Bachelor of Science

"Study of a pandemic: how can a contagious feature spread through a population?"

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Contents

1 Introduction

1.1 Preamble

Let us imagine a population composed of a finite number of individuals who interact with each other as a society. This population has a specific characteristic (an illness or a behaviour for example) that can be transmitted with a certain probability from one infectious individual to another by casual contact. In this paper, we will focus on the transmission of a disease.
More precisely, we assume that when an individual who is not ill meets an infectious individual, then there is a certain probability that afterwards, the first individual will become ill as well. Then, we consider that the infected individual is eliminated as soon as he is no longer infectious e.g. he may be dead or recovered. This is the most basic model that can be conceived, known as the SIR model.
We can also imagine other plausible models in which the individual cannot acquire immunity, and can remain infectious even if cured (SIS model).

All of these models together can give a fairly accurate representation of real life. Although, for the sake of simplicity, we will only consider the SIR and SIS models.
Here, we have imagined models based on diseases, but if we ignore deaths, these models also work for social or psychological behaviour. For example, gossip or new slang terms used in society operate like this as well.

1.2 Problem

From this extent, it is relevant to wonder : How does this transmissible characteristic evolve within the population in time ? And what could be the outcomes?
In this project, we will first see the basic SIR model applied in different situations, then we will study the SIS model.

Note:

For this project, we were inspired by the way such compartmental models are represented on the Internet. This means that we used the classical way of representing the model and setting the equations. However, all the proofs and the study of the evolution of the systems were completely done by us.

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2 The SIR model

2.1 Definition

We are willing to study the evolution of an infectious characteristic among a finite set of individuals as a function of time. We designate by:

- **S**: The proportion of **S**usceptible individuals. **Susceptible** individuals do not have the disease but may catch it when they make *contact* with an **infected** individuals. We will come back to the process of transmission later. All **susceptible** individuals are identical.
- **I**: The proportion of **I**nfectious individuals. **Infectious** individuals are infected by the disease. They can transmit it to another individual **or not**.
- **R**: The proportion of **R**emoved individuals (dead or healed). **Removed** individuals have already been infected by the disease and are either recovered or dead. They cannot be infected nor transmit the disease anymore.

Now, one can set several plausible parameters on which the evolution of the three compartments depend on. For this reason, we state some elementary assumptions:

- There is only one possible turn of event for each individual, that can be represented as follows:

$$\boldsymbol{S} \longrightarrow \boldsymbol{I} \longrightarrow \boldsymbol{R}$$

- All the individuals in a same compartment (S,I or R) are identical: there are no different stages of disease, no innate immunity, etc.

Obviously, we need to make various assumptions to simplify the real world phenomenon. Indeed, factors are just too complicated and diversified to express everything in a simple set of equations. Hence, we assume that:

1. The total number of individuals stays constant.
2. The rate of variation of the infectious people (**I**) is proportional to the contact between susceptible and infectious individuals. This occurs at a constant rate.
3. The removal of individuals occurs at a constant rate. This could be explained by constant death and recovery rates.

In order to model the evolution of each of the three compartments $\boldsymbol{S}$, $\boldsymbol{I}$ and $\boldsymbol{R}$, we need to determine how they influence on each other as a function of time. Therefore, we define three functions **S(t)**, **I(t)** and **R(t)** designing respectively the values of S, I and R as a function of time.

Since $I(t)$ represents the number of infectious individuals at time t, then its derivative, $I'(t)$ represents the number of *new* infectious individuals, minus the number of individuals who are no longer infectious at time t. If $I'(t)$ is **positive** then the number of infectious individuals is increasing, otherwise, the number of infectious individuals is decreasing or constant. Hence, we can write:

$$\begin{cases} S'(t) = -I_{new}(t) \\ I'(t) = I_{new}(t) - R_{new}(t) \\ R'(t) = R_{new}(t) \end{cases}$$

where $I_{new}(t)$ and $R_{new}(t)$ are respectively the *new infectious* and *new removed* individuals added respectively to **I** and **R** on an interval of time δt that goes to zero.

Let us look into those *new infectious* individuals. We know that the transformation of a **susceptible** individual into an **infectious** one is due to the transmission of the disease from an I-individual to an S-individual.

The number of new infectious individuals is equal to the probability for such a contact to cause the transmission of the disease multiplied by the number of contacts involving a susceptible and an infectious individual.

Assuming that the number of such contacts is proportional with the product of the number of susceptible individuals and the infectious ones (Assumption 2.), we get:

$$I_{new}(t) = \alpha S(t) I(t)$$

where α denotes the average number of contacts per person per unit of time multiplied by the probability of disease transmission between a susceptible and an infectious individual.

Now for the *new removed* individuals ; assuming that the removal of individuals occurs at a constant rate (Assumption 3.), we get:

$$R_{new}(t) = \beta I(t)$$

where β denotes **the transmission rate** (sum of the death and recovery rates, here assumed to be constants).

Plugging all that together, we finally obtain the system of equations:

$$\begin{cases} S'(t) = -\alpha S(t) I(t) \\ I'(t) = \alpha S(t) I(t) - \beta I(t) \\ R'(t) = \beta I(t) \end{cases}$$

with α, β positive constants, and with the initial condition:

$$S(0) = S_0 > 0 \quad and \quad I(0) = I_0 > 0$$

2.2 Equilibria

Let us try to look into the possible equilibria that our system could reach:
We know that at the equilibrium $(S^\star, I^\star, R^\star)$:

$$\begin{cases} -\alpha S^\star I^\star = 0 \\ (\alpha S^\star - \beta) I^\star = 0 \\ \beta I^\star = 0 \end{cases}$$

Which leads to the set of equilibria:

$$\Big\{ (l, 0, 1-l) : l \in [0, 1) \Big\}$$

In fact, every situation where $I = 0$ is an equilibrium. This can be explained by the uniqueness of the direction of the flow of individuals between compartments, but we will come back to this later.

2.3 Solution

The solution of the system above is given by:

$$\begin{cases} S(t) = S_0 \, e^{-\alpha \tau(t)} \\ I(t) = 1 - S(t) - R(t) \\ R(t) = R_0 + \beta \tau(t) \end{cases}$$

with τ a function of time satisfying the *ODE*:

$$\tau'(t) + \beta \tau(t) + S_0 \, e^{-\alpha \tau(t)} + R_0 = 1 \tag{1}$$

(Check out section *"A.1"* of appendix for complete proof)

2.4 Qualitative study

Since τ is the key function of our solution, it is interesting to plot $\tau(t)$ and $\tau'(t)$ so that we can deduce the variation of S, I and R.

Using a detailed study of τ, we distinguish two different cases depending on the value of $\rho = \frac{\beta}{\alpha}$. We we get the following variations of $\tau(t)$ and $\tau'(t)$:

First case: if $S_0 < \rho$

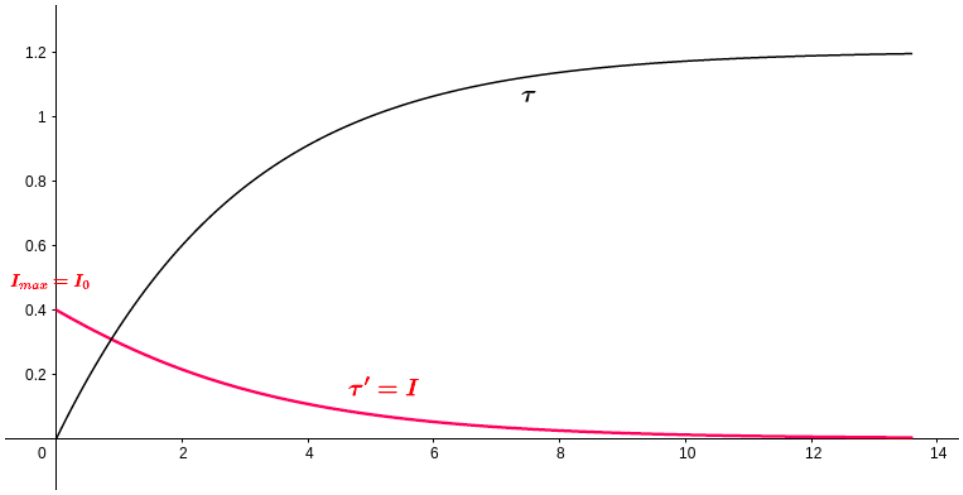


Figure 1

Second case: if $S_0 > \rho$

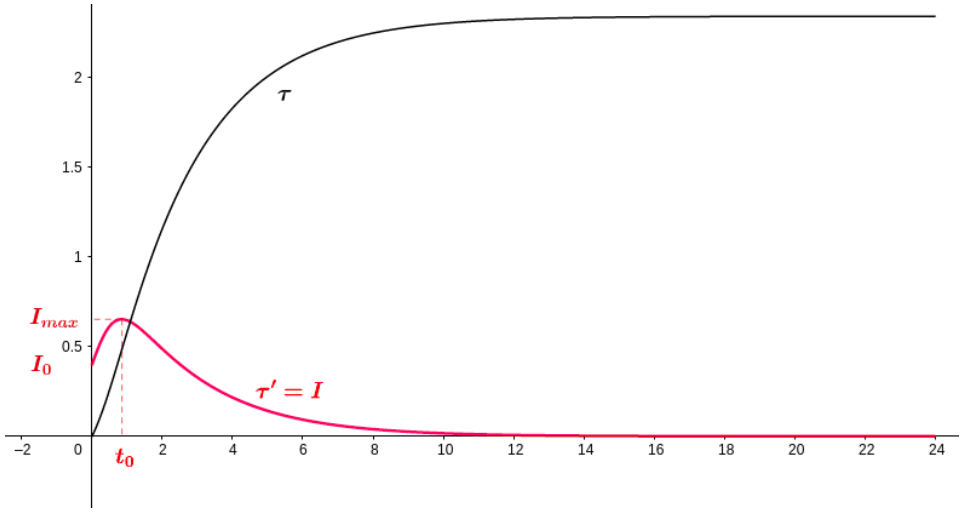


Figure 2

(Check out section *"A.2"* of appendix for the complete study leading to these plots.)

2.5 Numerical simulation

Now that we have the graph of the solution $\tau(t)$ and of its derivative $\tau'(t)$, we can predict the behavior of our system. In fact, the plot of $I(t)$ is given by the one of $\tau'(t)$, and the one of $R(t)$ has the same shape as the one of $\tau(t)$. Let us plot the variables S, I and R as a function of time.

Note about the simulations:

All the simulations and graphs in this document were coded in python3. Along with this PDF should be attached a Jupyter notebook with all the well-explained functions used to simulate the model. The timeline in the representations is not reliable. In fact, since the transmission/removal rate are given here without any unit of time, the timeline cannot be interpreted.

First, let us assume that 20% of the population is infectious at $t = 0$, then:

Transmission rate: $\alpha = 2$
Removal rate: $\beta = 0.3$

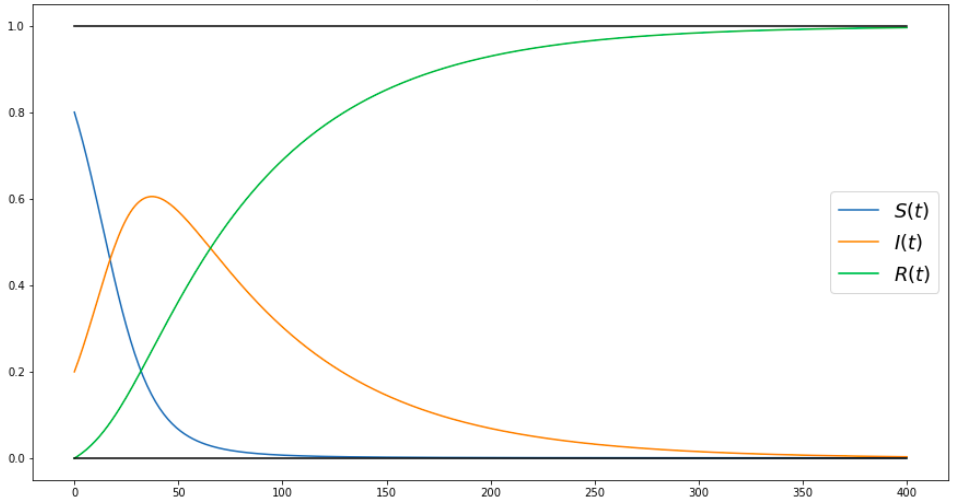


Figure 3

The proportion of susceptible individuals decreases while the one of the infectious ones increases to reach its maximum $I_{max} \approx 60\%$ at t_0. Thus, the proportion of the removed individuals keeps increasing, while the one of the infectious ones decreases down to 0, ending the pandemic ($I(t) \approx 00\%$).

Now let us consider a higher transmission rate α:

Transmission rate: $\alpha = 5$
Removal rate: $\beta = 0.3$

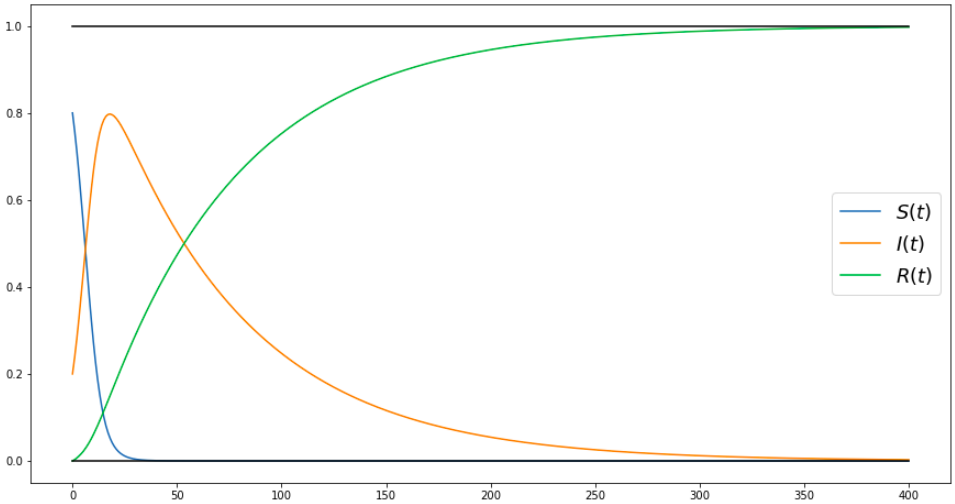


Figure 4

Here, the proportion of infectious individuals reaches a much higher value ($I_{max} \approx 80\%$) in a much shorter time frame, but the pandemic takes approximately as much time to end. This is explained by the fact that a lot of people get infected at the beginning because of the high transmission rate.
In real life, the transmission rate α can be changed as the government decides to which degree or how the social distancing measures are implemented. The more safety precautions are respected by the people, the less the transmission rate is consequent.
Hence, the government has to decide, based on data from real life, to what extent it has to implement social distancing. For example, if the government has enough hospital beds for only 60% of the population, then it has to take safety precautions such that $\alpha < 2$ (as we saw that for $\alpha = 2$ we got $I_{max} = 60\%$.)

Now let us consider a higher removal rate β:

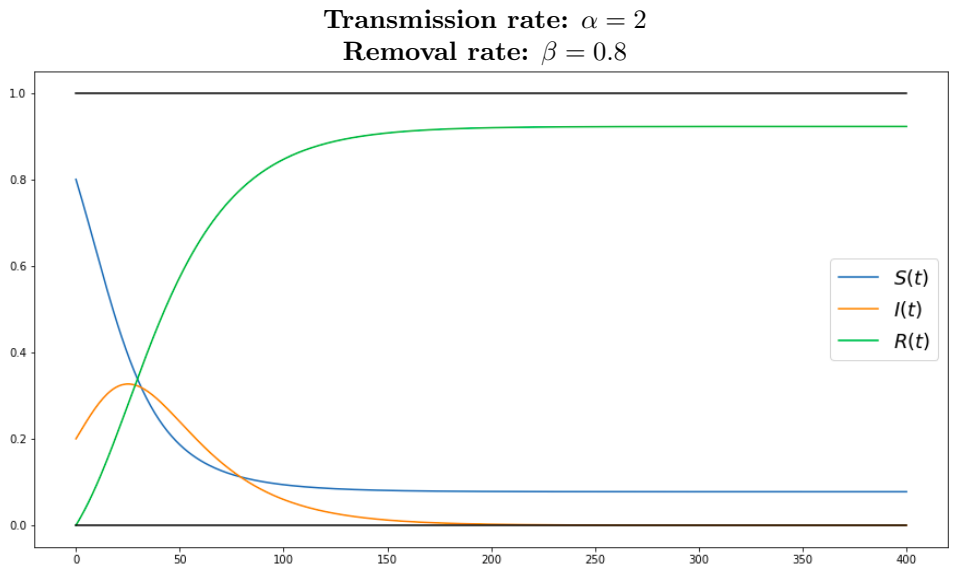


Figure 5

Here, at the end of the pandemic ($I(t) = 0$), the proportion of the susceptible individuals is still $S^\star > 0$ and the proportion of the removed individuals is also still $R^\star > 0$. This seems pretty obvious for a large removal rate (once again, remember that removed individuals are the ones that either recover or die due to the disease). In fact, infected individuals are removed so quickly that the pandemic does not affect **the whole** population.
It is interesting to notice that the removal rate β, unlike α, can be very difficult to reduce. In fact, since this constant is specific to the illness, it can only be reduced via medical achievements: treatments, vaccines, etc.

2.6 The SIR model in Real life and its plausible improvements

As mentioned previously the SIR model can be divided into only three compartments: S, I and R. This simple categorization allows us to have a primary understanding over how a disease will affect a given population over time. However, for quite simple calculations, the SIR model is most likely to over-simplify real-life infectious diseases due to its simplicity. For example, the SIR model does not take into consideration any diagnosis or incubation period. People are treated the same way which would actually have influenced on the transmission rate.

Furthermore the SIR model is based on simple assumptions. For example, it assumes a homogeneous population in which all individuals have "equal chance" of coming into contact with each other irrespective of the space and/or distance between each other. In reality, we, humans, have built social structures to stay in our own closed networks. Moreover, the SIR model assumes a closed population with no birth/death and no migration which is also not reflective in reality.

Another problem with the SIR model is that the parameters do not allow any quantification of uncertainty parameters. The inputs of the SIR model are point-estimates which means that it is up to the modeller to make his best guess with the given data. Unfortunately, these limitations make it not optimal to model real life diseases in its purest form. This is the reason why we will now look at other mathematical models that are more prone to succeed in modelling real life diseases.

3 The SIR model with Vital Dynamics

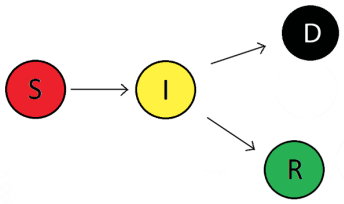
3.1 Definition

We have to admit that the model above is too simplistic for the real life. For instance, it does not take into consideration the newborns and people who die not because of the pandemic. So let us now try to introduce vital dynamics (birth and death) into our model.
For the sake of simplicity, we will assume that the size of the population does not change globally. To do that, we will not count people dying not because of the pandemic as "removed" people, but we are simply not going to count them in the population anymore. We will assume that the rate of birth μ_{birth} is equal to the rate of death not due to the pandemic μ_{death} and take:

$$\mu = \mu_{birth} = \mu_{death}$$

Wer are also going to introduce a new compartment 'D' for people who die because of the pandemic. So now the 'R' compartment is for people with immunity and the 'D' one is for dead ones. The process can hence be described as follows:

- A susceptible individual gets infected and moves to the infectious compartment.
- An infectious either dies because of the pandemic (we introduce the death rate δ), or cures and moves to the removed compartment (β is now considered to be the recovery rate).



- People keep dying in compartments (S, I and R), while newborns are added in S (we assume that the disease is not transmitted from an infected mother to her children during pregnancy.)

Hence, we get:

$$\begin{cases} S'(t) = -\alpha S(t)I(t) + \mu(S(t) + I(t) + R(t)) - \mu S(t) \\ I'(t) = \alpha S(t)I(t) - (\beta + \delta)I(t) - \mu I(t) \\ R'(t) = \beta I(t) - \mu R(t) \\ D'(t) = \delta I(t) \end{cases}$$

This system of differential equations has different solutions depending on whether $\delta \neq 0$, or $\delta = 0$ (the transmissible characteristic cannot cause death).

3.2 The case $\delta \neq 0$

3.2.1 Equilibria

Let us try and look into the possible equilibria that can be reached by this model.
We know that ($S^\star$,$I^\star$,$R^\star$, $D^\star$) is an equilibrium if and only if:

$$\begin{cases} -\alpha S^\star I^\star + \mu I^\star + \mu R^\star = 0 \\ \alpha S(t) I^\star - (\beta + \delta + \mu) I^\star = 0 \\ \beta I^\star - \mu R^\star = 0 \\ \delta I^\star = 0 \end{cases}$$

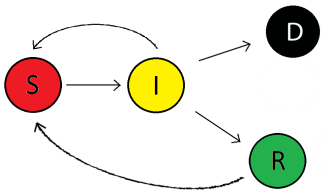
Which is equivalent to:

$$I^\star = 0 \quad and \quad R^\star = 0$$

Hence, the set of equilibria is:

$$\Big\{(l, 0, 0, 1-l) : l \in [0, 1]\Big\}$$

This can be explained by the fact that:
- Since people keep dying from S, I and R and newborns keep being added in S with the same rate $\mu_{birth} = \mu_{death}$, then this is as if individuals keep coming back to S.
- Now since there is a fraction of individuals in I that goes to D, then it is as if some individuals keep leaving this *"loop"* ($S \to I \to R \to S$) to go to D.



- At the end, all individuals end up dying, except those who did not catch the illness.

3.2.2 Numerical simulation

Let us see how all of this happens. Assume that 20% of the population is infectious at $t = 0$.

Transmission rate: $\alpha = 4$
Recovery rate: $\beta = 1.2$
Death rate (pandemic): $\delta = 0.3$
Vital dynamics rate: $\mu = 0.02$

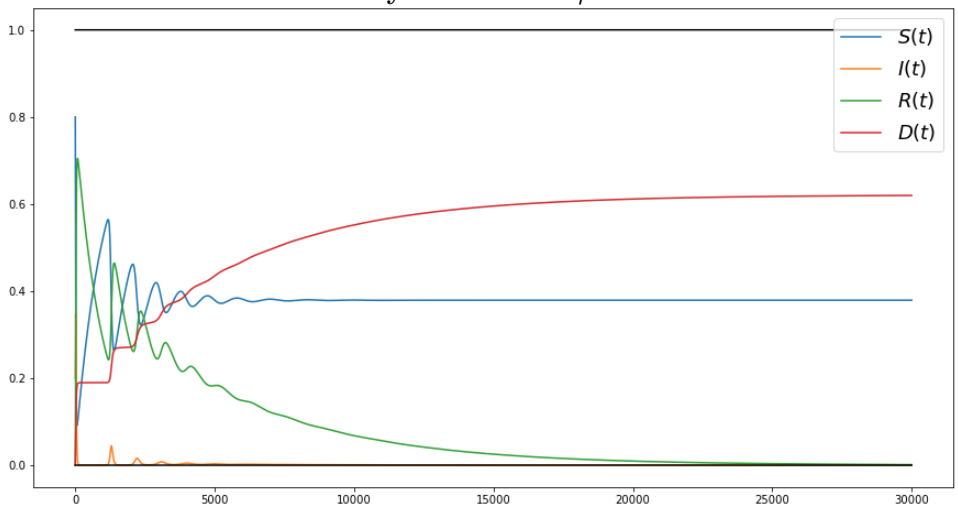


Figure 6

We see that after a lot of fluctuations, the system ends up stabilizing at:

$$(S^\star, I^\star, R^\star, D^\star) \approx (40\%, 00\%, 00\%, 60\%)$$

as predicted.

3.3 The case $\delta = 0$

In this case, the system above can be expressed as follows:

$$\begin{cases} S'(t) = -\alpha S(t)I(t) + \mu - \mu S(t) \\ I'(t) = \alpha S(t)I(t) - \beta I(t) - \mu I(t) \\ R'(t) = \beta I(t) - \mu R(t) \\ D(t) = 0 \end{cases}$$

3.3.1 Equilibria

Let us try and look into the possible equilibria that can be reached by this model.
We know that $(S^\star,I^\star,R^\star)$ is an equilibrium if and only if:

$$\begin{cases} -\alpha S^\star I^\star + \mu - \mu S^\star = 0 \\ \alpha S(t) I^\star - \beta I^\star - \mu I^\star = 0 \\ \beta I^\star - \mu R^\star = 0 \end{cases}$$

Which gives us two equilibria (we know that $\alpha \neq 0$, otherwise there is no transmission at all) :

$$(S^\star,I^\star,R^\star) = (1,\, 0,\, 0) \quad \text{or} \quad (S^\star,I^\star,R^\star) = (\frac{\beta+\mu}{\alpha},\, \frac{\mu}{\alpha}(\frac{\alpha}{\beta+\mu}-1),\, \frac{\beta}{\alpha}(\frac{\alpha}{\beta+\mu}-1))$$

We notice that here we have two possible equilibria:

1) $(S^\star,I^\star,R^\star) = (1,\, 0,\, 0)$:

This is the case where the epidemic eventually ends ($I = 0$). Due to vital dynamics, the state of the population returns to the normal ($I = 0$, $R = 0$).

2) $(S^\star,I^\star,R^\star) = (\frac{\beta+\mu}{\alpha},\, \frac{\mu}{\alpha}(\frac{\alpha}{\beta+\mu}-1),\, \frac{\beta}{\alpha}(\frac{\alpha}{\beta+\mu}-1))$:

In this case, the epidemic does not end. This is actually quite different from all the other situations we have seen so far. In fact, we were sure in the previous model that the direction of flow of individuals is unique ($\boldsymbol{S} \longrightarrow \boldsymbol{I} \longrightarrow \boldsymbol{R}$), which make numbers of the susceptible individuals S strictly decreasing.
However, here it is different: the term "$+\mu(1-S)$" in the expression of S' above compensates the term "$-\alpha SI$", making it possible for the epidemic to stay eternally.

3.3.2 Condition of termination

We have seen above that there are two possible equilibria for our model. Now the problem is: how can we predict where are we going to end up using only the characteristic constants of the pandemic and the initial state of the population?
For that, let us take:

$$R_0 = \frac{\alpha}{\beta+\mu}$$

In real life, R_0 represents the *basic reproduction number*. It designates the expected number of transmissions directly generated by one infectious individual in the population.

Our equilibria can now be expressed:

$$(S^\star,I^\star,R^\star) = (1,\, 0,\, 0) \quad \text{or} \quad (S^\star,I^\star,R^\star) = (\frac{1}{R_0},\, \frac{\mu}{\alpha}(R_0-1),\, \frac{\beta}{\alpha}(R_0-1))$$

Clearly, $R_0 = 1$ is the case where the two equilibria overlap.
Besides, if $R_0 \leq 1$, then the only possible equilibrium is $(S^\star,I^\star,R^\star) = (1,\, 0,\, 0)$.
Now assume $R_0 > 1$, and let us look into the equation:

$$I'(t) = (\alpha S(t) - (\beta+\mu))I(t)$$

Which now gives us:

$$I'(t) = \alpha I(t)(S(t) - \frac{1}{R_0})$$

Therefore:

$$\begin{cases} S(t) < \frac{1}{R_0} \implies I'(t) < 0 \\ S(t) > \frac{1}{R_0} \implies I'(t) > 0 \end{cases}$$

Hence, the condition for equilibrium is:

$$S(t) = \frac{1}{R_0}$$

Therefore, we end up at the equilibrium:

$$\begin{cases} S^\star = \frac{1}{R_0} \\ I^\star = \frac{\mu}{\alpha}(R_0-1) \\ R^\star = \frac{\beta}{\alpha}(R_0-1) \end{cases}$$

3.3.3 Conclusion

Finally, we conclude that if we take vital dynamics into consideration in our model, then the pandemic eventually ends if and only if:

$$R_0 = \frac{\alpha}{\beta + \mu} \leq 1$$

which is quite intuitive since if the number of transmissions generated by every infectious individual to be less then one, then the pandemic has to end at some time.
Otherwise, the population reaches a stable state where:

$$\begin{cases} S^\star = \frac{1}{R_0} \\ I^\star = \frac{\mu}{\alpha}(R_0 - 1) \\ R^\star = \frac{\beta}{\alpha}(R_0 - 1) \end{cases}$$

3.3.4 Numerical simulation

Now that we found the equilibrium partition of the population, let us try to visualize the model. We assume that 20% of the population is infectious at $t = 0$.
Let us start by taking an example where $R_0 = \frac{\alpha}{\beta + \mu} < 1$:

Transmission rate: $\alpha = 2$
Removal rate: $\beta = 2.1$
Vital dynamics rate: $\mu = 0.02$

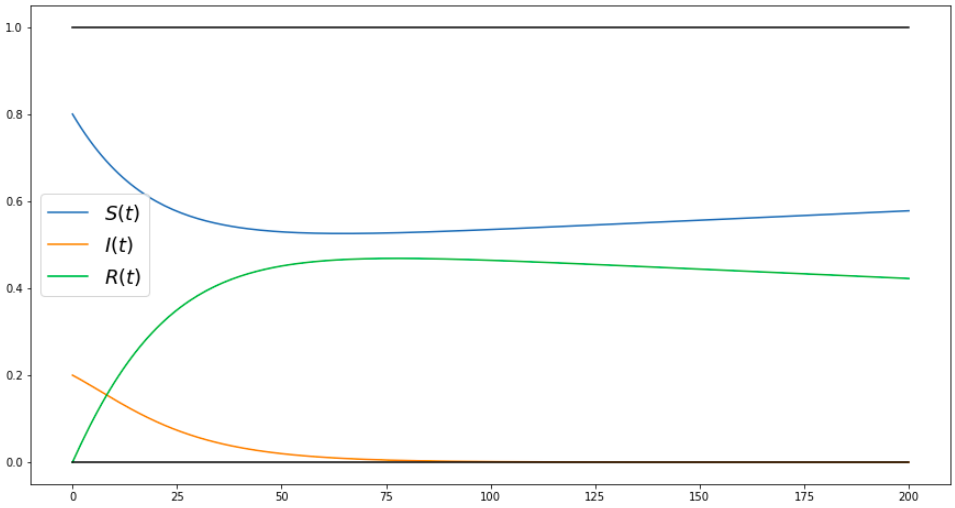


Figure 7

At the beginning (Figure 7), S and R do not seem to converge even though I converges to 0, but if we take a longer timeline:

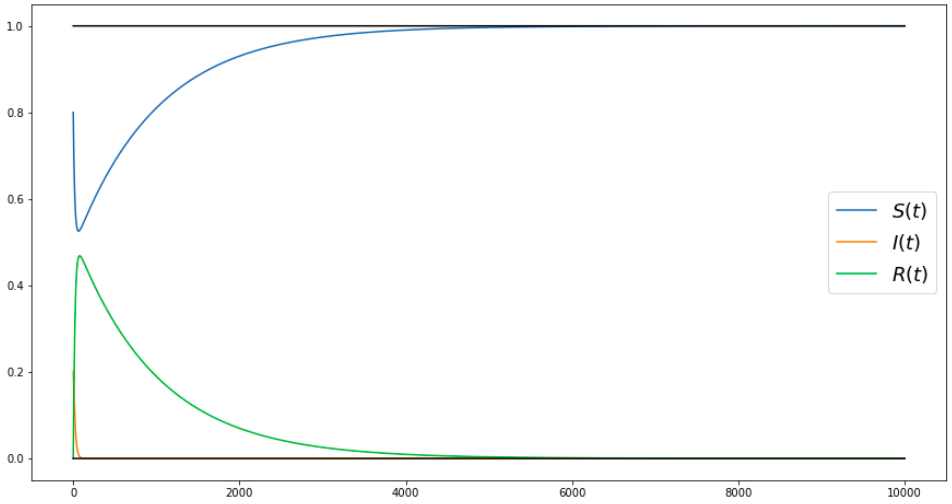


Figure 8

We can see that as predicted, the algorithm terminates ($I = 0$, $R = 0$). This can be explained by the fact that all the recovered individuals end up dying, and the new generation of individuals did not get infected.

Let us now try to change the constants of the model such that $R_0 = \frac{\alpha}{\beta + \mu} > 1$:

Transmission rate: $\alpha = 2$
Removal rate: $\beta = 0.8$
Vital dynamics rate: $\mu = 0.05$

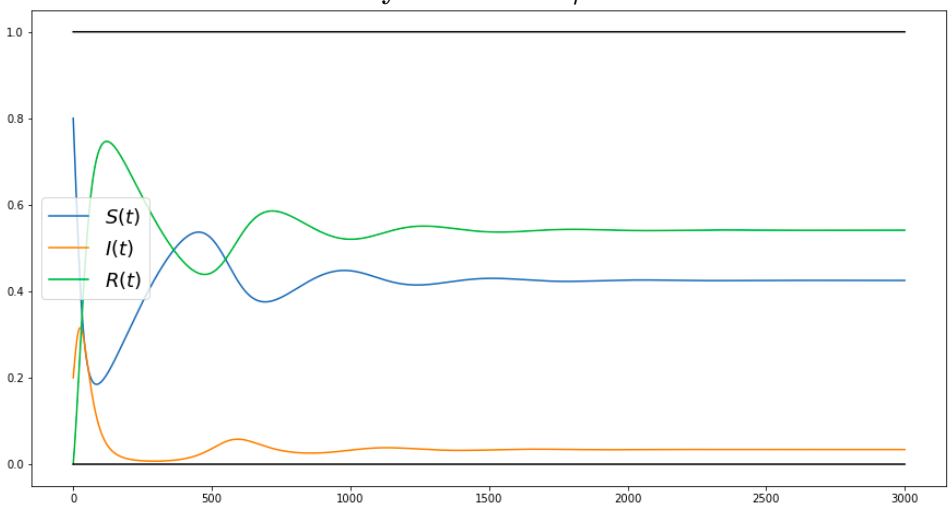
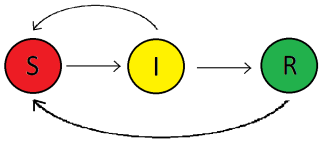


Figure 9

After a lot of oscillations, the distribution of the individuals ends up stabilizing as predicted at:

$$\begin{pmatrix} S^\star \\ I^\star \\ R^\star \end{pmatrix} \approx \begin{pmatrix} 0.425 \\ 0.034 \\ 0.541 \end{pmatrix} = \begin{pmatrix} \frac{\beta + \mu}{\alpha} \\ \frac{\mu}{\alpha^\alpha}(\frac{\alpha^\alpha}{\beta + \mu} - 1) \\ \frac{\beta}{\alpha}(\frac{\alpha}{\beta + \mu} - 1) \end{pmatrix}$$

This is actually an impressive phenomenon. In fact, one may think that the vital dynamics contribute to accelerate ending the pandemic since people keep dying in compartment I and are born in S (so in total, it is as if people keep coming back to S). However, what is happening is totally the opposite. Since people keep coming back to S, there are always susceptible individuals getting infectious!
This can be modelled by the following schema:

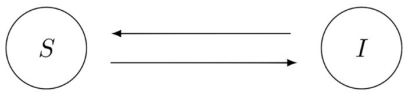


The figure above does not actually represent our situation, but as we said above, it is *"as if"* people keep coming back to S. And this explains why the pandemic never ends.

4 The SIS model

4.1 Definition

We have already seen the SIR model in its abstract form and by taking vital dynamics into consideration. However, we still cannot model every single transmissible characteristic. In fact, in real life, we observe that for many diseases, individuals may cure but cannot acquire total immunity, or do not die because of the disease. We can also think about as a physical, moral, social or psychological behaviour that be transmitted from an individual to another (not necessarily a disease). Such a behaviour does not follow the rules based on which we implemented the SIR model above, but a model where every individual is either **infectious** (has the behaviour), or **susceptible** (may catch it). The flow of individuals in the population follows the following schema:



This model can be expressed as the following:

$$\begin{cases} S'(t) = -\alpha S(t)I(t) + \beta I(t) \\ I'(t) = \alpha S(t)I(t) - \beta I(t) \end{cases}$$

4.2 Solution

This model is pretty simple since it only has two compartments (**S** and **I**).
In fact, when we substitute $I(t) = 1 - S(t)$, the problem can is to the differential equation:

$$S'(t) = \alpha(S(t))^2 - (\alpha + \beta)S(t) + \beta \tag{2}$$

Solving the differential equation above leads to the solution:

$$\begin{cases} S(t) = \dfrac{(\rho - S_0)e^{-\alpha t} + \rho(S_0 - 1)e^{-\beta t}}{(\rho - S_0)e^{-\alpha t} + (S_0 - 1)e^{-\beta t}} \\ \\ I(t) = \dfrac{(1 - \rho)(S_0 - 1)e^{-\beta t}}{(\rho - S_0)e^{-\alpha t} + (S_0 - 1)e^{-\beta t}} \end{cases}$$

where $\rho = \frac{\beta}{\alpha}$.
(Check out section *"A.3"* of appendix for complete proof).

4.3 Conclusion

Based on the expressions of $S(t)$ and $I(t)$ we found above, we can deduce the equilibrium partition of the individuals:

$$\lim_{t\to\infty} I(t) = \lim_{t\to\infty} \frac{(1-\rho)(S_0-1)e^{(\alpha-\beta)t}}{\rho - S_0 + (S_0-1)e^{(\alpha-\beta)t}} = \lim_{t\to\infty} \frac{(1-\rho)(S_0-1)e^{(1-\rho)\alpha t}}{\rho - S_0 + (S_0-1)e^{(1-\rho)\alpha t}}$$

Hence,

$$\lim_{t\to\infty} I(t) = \begin{cases} 1-\rho & if\ \rho < 1 \\ 0 & if\ \rho \geq 1 \end{cases}$$

Therefore, we conclude:

$$(S^\star, I^\star) = \begin{cases} (\rho, 1-\rho) & if\ \rho < 1 \\ (1, 0) & if\ \rho \geq 1 \end{cases}$$

4.4 Numerical simulation

Now that we found the mathematical expression of the distribution of the population as a function of time, let us try to visualize the model. We assume that 30% of the population is infectious at $t = 0$.
Let us start by taking an example where $\rho > 1$:

Transmission rate: $\alpha = 1.2$
Removal rate: $\beta = 1.3$

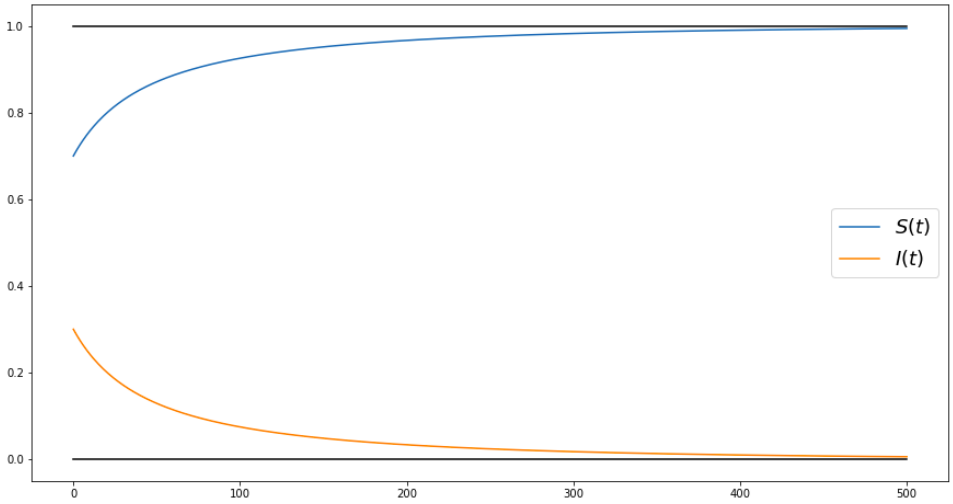


Figure 10

We can see that as predicted, the pandemic ends ($S^\star = 1$, $I^\star = 0$).

Let us now change the constants of the model such that $\rho < 1$:

Transmission rate: $\alpha = 2$
Removal rate: $\beta = 0.8$

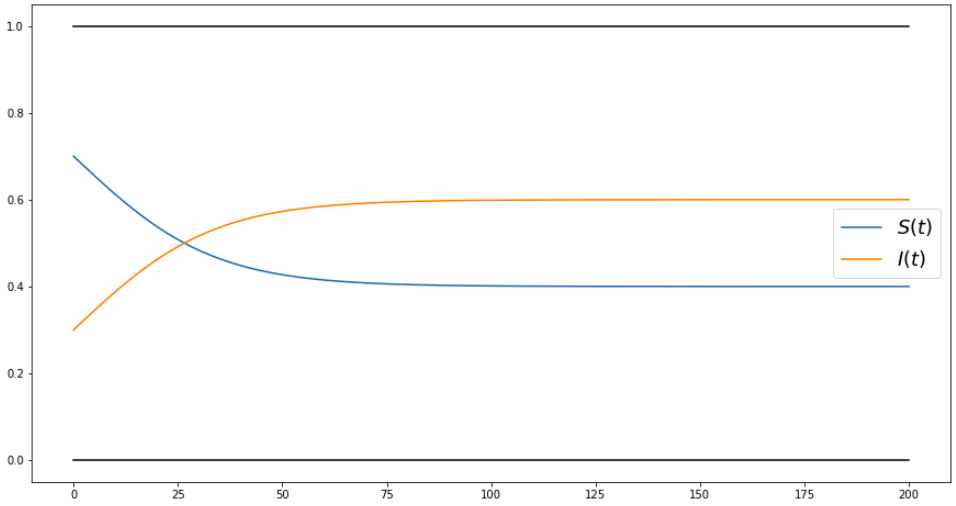


Figure 11

The distribution of the individuals ends up stabilizing as predicted at:

$$\begin{pmatrix} S^\star \\ I^\star \end{pmatrix} = \begin{pmatrix} 0.4 \\ 0.6 \end{pmatrix} = \begin{pmatrix} \rho \\ 1-\rho \end{pmatrix}$$

This is another situation where the pandemic never ends (maybe here we cannot really call it *"pandemic"* since it is not deadly, but whatever it is, it does not end).

5 Conclusion

Compartmental models are powerful tools for representing the evolution of an infectious disease in a population. They can take into account several variables which must be precise if they are to be as close as possible to real life situations.
In our project, we first decided to consider a few types of variables such as transmission and removal rates of the disease, and then to a larger extent, we took into account the vital dynamics and added the deaths to the model.

After studying different situations that could have actually occurred in a society, we found that the number of infectious individuals always reaches zero while the rate of sane (who have never been infected) and removed individuals does not always reach the extremes (0 or 1). Indeed, the whole population is not necessarily infected. We also concluded that in the event of a pandemic, the government must make decisions about security measures. Indeed, by implementing social distancing, the government can vary the transmission rate α, thus reducing the effect of the disease. However, the removal rate β may be very difficult to reduce, as this can only be done through medical discoveries: treatments, vaccines, etc.

Then, by taking into account demographic variables (birth and natural death), we obtained different results. It was interesting to see that in the case of a transmissible characteristic that cannot cause death (social/ psychological behavior for example), it is possible depending on the value of the basic reproduction number R_0 that the contagious characteristic remains in society for eternity (there will always be contagious individuals). In the case of a transmissible disease, we have seen that the pandemic ends, resulting in a new generation of people who were never infected.

With the SIS model, we distinguished two cases according to the relationship between the transmission rate and the recovery one. In one case, all individuals recover, leading to the end of the pandemic. In the other, the population stabilises at a constant distribution of susceptible and infectious individuals.

As neither two diseases nor societies are the same, we will always have different outcomes. This is why is was very interesting to consider different options and variables, and to study different interactions between the models.

A Proofs

A.1 Proof of "2.3 Solution"

Equivalently to the initial system, we can write:

$$\begin{cases} \frac{S'(t)}{S(t)} = -\alpha I(t) \\ R'(t) = \beta I(t) \\ I'(t) = -S'(t) - R'(t) \end{cases}$$

Integrating from 0 to t the first two lines:

$$\begin{cases} \ln \frac{S(t)}{S(0)} = -\alpha \int_0^t I(u)\,du \\ R(t) - R(0) = \beta \int_0^t I(u)\,du \\ I'(t) = -S'(t) - R'(t) \end{cases}$$

Setting: $\tau(t) = \int_0^t I(u)\,du$, where $\tau(t)$ is such that:

$$S(t) + I(t) + R(t) = 1$$

the solution to our problem can be expressed as the following:

$$\begin{cases} S(t) = S_0\, e^{-\alpha\tau(t)} \\ I'(t) = -S'(t) - R'(t) \\ R(t) = R_0 + \beta\tau(t) \end{cases}$$

with τ a function of time satisfying the *ODE*:

$$\tau'(t) + \beta\tau(t) + S_0\, e^{-\alpha\tau(t)} + R_0 = 1$$

A.2 Study of "2.4 Qualitative Study"

Since we know that $\tau'(t) = I(t)$, we can say that:

- $\tau'(t=0) = I_0 > 0$
- $\tau'(t) = I(t) > 0 \quad \forall\, t$
- $\lim\limits_{t\to+\infty} \tau'(t) = \lim\limits_{t\to+\infty} I(t) = 0$

The third statement can be explained by the fact that the proportion of the population represented by $S(t) + I(t)$ is strictly decreasing and non-negative, and $0 < I(t) < S(t) + I(t)$.

Hence, we can deduce the variations of $\tau(t)$:

t	0		$+\infty$
$\tau'(t)$	I_0	+	0
$\tau(t)$	0	↗	L

where L is the equilibrium value of $\tau(t)$ (τ converges since the population if finite).
Substituting in the differential equation (1) above, for $t = +\infty$:

$$\lim_{t\to\infty} (\tau'(t) + \beta\tau(t) + S_0\, e^{-\alpha\tau(t)} + R_0) = 1$$

We get the following condition satisfied by L:

$$\beta L + S_0\, e^{-\alpha L} = 1 - R_0$$

We want the maximum number of infections I_{max} and time t_0 when it will be reached. Since $I = \tau'$, let us study the variations of $\tau'(t)$. We are looking for t_0 such that:

$$\tau''(t_0) = 0$$

Differentiating the two sides in the differential equation (1) above, we get:

$$\tau''(t) + \beta\tau'(t) - \alpha S_0 \tau'(t)\, e^{-\alpha\tau(t)} = 0$$

And now substituting $t = t_0$:

$$\beta\tau'(t_0) - \alpha S_0 \tau'(t_0)\, e^{-\alpha\tau(t_0)} = 0$$

Equivalently:

$$\alpha\tau'(t_0)(\rho - S_0\, e^{-\alpha\tau(t_0)}) = 0$$

where $\rho = \frac{\beta}{\alpha}$

Since $\tau' \neq 0$, it is equivalent to:

$$\rho = S_0\, e^{-\alpha\tau(t_0)}$$

Or equivalently:

$$t_0 = -\frac{1}{\alpha} \ln \frac{\rho}{S_0}$$

Since this value only positive (as we would wish for a time value to be) when $S_0 > \rho$, we have to distinguish two cases:

First case:

Here we assume that $S_0 < \rho$:
From the differential equation, we get:

$$\tau''(t) = \alpha\tau'(t)(S_0\, e^{-\alpha\tau(t)} - \rho)$$

Since $\tau(t) \geq 0$, then $e^{-\alpha\tau(t)} \leq 1$, then:

$$\tau''(t) \;=\; \alpha\tau'(t)(S_0\, e^{-\alpha\tau(t)} - \rho) \;\leq\; \alpha\tau'(t)(S_0 - \rho) \;<\; 0$$

Hence, we can deduce the global variations of the solution:

t	0		$+\infty$
$\tau''(t)$		$-$	0
$\tau'(t)$	I_0 ↘		0
$\tau'(t)$	I_0	$+$	0
$\tau(t)$	0 ↗		L

The solution τ and its derivative $\tau' = I$ should look like this:

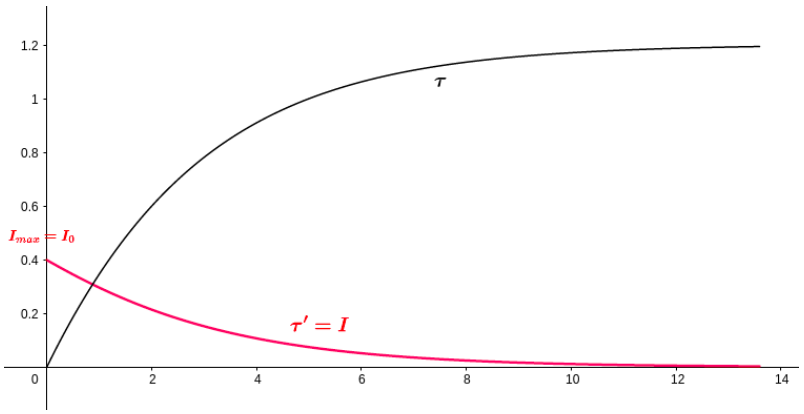


Figure 1

Second case:

Here we assume that $S_0 > \rho$:
Then:

t	0		t_0		$+\infty$
$\tau''(t)$		$+$	0	$-$	0
$\tau'(t)$	I_0	↗	I_{max}	↘	0

Hence, the solution τ and its derivative $\tau' = I$ should look like this:

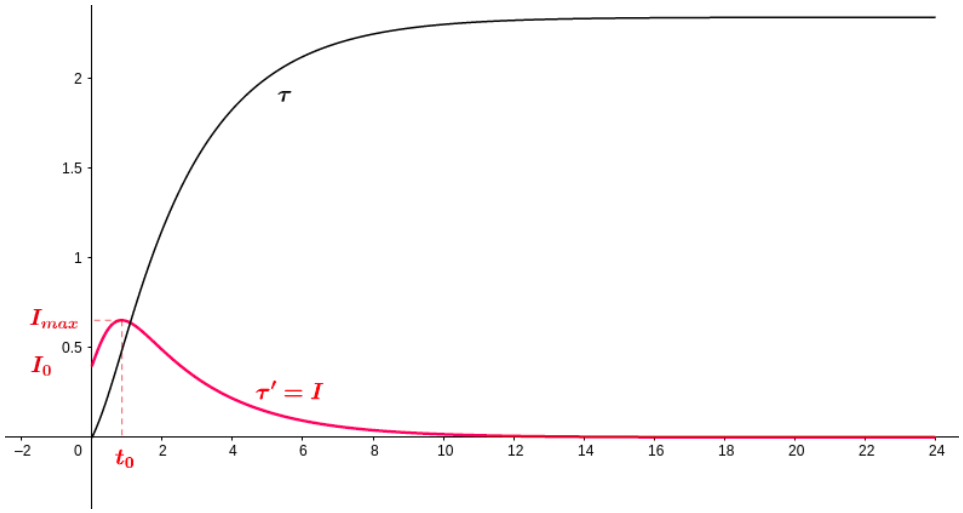


Figure 2

A.3 Proof of "4.2 Solution"

We have established that out system can be reduced to:

$$S'(t) = \alpha(S(t))^2 - (\alpha + \beta)S(t) + \beta$$

Let $u(t)$ be such that $\forall t \geq 0$:

$$u(t) = e^{-\alpha \int S(t)\, dt}$$

In fact, using this substitution , we get:

$$u'(t) = -\alpha S(t)u(t)$$

and

$$u''(t) = -\alpha S'(t)u(t) - \alpha S(t)u'(t)$$

Hence,

$$u''(t) = \alpha u(t)(\alpha(S(t))^2 - S'(t))$$

Now using the equality given by equation (2), we get:

$$u''(t) = \alpha u(t)((\alpha + \beta)S(t) - \beta)$$

Which is equivalent to:

$$u''(t) = -(\alpha + \beta)(-\alpha S(t)u(t)) - \alpha\beta u(t)$$

Hence, equation (2) can be expressed as:

$$u''(t) + (\alpha + \beta)u'(t) + \alpha\beta u(t) = 0$$

Which is a 2^{nd} order homogeneous linear differential equation with constant coefficients. Its solution is given by:

$$u(t) = c_1 e^{r_1 t} + c_2 e^{r_2 t}$$

Where r_1 and r_2 are the solutions of

$$r^2 + (\alpha + \beta)r + \alpha\beta = 0$$

which are clearly $(r_1, r_2) = (-\alpha, -\beta)$.
Hence,

$$u(t) = c_1 e^{-\alpha t} + c_2 e^{-\beta t}$$

Now returning back to the original variable:

$$S(t) = -\frac{u'(t)}{\alpha u(t)} = \frac{\alpha c_1 e^{-\alpha t} + \beta c_2 e^{-\beta t}}{\alpha c_1 e^{-\alpha t} + \alpha c_2 e^{-\beta t}}$$

Which leads to:

$$\begin{cases} S(t) = \dfrac{c_1 e^{-\alpha t} + \rho c_2 e^{-\beta t}}{c_1 e^{-\alpha t} + c_2 e^{-\beta t}} \\ \\ I(t) = 1 - S(t) = \dfrac{(1-\rho) c_2 e^{-\beta t}}{c_1 e^{-\alpha t} + c_2 e^{-\beta t}} \end{cases}$$

Where $\rho = \frac{\beta}{\alpha}$.

Using the initial condition $S(0) = S_0$:

$$S_0 = \frac{c_1 + \rho c_2}{c_1 + c_2}$$

Which gives:

$$c_2 = \frac{S_0 - 1}{\rho - S_0} c_1$$

Hence:

$$\begin{cases} S(t) = \dfrac{(\rho - S_0)e^{-\alpha t} + \rho(S_0 - 1)e^{-\beta t}}{(\rho - S_0)e^{-\alpha t} + (S_0 - 1)e^{-\beta t}} \\ \\ I(t) = \dfrac{(1-\rho)(S_0 - 1)e^{-\beta t}}{(\rho - S_0)e^{-\alpha t} + (S_0 - 1)e^{-\beta t}} \end{cases}$$

where $\rho = \frac{\beta}{\alpha}$.