally, based on previous models, we determine possible features of the diseases, guess its possible origin, predict its future development and give some effective suggestions.

Pandemic on the Way

Team#23578992

November 21, 2022

Contents

Section 1 Introduction

§1.1 Background

Diseases, especially pathogans, play a vital role in human's survival and death. It is certain that: in the future, there will be another novel pathogan which will bring about a global pandemic. WHO points out that, in such pandemics, pathogen genenic data and analyses are very useful to investigate the disease transmission.

What's the genetic sequencing? It refers to detecting the genetic sequence related to the pathogan. By finding out the difference between the one and the previous infector.

So, let us suppose that there is an outbreak of respiratory diseases. If experts find its reationship with the sudden change of genetic sequence. We can thus conduct investigations on this genetic related pathogan.

§1.2 Problem Overview

Given concerned data, our work can be mainly divided into the following three parts:

- Calculating the mutation speed of this pathogan.
- Using effective and reassuring methods to predict the possible date when the first case broke out and how the pandemic will develop in the days to come.
- Determining the possible features of this disease based on the model and analyses above, and carry out the best prevention and control plans to make sure that infectors can be effectively quarantined.

§1.3 General Assumptions

1. **In a certain period when pathogens are passed from person to person and complete the mutation process, the velocity is constant.**

   The mutation process of pathogans from one to another is long, secret and thus hard to detect. To facilitate our calculations, we naturally assume that the velocity with which the disease developsis constant in a certain period (here refers to the developing part).

2. **The mutating process is continous.**

   As explained above, given the complex process of mutation, incubation period is mysterious. To facilitate our calculations, we neglect the existence. In other words, the process is continous.

Section 2 Model A

§2.1 Model Overview

In the model, we investigate mutation index of this pathogan. We first focus on single mutations, then combine them and get out the result. Also, by defining speed of mutation and infection, we optimize our calculations and receive the final result.

§2.2 Notation

Table 1:

variable	definition
t	the moment when relevant data are captured
τ	time evolvement
$x(t)$	number of mutations
A, k	constants in the expontional function

§2.3 Calculating mutations

To study the mutation index of this pathogan, we first need to speculate the mutation index of a certain infector, we define the infector as x_{nj}. Here, n refers to the generations x_{nj} has experienced. The picture below illustrates the generations relationship between such infectors. We define **001** and **004** as the first generation.

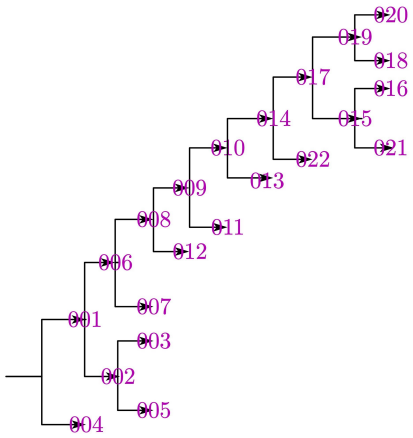


Figure 1: Generaton relationship between infectors

Given the phylogenetic tree of this pathogan,we can analyze the relationship between generation n, number of mutation and time lapses (time when one pathogan pass down to another). This can facilitate our further analyses.

We choose the simulation method to build our model. Based on General Assumption 2, the process is continous, which means certain breaks don't exist. Therefore, simulation is precise and rational.

To begin with, we want to find out how generations affect number of mutations, in other words, how fast the pathogan evolves. Surprisingly, we find out that the mutation-time pattern is linear. It measns that time lapse has only a tiny impact on pathogan development in initial developments. The figure below shows the discrete points and simulating results.

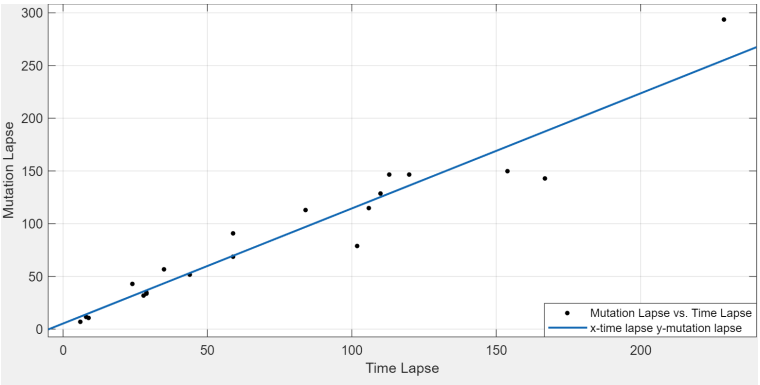


Figure 2: Relationship between time lapse and mutation lapse

Here, the slope of this staight line is 1.092. We can thus address a preliminary

conclusion that the evolution rate of this pathogan is 1.092 mutations per day.

§2.4 Expontional pattern of mutations

The result we have found is astonishing: the linear pattern is so simple that it may reduce preciseness of our model. Therefore, iy's necessary for us to introduce a reasonable explanation.

There may be one possible explanation for this phenomenon: This may reflect some of the features of this disease: stable and then significantly mutated many generations later. Therefore, it's genenic breakout is hard to detect since there are only nine generations provided. We can thus give an assumption that mutation velocity is an expontional function.

Let us suppose that at moment t, number of mutations obeys the function $x(t)$, then after τ, number of mutations may grow to $x(t+\tau)$. Therefore, according to previous analyses and figures, it's easy to find that:

$$x(t+\tau) - x(t) = kx(t) \cdot \tau$$

Hence, if τ is extremely small, the following differential equation can be introduced:

$$\mathrm{d}x(t) = kx(t) \cdot \mathrm{d}t$$
$$\frac{\mathrm{d}x(t)}{\mathrm{d}t} = kx(t)$$

And then, we can solve the equation above and find that:

$$\int \frac{\mathrm{d}x(t)}{x(t)} = \int k\mathrm{d}t$$
$$x(t) = A \cdot e^{kt}$$

(here, A and k are constants).

Thus, we can explain why the mutation pattern is linear: it's just an expontional function and is still in the incubation period. So, using Matlab, we can get the following function describing the relationship between mutations and time lapse:

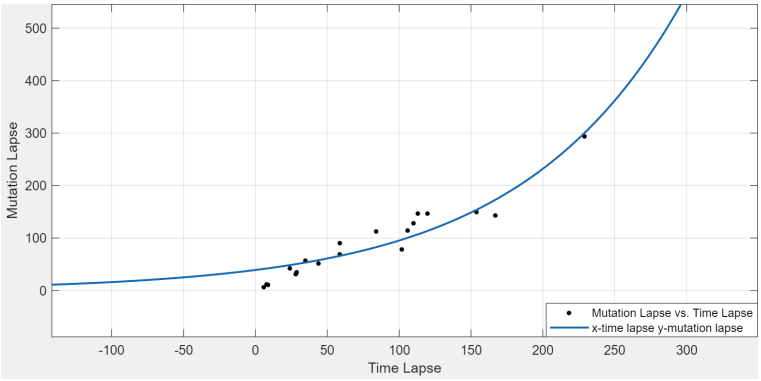


Figure 3: Expontional Function drawn between time lapse and mutation lapse

(according to precise calculations, $A = 39.14$ and $k = 0.008898$)

§2.5 Dating back to Infector No.0

To take a closer look at the pandemic, we also need to find out headstream of it, in other words, when the pathogan mutates into one that can spread between human. Here, we define the time of infectious disease outbreak as the time of the common ancestor (who is also the infected patient) called Infector No.0.

We use the linear model which is actually more suitable to the real case. As the phylogenetic tree demonstrates, Infector No.0 experienced 50 mutations and create the pathogan in infector 001. According to the function, it takes 41 days for the muation to complete.

Therefore, the pandemic appears at March 8^{th}, 2020.

We can get the final result: the mutation rate is 1.092 mutations per day. The pandemic first broke out at March 8^{th}, 2020. In the next section, we will explain why the result shapes like a line and how we predict the future of this pandemic.

Section 3 Model B

§3.1 Model Overview

In this section, we will use SIR model to dig out equations involving the susceptible,the exposed, the infected and the removed as well as predict number of infected people and viral virulence with the evolvement of time. Also, we will give some reasonable suggestions on prevention and control time of this disease based on previous calculations and predictions.

§3.2 Model Assumptions

1. **The time during which a person is infected is constant.**

   In this model, we will mainly focus on a certian pathogan and then two relevant pathogans. This means the time for the pathogan to infect others will not change until it mutate into a new pathogan.

2. **An infector can infect a constant number of susceptible people.**

   We assume that infectivity of this pathogan will not differ because of the genenic difference of people. Therefore, the people that an infector can infect will not differ too.

3. **Number of susceptible people is proportional to population of infectors.**

   According to the previous assumptions, we can without doubt receive this assumption.

4. **Population of the removed confluence is proportional to number of the infected.**

   As the virus behaves almost the same, there is always a certain amount of people who recover from the disease every day. As the time goes, we can prove this assumption right.

5. **The number of transmission from the removed to the susceptible is proportional to the removed.**

   As time passes, the people who recovered from the disease will lose immunity, it's thus necessary to take this condition into consideration. Same to the

last assumption, the disease will not have any other disturbance on the tranformation from the removed to the susceptible.

6. **Number of deaths is neglected.**

   As a mysterious disease, whether this pathogan will cause a large number of deaths remains unknown. To facilitate our building the dynamic model, number of deaths will not be considered.

7. **When two relevant pathogans coexist in one infector, the one with a stronger virulence will immediately replace the other one.**

   To facilitate our calculation, we can simplify the time in which one replace another.

8. **Two pathogans have the same susceptible confluence.**

   Though there are slight differences in two pathogans, they share more similarity and develop from the same ancestor. Considering the common features, the conclusion can be drawn that they face the some susceptible confluence.

§3.3 Notation

Table 2:

variable	definition
P	population in a society
S	population of susceptible confluence
I	population of infected confluence
I_A, I_B	population of infected confluence for pathogan A and B
R	population of removed confluence
t	moment of the whole process
σ	the average duration a person is infected
λ	number of person an infector can infect
η	population of people who is losing immunity

§3.4 Using SIR model to monitor dynamic change

To accurately predict the trend of this disease, we introduce the SIR model. It can effectively help us illustrate the relationship between infectors and others.

In this model, we first divide our society into three parts: the susceptible S, the infected I and the removed R. If the number of people in this society is P,

since number of deaths is not taken into consideration, it's obvious that:

$$\begin{cases} S+I+R=P \\ \dfrac{\mathrm{d}S}{\mathrm{d}t}+\dfrac{\mathrm{d}S}{\mathrm{d}t}+\dfrac{\mathrm{d}I}{\mathrm{d}t}=\dfrac{\mathrm{d}R}{\mathrm{d}t}=0 \end{cases}$$

As the pandemic cannnot appear overnight. It is common knowledge that a wide range of pandemic is actually caused by one or a few initial infectors. Therefore, it's necessary to conduct some investigations on the initial value of this society and confluences. That is S_0, I_0 and R_0. Also, if we assume that there are ϵ people who get infected at the beginning. We can descibe the society without the pandemic:

$$\begin{cases} S_0 &= P-\varepsilon \\ I_0 &= \varepsilon \\ R_0 &= 0 \end{cases}$$

A certain judging criteria of a certain disease is also needed. Here, we introduce the virus index $I(T)$ for a certain pathogan T. It is related with σ and λ. The former represents how long it takes for this disease to die out in ones body, in other words, how long it takes for the one to recover, while the latter symbolizes how many people a person can infect.

We will then discuss the transmission between these confluences. The process mainly involves two kinds of tranformation: the susceptible get infected and turn into the infected, while some infected get healed and turn into the removed. The figure below reveals the change between these confluences:

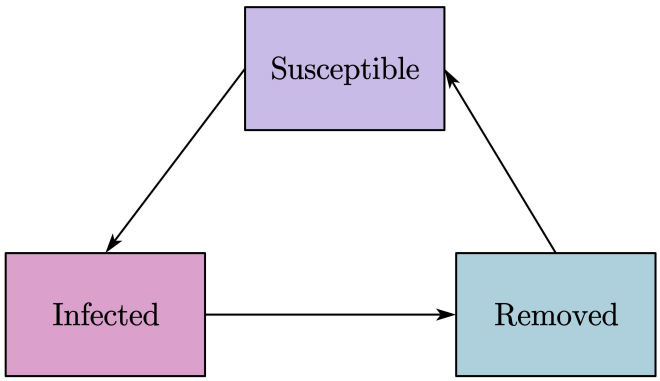


Figure 4: SIR transmission

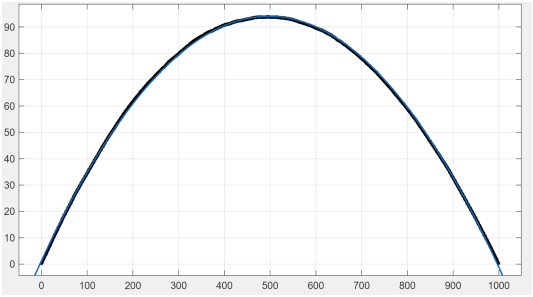
Then, we can discuss the mathematical relationship of these conflue. Using calculus, its dynamic feature can be illustrated. The details are as follow:

- $S \to I$

  Based on Model Assumption 2, we can also assume that in unit time, number of people an infector can infect is proportional to S. It means that:

$$\frac{\mathrm{d}(S \to I)}{\mathrm{d}t} = \lambda \cdot S \cdot I$$

  The figure below shouws how the S_I pattern evolves, it shapes like a quadratic function:



- $I \to R$

  According to Model Assumption 1, the infection time is constant. It means that after σ, an infector will tranform into a removed. After that, he or she will remain as a removed for a period of time, and finally become a susceptible person again. The equation below shows this change:

$$\frac{\mathrm{d}(I \to R)}{\mathrm{d}t} = \frac{\mathrm{d}(S \to I) \cdot (t - \sigma)}{\mathrm{d}t}$$

- $R \to S$

  During this process, we mainly focus on how people lose their immunity. We suppose that number of people who meet this demand is proportional to R. We introduce a parameter η to describe this tribe of people. Since the secret of this mechanism lies in human body, it's not related to the pathogan, and thus making η a unique parameter. So:

$$\frac{\mathrm{d}(R \to S)}{\mathrm{d}t} = \eta \cdot R$$

This is only the dynamic description of these relationships. Now we need to study how the disease spread. Since the process is hard to define only using equations, we conduct a true value simulation on data in being.

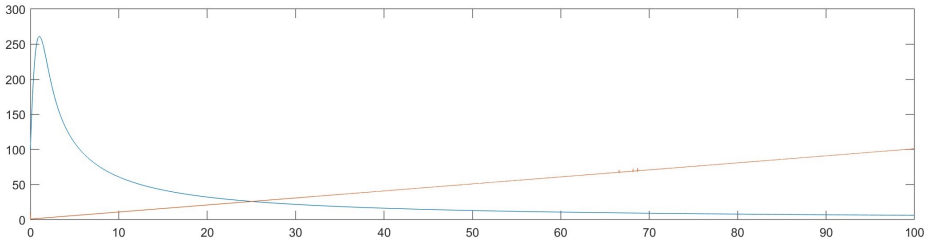


Figure 5: Numeral solution of this differential equation

The image above illustrates the solution to the differential equations. The blue line demonstrates the S pattern while I is shown through the yellow line. It can be discovered that both of them reach a stable state after a period of time, which verifies the correctness of the stable-status analysis.

From the image above, we can find that the infector pattern fits in the linear pattern. This is consistent with our inference in the second section.

So, when we conbine the dynamic equations with the static state, we can find that:

$$\begin{cases} \frac{\mathrm{d}S}{\mathrm{d}t} &= \eta \cdot R - \lambda \cdot S \cdot I \\ \frac{\mathrm{d}I}{\mathrm{d}t} &= \lambda \cdot S \cdot I - \lambda \cdot S(t-\sigma) \cdot I(t-\sigma) \\ \frac{\mathrm{d}R}{\mathrm{d}t} &= \lambda S(t-\sigma) I(t-\sigma) - \eta \cdot R \end{cases}$$

We then integrate out previous results:

$$\begin{aligned} I &= \int_0^t \frac{\mathrm{d}I}{\mathrm{d}t} = \int_0^t [\lambda \cdot S \cdot I - \lambda \cdot S(t-\sigma) \cdot I(t-\sigma)] \\ &= \sigma \cdot \lambda \cdot S \cdot I \end{aligned}$$

$$\therefore S = \frac{1}{\lambda \cdot \sigma}$$

And, when we substitute it into the previous equation, we can find that:

$$\eta(P - I - \frac{1}{\lambda \cdot \sigma}) = \frac{I}{\sigma}$$

$$\therefore I = \frac{P \cdot \sigma - \frac{1}{\lambda}}{\sigma + \frac{1}{\eta}}$$

$$\begin{aligned} R &= P - I - \frac{1}{\lambda \cdot \sigma} \\ &= P - \frac{P \cdot \sigma - \frac{1}{\lambda}}{\sigma + \frac{1}{\eta}} - \frac{1}{\lambda \cdot \sigma} \end{aligned}$$

Later, we define the stable status of this pandemic (this doesn't mean there are no newly infected people). That is:

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} &= 0 \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} &= 0 \\ \dfrac{\mathrm{d}R}{\mathrm{d}t} &= 0 \end{cases}$$

Finally, we can get a synthetical formula describing the population of I, for a single pathogan named T, whose virus parameter is σ_T and λ_T, its virus index can be described as:

$$I(T) = \frac{P \cdot \sigma_T - \frac{1}{\lambda_T}}{\sigma_T + \eta}$$

After explaining how the SIR model works and how we define the stable status, we can now make a categorization discussion about the future of this disease:

- If $\dfrac{1}{\lambda \cdot \sigma} < P$, the pandemic will remain at a stable status. At this moment, $I = \dfrac{P \cdot \sigma - \frac{1}{\lambda}}{\sigma + \frac{1}{\eta}}$. Of course, the infected will soon get healed, so this status will not last for a long time.
- If $\dfrac{1}{\lambda \cdot \sigma} \geq P$, the pandemic will die out.

§3.5 Two pathogans competing for dominance

There are definitely mutations of pathogans as time passes, and it is known to all that as time passes, its virulence will decline while its infectivity will rise. So, how will the disease develop when two pathogans are alive?

According to Model Assumption 7, there is no need in dividing the susceptible confluence and the removed ones. But for the infected, we define I_A and I_B for pathogan A and B.

Here, we introduce a new concept—-dominance. As the word shows, the one with more dominance will dominate the infector's body. Here, we assume that the moment the more dominant one enters an infector's body, it will kill the one

with less dominance. In other words, there is always one pathogan in one's body. Given the close relationship between dominance and infectivity, we use λ to judge its dominance. Under our circumstance, we assume that $\lambda_A < \lambda_B$.

So, we can illustrate the relationship between these two pathogans with the picture below:

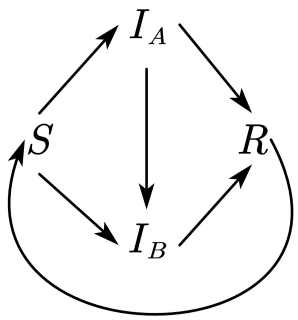


Figure 6: Transformation between confluences

So, we can find that for A, $S_A = S, R_A = R$ and for B, $S_B = S \bigcap I_A, R_B = R$.

According to Model Assumption 2 and previous calculations, we can first apply our model to a single pathogan A, that is:

$$\frac{\mathrm{d}(S \to I_A)}{\mathrm{d}t} = \lambda_A \cdot S \cdot I_A$$

And when B comes to the battle, all of this is going to change. Based on previous findings:

$$\frac{\mathrm{d}(S \to I_B)}{\mathrm{d}t} = \lambda(S + I_A) \cdot I_B \cdot \frac{S}{S + I_A} = \lambda_B \cdot S \cdot I_B$$

$$\frac{\mathrm{d}(I_A \to I_B)}{\mathrm{d}t} = \lambda_B \cdot (S + I_A) \cdot I_B = \lambda_B \cdot I_A \cdot I_B$$

These two equations descibe how the inspected change, the former focuses on how the susceptible confluence is forced to get B rather than A, and the latter illustrates how infectors who already got A are forced to get B instead.

In addition, based on Model Assumption 4, we can rebuild the transmission from I to R:

$$\frac{\mathrm{d}(I_A \to R)}{\mathrm{d}t} = \frac{\mathrm{d}(S \to I_A)(t - \sigma)}{\mathrm{d}t}$$

$$\frac{\mathrm{d}(I_B \to R)}{\mathrm{d}t} = \frac{\mathrm{d}(S \to I_B)(t-\sigma)}{\mathrm{d}t} + \frac{\mathrm{d}(I_A \to I_B)(t-\sigma)}{\mathrm{d}t}$$

Finally, we need to discuss the tranformation of R to S. According to Model Assumption 5, we can define that:

$$\frac{\mathrm{d}(R \to S)}{\mathrm{d}t} = \eta \cdot R$$

So, we can now descibe these confluences synthetically:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \eta \cdot R - \lambda_A \cdot S \cdot I_A - \lambda_B \cdot S \cdot I_B$$

$$\frac{\mathrm{d}I_A}{\mathrm{d}t} = \lambda_A \cdot S \cdot I_A - \lambda_B \cdot I_A \cdot I_B - \lambda_A \cdot S(t-\sigma) \cdot I_A(t-\sigma)$$

$$\begin{aligned}\frac{\mathrm{d}I_B}{\mathrm{d}t} &= \lambda_B \cdot S \cdot I_B + \lambda_B \cdot I_A \cdot I_B - \lambda_B \cdot S(t-\sigma) \cdot I(t-\sigma) - \lambda_B \cdot I_A(t-\sigma) \cdot I_B(t-\sigma) \\ &= \lambda_B \cdot (S + I_A) I_B - \lambda_B \cdot [S(t-\sigma) + I_A(t-\sigma)] \cdot I_B(t-\sigma)\end{aligned}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \lambda_A \cdot S(t-\sigma) \cdot I_A(t-\sigma) + \lambda_B \cdot [S(t-\sigma) + I_A(t-\sigma)] \cdot I_B(t-\sigma) - \eta \cdot R$$

Plus, because $S + I_A + I_B + R = P$, it's clear that:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \eta \cdot (P - S - I_A - I_B) - \lambda_A \cdot S \cdot I_A - \lambda_B \cdot S \cdot I_B$$

$$\frac{\mathrm{d}I_A}{\mathrm{d}t} = \lambda_A \cdot S \cdot I_A - \lambda_B \cdot I_A \cdot I_B - \lambda_A \cdot S(t-\sigma) \cdot I_A(t-\sigma)$$

$$\begin{aligned}\frac{\mathrm{d}I_B}{\mathrm{d}t} &= \lambda_B \cdot S \cdot I_B + \lambda_B \cdot I_A \cdot I_B - \lambda_B \cdot S(t-\sigma) \cdot I(t-\sigma) - \lambda_B \cdot I_A(t-\sigma) \cdot I_B(t-\sigma) \\ &= \lambda_B \cdot (S + I_A) I_B - \lambda_B \cdot [S(t-\sigma) + I_A(t-\sigma)] \cdot I_B(t-\sigma)\end{aligned}$$

The process in which viruses spread is nearly the same with the condition where only one pathogan is considered. Thus, we will not explain it in detail again.

Finally, it comes to the analyses on stable status, it is clear that:

$$\begin{cases} \frac{\mathrm{d}S}{\mathrm{d}t} &= 0 \\ \frac{\mathrm{d}I_A}{\mathrm{d}t} &= 0 \\ \frac{\mathrm{d}I_B}{\mathrm{d}t} &= 0 \end{cases}$$

Implementing it on our previous findings, we can find that:

$$\eta \cdot R = \lambda \cdot S \cdot I_A + \lambda_B \cdot S \cdot I_B$$

$$\lambda_B \cdot I_A \cdot I_B = 0$$

When we integrate the first formula above, we can receive the equation:

$$\begin{aligned} I_B &= \int_0^t \frac{\mathrm{d}I_B}{\mathrm{d}t} \\ &= \int_0^t [\lambda_B(S + I_A) \cdot I_B - \lambda_B S(t-\sigma) + I_A(t-\sigma) \cdot I_B(t-\sigma)] \\ &= \sigma\lambda_B(S + I_A) \cdot I_B \end{aligned}$$

$$\therefore S + I_A = \frac{1}{\sigma \cdot \lambda_B}, I_B > 0$$

And, given the description of stable status, it's obvious that $I_A = 0$, therefore:

$$S = \frac{1}{\sigma \cdot \lambda_B}$$

Looking back to previous equations, we can get the result:

$$\eta(P - I_B - \frac{1}{\sigma \cdot \lambda_B}) = \frac{I_B}{\sigma}$$

$$\therefore I_B = \frac{P \cdot \sigma - \frac{1}{\sigma}}{\sigma + \frac{1}{\eta}}$$

The image above illustrates how the equation actually works. Note that similar to the simple SIR model, this comparison and competing process is iterable.

Section 4 Optimization and prediction

§4.1 Determining the disease

To further study the pathogan, we first need to predict what the disease is and its features. After investigations and comparison, we find that the pathogan is likely to be one mutant strain of influenza. Comparing it to H7N9 avian influenza, we find our inference proper:

- Influenza is a nootic respiratory infectious disease. It's spread meets the demand of our previous analyses.
- Unlike ordinary influenzas, H7N9 avian influenza can not only spread between chicken-human and chicken-chicken but also pass down from human to human as well.
- Nootic respiratory infectious diseases usually involve wild lives, which are often the source of infection. However, chicken become the main source of infection, which fits the description of this pathogan.

§4.2 Defining features

After learning that, we search for the infectivity of this virus[1] and compare it with other pathogans including the variants of Covid 19. We weigh them mainly by λ. For example, one avian Influenza infector can infect about 1.4 people. So its $\lambda = 1.4$. The exact number is shown below:

	H7N9 avian Influenza	Covid 19	Delta variant	Omicron variant	new variants
λ	1.4	2-3	5	8	>16

From the data above, we can find that: compared with other infectious diseases, influenza has low infectivity. And because of the high lethality, it's difficult for this pathogan to cause a large-scale pandemic. According to previous calculations, we predict that the pandemic will soon die out with the assistance of proper measures.

However, we should not lose vigilance. These effective measures will be detailedly explained in the sections to come.

Section 5 Strengths and Weaknesses

Strengths

- **Universality**

  We build a model describing the spread of virus and mainly focus on dynamic tranformations. This makes our model practical. This model can also be applied to other infectious diseases since relevant parameters bear utility.

- **Effiency**

  Our model is easy to understand and does't involve any complex calculations. As long as related data is given, we can work out an accurate prediction and infer in how many days the pandemic will stop. This makes our model efficient.

Weaknesses

- **Limitations**

  Although we have given a detailed and reasonable explanation about why the mutation rate fits in a linear pattern through SIR model, several other factors like control of governments, sudden deaths are neglected, causing limitations in our model.

Section 6 A new nootic disease is on the way

Recently, a piece of astonishing news has come to our eyesight: a respiratory infectious disease shown the sign of going viral. Now, scientists have found its similarity in another pathogan which has been found in the body in chicken. It may be one of the most serious pandemic since the breakout of corona virus.

After close and precise investigations, these clues point to the same possibility: the disease is most likely to be one mutant strain of influenza! Highly pathogenic avian influenza spreads quickly, is seriously ill and has a high case fatality rate. Because the virus gene is prone to mutation, it can infect people and cause respiratory system and multiple organ failure, which is called "human avian influenza disease". The disease progresses rapidly and has a high fatality rate. In addition, unlike normal influenza, this disease can also spread between human and human through spit, food and etc. We must not lose vigilance for this disease.

So, what should we do? Experts made the following suggestions:

- **Protect yourself.** As most respiratory diseases spread through air, wearing masks during the pandemic is vital in protecting oneself. Also, having less contact with people, stopping going outside during weekends and washing your hands often can reduce the possibility you get this disease.
- **Isolate pathogens (infected people) in time.** As has been explained before, the pathogan has a low infectivity but a high lethality. Therefore, one infectors are well quarantined, the pandemic will soon disappear.
- **Detect the variant disease strains in time and do the corresponding work.** Governments should take effective actions, study the pathogan in time and inform the public of its relevant features.

References

[1] Tracking of Influenza Subtypes, GISAID
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