SIR Models and Neural Networks in Epidemiology

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Contents

1 Abstract

1.1 Epidemics and Epidemiology

An epidemic is defined as an unusually large, short-term disease outbreak. There are various factors influence a disease's spread from person to person, which include the infectious agent itself, its mode of transmission, infectious period, and its susceptibility and resistance to treatments and vaccines. There are asymptomatic infectives, mostly unreported, and the proposed algorithm learns the proportion of the total infective individuals that are asymptomatic infectives.

Infectious diseases progress within populations both due to the behavior of the infectious agent and the population itself. Models of how they progress in an epidemic are based on a set of assumptions and statistics which are used to establish a set of parameters that inform how effective intervention will be (i.e, social distancing or mass vaccination).

The study of epidemics, general disease, and even health conditions that are not caused by disease is called epidemiology. The origins of the term lie in ancient Greece with the physician Hippocrates of Kos, who was the first to make the distinction between epidemic and endemic diseases. Epidemiology is broadly termed the science of public health involving quantitative investigation of disease outbreak with the aim of controlling epidemics. It provides essential quantitative and analytical methods, principles of logical inquiry, and rules for evidence for disease management. It helps in measuring and projecting the health needs of community and populations and determining how to allocate and manage health care resources.

1.2 Epidemiology Models

There is a familiar term in mathematical models which describes the simplified representations of real-wrold processes expressed in mathematical language. In epidemiology, models are often used the way infectious diseases spread through populations.

The complexity of epidemiologic models varies. They can be simple deterministic models or complex spatially explicit stochastic simulations. The approach chosen by epidemiologists depends on several variables including how much is known about the disease's epidemiology, the purpose of the study, and the amount of data available, and its quality.

2 Introduction

In December 2019, a new respiratory illness began to spread throughout Wuhan, China. The virus responsible for this illness is the SARS-CoV-2 and the disease is called COVID-19. It quickly spread through Wuhan, a city of 11 million people in Hubei province. It infected tens of thousands of people over the ensuing weeks. China imposed major restrictions on travel and work, and by the end of February, cases of COVID-19 had slowed inside the country while spiking all over the world. The appearance of COVID-19 made 2020 a very memorable year. It affected nearly all of the countries in the world, leading to quarantines and regulations that the people of the 21st century have never experienced before, while also delivering a heavy blow to the global economy, thus resulting in people losing their jobs.

On 20 February 2020, a locally acquired coronavirus disease (COVID-19) case was detected in Lombardy, Italy. This was the first signal of ongoing transmission of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in the country. The number of cases in Italy increased rapidly and the country became the first in Europe to experience a SARS-CoV-2 outbreak. In subsequent days and weeks, case counts and death tolls increased rapidly, at first in northern Italy and then in the rest of the country. Not so simiral cases started to unfold across Europe in several countries, such as Germany, France and the United Kingdom.

Many researching teams around the globe started looking at the data concerning the recent events, in search of a prediction model about the virus spread rate which could be beneficial for every country in the world. The parameters around their research were country profiles, confirmed COVID-19 cases, policy responses, hospitalizations and mortality risk.

The goal of our research is to show that mathematical models, which are used frequently in investigating disease spread and epidemiology can be applied to the training of neural network models as a pretraining step, resulting in a more accurate model. In the following pages, we will unfold the benefits of SIR models and artificial neural networks for epidemiology studying and giving predictions, based on real-world data.

3 Mathematical and Epidemiological Models

3.1 Mathematical Models

Mathematical models are key tools in many scientific fields (Magnani & Casadio, 2016; Weisberg, 2013). Models help the researchers to learn about target systems, explain observed phenomena, organise knowledge, develop concepts, etc. The mathematical models are usefull for experimenting in order to obtain information and test hypothesis. However, the data for infectious diseases are not easily interpreted due to several parameters which may vary and the conducted experiments are often incomplete due to underreporting. This lack of reliable data makes accurate parameter estimation difficult so that it may only be possible to estimate a range of values for some parameters, although calculations can easily be done for variety of parameter values and datasets.

The transmission mechanism from an infective to susceptibles is understood ror nearly all infectious diseases and the spread of diseases through a chain of infections is known. However, the transmission interactions in a population are very complex so that it is difficult to comprehend the large scale dynamics of disease spread without the formal structure of a mathematical model. An epidemiological model uses a microscopic description (the role of an infectious individual) to predict the macroscopic behavior of disease spread through a population.

Epidemiological models are useful in comparing the effects of prevention or control procedures. Communicable disease models are often the only practical approach to answering questions about which prevention or control procedure is most effective. Quantitative predictions of epidemiological models are always subject to some uncertainty since the models are idealized and the parameter values can only be estimated. However, predictions of the relative merits of several control methods arc often robust in the sense that the same conclusions hold over a broad range of prameter values and a variety of models.

3.2 Epidemiological Models during COVID-19 outbreak

During the COVID-19 pandemic, maybe even more clearly than in previous health crises, epidemiological models have played a crucial role (Eubank et al., 2020; Holmdahl & Buckee, 2020; Rhodes et al., 2020). They have been used to predict the evolution of the pandemic, to estimate the effect of health interventions, to anticipate side effects, etc. In the United Kingdom, for instance, the government's approach to the pandemic was mainly informed

by epidemiological models run at the Imperial College London. In this sense, an update to a model related with intensive-care unit (ICU) bed occupancy (from 15 to 30% of hospital cases), which had a great impact on the number of predicted deaths, was highly relevant to the implementation of the first lockdown on 23th March (Adam, 2020).

Epidemiological models with constant parameters may not capture satisfactory infection patterns in the presence of mitigation measures during a pandemic, since infectiousness is a function of time. There are asymptomatic infectives, mostly unreported, and the proposed algorithm learns the proportion of the total infective individuals that are asymptomatic infectives. Besides that, the symptomatic infectives' data are usually enough to simulate our models and make valuable conclusions for the situation which is examined. This pattern was helping most researchers and analysts of global events, regarding the COVID-19 crisis, at the early 2020, while the virus was constantly spreading and there was little to no understanding of its components, let alone a cure to it.

Later on, several vaccines were reported to be eligible for the public. However, not everyone was supposed to get vaccinated, depending on his condition and health status. The immediate goal of the global COVID-19 vaccination strategy was to minimize deaths, severe disease and overall disease burden; curtail the health system impact; fully resume socio-economic activity; and reduce the risk of new variants. Thus, there was a need for an optimal vaccination strategy which directed many researchers to epidemiological modeling helping the World Health Organisation(WHO) identify the vaccination strategy by seperating the global population into compartments based on their age, health conditions, susceptibility etc.

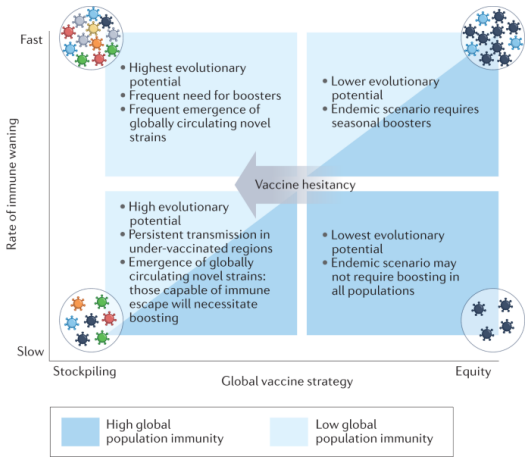

Figure 1: Vaccination hesitancy

4 SIR Model

SIR is a general virus spread model that can be easily interpreted and it was presented by Kermack and McKendrick in 1927. It is a set of three ordinary differential equations.Furthermore, in terms of behaviour it is a non-linear system. It is primarily recommended to be used for viruses where infected individuals cannot develop long-lasting immunity after recovery. Depending on the variables taken into account, there are several SIR models and we will have a closer look into SIR model, whether asymptomatics are considered as a variable in a study.

4.1 Basic Assumptions

The basic assumptions about an epidemiology model, i.e SIR model are the following:

1. The population considered has constant size N which is sufficiently large so that the sizes of each class can be considered as continuous variables. If the model is to include vital dynamics, then it is assumed that births and natural deaths occur at equal rates and that all newborns are susceptible.

2. The population is homogenously mixing. That translates as all infected individuals transmit the disease at rate β and the recipient is chosen uniformly at random from the population.

3. Individuals recover and are removed from the infective class at a rate proportional to the number of infectives with proportionality constant γ, called the daily recovery removal rate. The latent period is zero (it is defined as the period between the time of exposure and the time when infectiousness begins).

4. The basic reproduction number σ is defined as the average number of people an infected person can infect. When the basic reproduction number is less than one, the infectious disease progressively vanishes. In the SIR model the basic reproduction number is computed as the ratio of the transmission rate to the recovery rate. There are two cases, regarding the state of σ, where the transmission and recovery rates are constants, thus the basic reproduction number is given by the ratio of the transmission rate to a weighted sum of the symptomatic and asymptomatic recovery rates, or the transmission rate is time-varying, then we use a modified reproduction number which demonstrates the spread pattern of COVID-19 throughout the duration of the pandemic.

4.2 Symptomatic SIR model

First, we concentrate in the simpliest form of an SIR model which **does not** consists asymptomatics as a factor. This SIR model is a set of three ordinary differential equations which try to describe the rate of change in relation to three different compartments in a particular population: susceptible (S), infected and infectious (I), and recovered and neither able to be infected again nor to spread the disease (R).

$$\frac{dS(t)}{dt} = -\beta S(t)I(t)$$
$$\frac{dI(t)}{dt} = \beta S(t)I(t) - \gamma I(t)$$
$$\frac{dR(t)}{dt} = \gamma I(t)$$

The parameters β and γ are the *infectivity rate* and the *recovery rate*, respectively. The β parameter depends on the number of contacts an infected individual has per time unit (κ) and the *transmission rate*, that is, the probability of transmitting the infection to susceptible contacts (τ). In addition, N is the total poulation and σ_1 is the basic reproduction number which play a big part in defining the SIR model. Then,

- $\beta = \kappa \cdot \tau$
- $\gamma = \frac{1}{D}$, where D is the duration of the infection
- $S(t) + I(t) + R(t) = 1$, for every time point
- $\sigma_1 = \frac{\beta}{\gamma}$

4.3 Mathematical Interpretation of the Symptomatic SIR Model

Lemma 4.1 *Given the fact that $S(0), I(0), R(0) > 0$, the solutions of the ordinary differential equations are positive for every time point $t > 0$.*

Proof Let $A(t) = min\{S(t), I(t), R(t)\}, \forall t > 0$ with $A(t) > 0$.

Assuming that there is a $t_1 > 0$ such that $A(t_1) = 0$ and $A(t) > 0, \forall t \in [0, t_1)$.

Without loss of generality, $A(t_1) = S(t_1)$, thus $I(t) \geq 0, R(t) \geq 0, \forall t \in [0, t_1]$.

Given the equations of our model, we obtain $\frac{dS}{dt} \geq -\beta S(t)I(t)$, $t \in [0, t_1]$,

leading to $S(t) \geq S(0) \cdot e^{-\int_0^{t_1} \beta S(t)I(t)\,dt} \geq 0 \Longrightarrow S(t) > 0$, $\forall t \geq 0$.

Similarly, we prove that $I(t) > 0$ and $R(t) > 0$, $\forall t \geq 0$. □

Lemma 4.2 *The solutions of the ordinary differential equations are bounded.*

Proof Since the the sum of the susceptible (S), infective (I) and recovered (R) is equal to 1, we conclude that every population group is bounded. Therefore, every solution is obtained from the model's system of equations is bounded. □ Hence, the solutions of the SIR model's equations are positive and bounded $\forall t \in [0, t_1]$.

4.3.1 Solution Approximation

Kermack and McKendrick have created a method for advanced problem-solving during short periods of time. We will attempt to use it for a short period of COVID-19 epidemic in order to obtain an analytical expression for the solutions to the SIR equations.

Our estimations will be valid in cases of small recovered (R) to recovery rate(γ) ratio until $R(t) \ll \gamma$. This analytical expression can be compared to early epidemiological data and used to estimate the parameter γ. Afterwards, we are able to study our model numerically, although there is a possibility of error in our calculations/estimations.

Given the SIR model's equations, we could make several observations about our variables:

- $\frac{dS}{dR} = -\frac{S}{\gamma} \Longrightarrow S(R) = S_0 \cdot e^{-(R-R_0)/\gamma}$
- $\frac{dI}{dR} = -1 + \frac{S}{\gamma} \Longrightarrow I(R) = I_0 + S_0(1 - e^{-(R-R_0)/\gamma}) - (R - R_0)$

We conclude that the susceptible (S) and the infective (I) population can be described as functions of R. Now, we introduce a new variable,

$$P(t) := R(t) - R_0, \quad where \quad P(0) = 0 \quad and \quad \frac{dP}{dt} = \frac{dR}{dt}$$

Thus, the infective (I) can be described best as

$$I(P) = I_0 + S_0(1 - e^{-P/\gamma}) - P$$

If we combine the equation above with the differential equation of the infectives (I) , the final result is

$$\frac{dP}{dt} = \beta \cdot [I_0 + S_0(1 - e^{-P/\gamma}) - P]$$

4.3.2 Equilibrium Points

An equilibrium point is one where the state variables are constant and unchanging. Since the derivatives represent changes in the state variables, this statement is equivalent to saying the derivatives for the model are 0 at equilibrium points. In epidemiology, we are interested in two kinds of equilibrium points:

1. **Disease-Free Equilibrium** : The total infectives are zero.
2. **Endemic**[1] **Equilibrium Points** : Endemic-steady point, $\sigma_1 \cdot S = 1$.

When the total infectives (I) are zero, then $I = 0$ for every point of time t. Therefore, we can solve the ordinary differential equations system, claiming a unique equilibrium point because we are dealing with a set on linear differential equations. Otherwise, in search of endemic equilibrium points, we assume that the group of infectives contains a part of the total population.

4.3.3 Vital Dynamics

It is common for many researchers-authors to ignore one of the three groups (usually the recovered population) that describe our model, in order to study β and γ. Thus, we examine the infectivity rate and the recovery rate of the population N, having in mind that $\mathbb{S} = \{(S, I) : S \geq 0, I \geq 0, S + I \leq 1\}$. According to that, whenever the number of the infectives is zero, we observe an equilibrium of the system, thus there is a continium of equilibria.

Theorem 4.1 *Let the point (S_0, I_0) be a starting condition of our set of differential equations.*
a) If $\sigma_1 \cdot S_0 \leq 1$, then $I(t) \to 0$ as $t \to \infty$.
b) Otherwise, $I(t)$ increases at first and then decreases to 0, while $S(t) \to S_\infty$, where S_∞ is the unique solution in (0,1/σ_1) of the equation

$$1 - S_\infty + \frac{ln(S_\infty/S_0)}{\sigma_1} = 0.$$

Proof The differential equations we are working on are the following,

$$\frac{dS}{dt} = -\beta S(t) I(t)$$

$$\frac{dI}{dt} = \beta S(t) I(t) - \gamma I(t)$$

[1] A disease outbreak is endemic when it is consistently present but limited to a particular region

Dividing the second equation by the first, we obtain the following equation,

$$\frac{dI}{dS} = -1 + \frac{\gamma}{\beta \cdot S} \Longrightarrow dI = -dS + \frac{dS}{\sigma_1 \cdot S}$$

Assuming that $R(0) = 0$ and $I(0) + S(0) = 1$, then the solution of the equation we obtained before is

$I = 1 - S + \frac{ln(S/S_0)}{\sigma_1}$, where $I_0 = I(0)$ and $S_0 = S(0)$

Therefore, when $t \to \infty$, the differential equations are tending to zero and $I_\infty = 0$, leading us to the requested equation. □

Theorem 4.2 *For a given σ_1, S_0 with $\sigma_1 \cdot S_0 > 1$ and $R(0) = 0$, the maximum value of $I(t)$ occurs when $S(t) = 1/\sigma_1$, given by*

$I_{max} = C(\sigma_1) - \frac{ln(S_0)}{\sigma_1}$, *where* $C(\sigma_1) := 1 - \frac{1 + ln(\sigma_1)}{\sigma_1}$

Proof We will look for the values that solve the equation

$$\frac{dI}{dt} = 0 \Longrightarrow I(t)(\beta S(t) - \gamma) = 0 \Longrightarrow I(t) = 0 \quad or \quad S(t) = \frac{\gamma}{\beta} = \frac{1}{\sigma_1}$$

The maximum number of infectives cannot be zero, so the solution of the equation is $S(t) = 1/\sigma_1$ and subtituting this value into

$$I = 1 - S + \frac{ln(S/S_0)}{\sigma_1}$$

we obtain

$$I_{max} = 1 - \frac{1}{\sigma_1} + \frac{ln(1/(\sigma_1 S_0))}{\sigma_1} = 1 - \frac{1}{\sigma_1} - \frac{ln(\sigma S_0)}{\sigma_1} = 1 - \frac{1 + ln\sigma_1}{\sigma_1} - \frac{lnS_0}{\sigma_1}$$

Thus, we get the requested equation, given the hypothesis for $C(\sigma_1)$. □

Lemma 4.3 *When the population of infected is "maximized", then the total of the infected and the recovered is equal to*

$$I(t) + R(t) = 1 - S(t) = \frac{\sigma_1 - 1}{\sigma_1}$$

4.4 Asymptomatic SIR model

Classical SIR dynamic model and its derivative improved model may not accurately describe the epidemic situation similar to COVID-19 with characteristics of relative long incubation period and a large number of asymptomatic infections. Based on that, we introduce the asymptomatic SIR model which assumes that some of the infectives are asymptomatic infectives. This group is infectious despite not showing symptoms of COVID-19, probably are not tested, and are usually unreported in the various publicly available data.

The asymptomatic-SIR model considers the following population compartments: the Susceptible (S), the symptomatic Infectives (I) which correspond to the reported infectives in the publicly available data, and the asymptomatic Infectives (J) which correspond to the unreported infectives. The total infectives are $I + J$. Basically, those who went through an asymptomatic infection and are removed from the epidemic dynamics only once they are naturally healed. The model assumes that both classes of infectives are equally infective. Therefore, there are the symptomatic Recovered (R) and the asymptomatic Recovered (U). The symptomatic Infectives (I) recover at the rate γ through isolation, and the asymptomatic Infectives (J) recover at the rate μ spontaneously. The SIR model, in this case, is best described as it follows:

$$\begin{aligned}
\frac{dS(t)}{dt} &= -\beta(I(t) + J(t))S(t) - \nu S(t) \\
\frac{dI(t)}{dt} &= \beta\xi(I(t) + J(t))S(t) - \gamma I(t) \\
\frac{dJ(t)}{dt} &= \beta(1-\xi)(I(t) + J(t))S(t) - \mu J(t) \\
\frac{dR(t)}{dt} &= \gamma I(t) + \nu\xi S(t) \\
\frac{dU(t)}{dt} &= \mu J(t) + \nu(1-\xi)S(t)
\end{aligned}$$

In addition, $S(t) + I(t) + J(t) + R(t) + U(t) = 1$, $t \geq t_0$ and the initial conditions are denoted by $S(t_0) = S_0$, $I(t_0) = I_0$, $J(t_0) = J_0$, $R(t_0) = R_0$ and $U(t_0) = U_0$, where $t \geq t_0$.
At this point, the vaccinated population ($V = \nu S$) plays an important role in the model, as we experience a loss from the susceptible compartment and an addition to the recovered compartments. Furthermore, the transmission rate β depends on the infection vector and contacts between individuals.

In addition, ν is the average percentage of individuals that are vaccinated daily and ξ represents the probability that an infective individual is reported, while $(1-\xi)$ is the probability that an infective is an asymptomatic infective. Based on the information above, the portion of the total infectives that are symptomatic and reported corresponds to $\xi(I+J)$, while $(1-\xi)(I+J)$ represents the asymptomatic infectives.

4.5 Mathematical Interpretation of the Asymptomatic SIR Model

Lemma 4.4 *Given the fact that $S(0), I(0), J(0), R(0), U(0) > 0$, the solution of the ordinary differential equations are positive for every time point $t > 0$.*

Proof Let $B(t) = min\{S(t), I(t), J(t), R(t), U(t)\}, \forall t > 0$ with $B(t) > 0$.

Assuming that there is a $t_1 > 0$ such that $B(t_1) = 0$ and $B(t) > 0$, $\forall t \in [0, t_1)$.

Without loss of generality, $B(t_1) = S(t_1)$, thus $I(t) \geq 0$, $J(t) \geq 0$, $R(t) \geq 0$ and $J(t) \geq 0$, $\forall t \in [0, t_1]$.

Considering the equations of our model, we obtain

$$\frac{dS}{dt} \geq -\beta(I(t) + J(t))S(t) - \nu S(t),\ t \in [o, t_1],$$

leading to $S(t) \geq S(0) \cdot e^{-\int_0^{t_1} \beta(I(t)+J(t))S(t)+\nu S(t)\, dt} \geq 0 \Longrightarrow S(t) > 0$, $\forall t \geq 0$.

Similarly, we prove that $I(t) > 0$, $J(t) > 0$, $R(t) > 0$, $U(t) > 0$ $\forall t \geq 0$. □

Lemma 4.5 *The solutions of the ordinary differential equations are bounded.*

Proof Since the sum of the susceptible (S), infective $(I + J)$ and recovered $(R + U)$ is equal to 1, we conclude that every population group is bounded. Therefore, every solution of the equation system that describes our model is bounded. □

Hence, the solutions of the SIR model's equations are positive and non-negative $\forall t \in [0, t_1]$.

4.5.1 Solution Approximation

We will use again the Kermack and McKendrick method for advanced problem-solving during a short period of COVID-19 epidemic, in order to obtain an analytical expression for the solutions to the SIR equations. Our estimations will be valid when each recovered to recovery rate ratio is small

until their sum tends to 0. In mathematical terms, we have

$$\frac{R(t)}{\gamma} + \frac{U(t)}{\mu} < 1, \quad until \quad \frac{R(t)}{\gamma} + \frac{U(t)}{\mu} \simeq 0$$

This analytical expression can be compared to early epidemiological data and used to estimate γ. Afterwards, we are able to study our model numerically, although there is a possibility of error in our calculations/estimations. However, we are taking into account the asymptomatic part of the population, hence the possible error will be reduced, compared to the symptomatic model. We need to group the total infectives and the total recovered in order to make several observations.

- $\frac{dI}{dt} + \frac{dJ}{dt} = \beta(I(t) + J(t))S(t) - \gamma I(t) - \mu J(t)$
- $\frac{dR}{dt} + \frac{dU}{dt} = \gamma I(t) + \mu J(t) + \nu S(t)$

In this case, we cannot relate the total infectives and recovered because the recovery rate is not similar for the symptomatics and asymptomatics.

4.5.2 Equilibrium Points

An equilibrium point is one where the state variables are constant and unchanging. Since the derivatives represent changes in the state variables, this statement is equivalent to saying the derivatives for the model are 0 at equilibrium points. In epidemiology, we are interested in two kinds of equilibrium points:

1. **Disease-Free Equilibrium** : The total infectives $(I + J)$ are zero
2. **Endemic**[2] **Equilibrium Points** : Endemic-steady point, $\sigma_2 \cdot S = 1$.

When the total infectives (I) are zero, then $I + J = 0 \Longrightarrow I = J = 0$ for every point of time t. Therefore, we can solve the ordinary differential equations system, claiming a unique equilibrium point because we are dealing with a set on linear differential equations. Otherwise, in search of endemic equilibrium points, we assume that the total infectives are not null. Searching for the equilibrium points requires several assumptions about our model, which we will make in the following section.

[2] A disease outbreak is endemic when it is consistently present but limited to a particular region

4.5.3 Vital Dynamics

It is common for many researchers-authors to ignore the total recovered population that describe our model, in order to study β and γ. Thus, we examine the infectivity rate and the recovery rate of the population N, having in mind that $\mathbb{S}_2 = \{(S, I, J) : S \geq 0, I \geq 0, J \geq 0, S + I + J \leq 1\}$. The set of equations we are remaining interested in for our study,

- $\frac{dS}{dt} = -\beta(I(t) + J(t))S(t) - \nu S(t)$
- $\frac{dI}{dt} = \beta\xi(I(t) + J(t))S(t) - \gamma I(t)$
- $\frac{dJ}{dt} = \beta(1 - \xi)(I(t) + J(t))S(t) - \mu J(t)$

Considering the system of equations we are studying, we make certain claims about the equilibrium points.

1. Whenever the number of the infectives is zero, we observe an equilibrium of the system, hence there is a continium of equilibria. If the total infectives are 0, we obtain a disease-free equilibrium point $E_0 = (1, 0, 0)$

2. In case of an endemic equilibrium point, we observe that $\beta > \gamma$ which translates to $\sigma_2 > 1$, showing that the basic reproduction ratio is not low enough for us to claim that the epidemic is showing any signs of declining.

Theorem 4.3 *If $\sigma_2 \leq 1$, then the disease-free equilibrium point of the system is globally asymptomatically stable on $\mathbb{S}_2$.*

Proof The Lyapunov function $V : \mathbb{S}_2 \to \mathbb{S}_2$, $V(S, I, J) = I(t) + J(t)$ is a useful tool for the search of an equilibrium point, hence we are going to use that scalar function. [3]

$$\frac{dV}{dt} = \frac{dI}{dt} + \frac{dJ}{dt} = \beta(I(t) + J(t))S(t) - \gamma I(t) - \mu J(t)$$

Examining which values of the basic reproduction ratio of our model (σ_2) influence the value of the function's sign,

$$\frac{dV}{dt} \leq 0 \Longleftrightarrow \beta(I(t) + J(t))S(t) \leq \gamma I(t) + \mu J(t) \Longrightarrow$$

$$I(t)\Big(S(t) - \frac{\gamma}{\beta}\Big) + J(t)\Big(S(t) - \frac{\mu}{\beta}\Big) \leq 0 \Longrightarrow S(t) - \frac{\gamma}{\beta} \leq 0 \quad \textit{and} \quad S(t) - \frac{\mu}{\beta} \leq 0$$

[3] A function associating a single number to every point in a space

Considering that $\sigma_2 = \dfrac{\beta+\mu}{\gamma}$, we draw the conclusion about σ_2 :

$$S(t) \leq \frac{1}{\sigma_2} \Longrightarrow S(t)\cdot\sigma_2 \leq 1 \Longrightarrow \sigma_2 \leq 1$$

Based on these results, if $\sigma_2 = 1$, then $V'(t) = 0$, thus the disease-free equilibrium point is globally asymptomatically stable, considering the Lasalle invariance principle. □

Theorem 4.4 *The endemic equilibrium point $E(S_k, I_k, J_k)$ of the system is globally asymptomatically stable on $\mathbb{S}_2$.*
Proof Once again we are going to use a Lyanupov function $L : \mathbb{S}_{2+} \to \sigma_2$ where $\mathbb{S}_{2+} = \{S(t), I(t), J(t) \in \mathbb{S}_2 : S(t) > 0, I(t) > 0, J(t) > 0\}$ and

$$L(S,I,J) = S(t) - S_k \cdot ln\Big(\frac{S(t)}{S_k}\Big) + I(t) - I_k \cdot ln\Big(\frac{I(t)}{I_k}\Big) + J(t) - J_k \cdot ln\Big(\frac{J_k}{J(t)}\Big)$$

Following the same approach as before, we calculate the derivative of our function L,

$$\frac{dL}{dt} = -\frac{dS}{dt}\cdot\frac{S_k}{S(t)} - \frac{dI}{dt}\cdot\frac{I_k}{I(t)} - \frac{dJ}{dt}\cdot\frac{J_k}{J(t)}$$

We will examine the value of $E(S_k, I_k, J_k)$, regarding the sign of the function,

$$\frac{dL}{dt} < 0 \Longrightarrow \frac{dS}{dt}\cdot\frac{S_k}{S(t)} + \frac{dI}{dt}\cdot\frac{I_k}{I(t)} + \frac{dJ}{dt}\cdot\frac{J_k}{J(t)} > 0, \text{ true } \forall E \in \mathbb{S}_{2+}.$$

Given that result, the endemic equilibrium point is globally saymptomatically stable on $\mathbb{S}_{2+}$. □

5 Basic Stochastic Model

Firstly, we need to define the term "stochastic basis", in order to construct a **stochastic model** that relies on the asymptomatic SIR model we studied in section **4.4**. Thus, there will be enough data in order to examine thouroughly and construct a basic stochastic model.

Definition 5.1 *A filtration on a measurable space (Ω, F) is a nondecreasing family $F = (F_t)_{t\in\mathbb{R}_+}$ of sub-σ algebras of the σ-algebra $F : F_s \subseteq F_t \subseteq F$ for all $s < t$, while $s, t \in \mathbb{R}$. A stochastic basis is a probability space equipped with a filtration and if a discrete filtration is given, then it can be extended to a filtration in a natural way.*

Definition 5.2 *A stochastic basis $B = (\Omega, F, F_t, P)$ is a complete if the σ-algebra F_0 contains all sets of measure 0 from F. We say that a stochastic basis $B = (\Omega, F, F_t, P)$ satisfies the usually demanded conditions if it right-continuous and complete.*

For time point $t > 0$, let $Y(t)$ denote the number of confirmed to be reported on day t. Assuming that $Y(t) \sim N(\mu(t), \sigma_t^2)$, a confirmed case only refers to the symptomatics, although we are going to work with the asymptomatic SIR model. The estimation of possible infectives with no symptoms is closer to reality, thus the results are higher regarded compared to those the classic SIR model would produce. If we consider N_c the average proportion of symptomatic cases that are confirmed on any given day, then $I(t)/N_c$ represents the mean number of confirmed cases on day t. Hence, it is clear that the mean value of the stochastic sequence $\mu(t) = I(t)/N_c$ is connected to the A-SIR model of ours.

The goal of our model would be to make efficient and useful predictions about reported cases, based on past and present data. Thus, we set as a fundamental unit of time to be a day ($t_0 =$ day 1) and

$$Y(t) = \{y(t) : t = t_0, t_1, \ldots, t_n\}$$

Based on the asymptomatic SIR model we introduced, we consider the following stochastic model,

$$\begin{aligned}
dS(t) &= -\beta(I(t) + J(t))S(t)dt - \nu S(t)dt - \sigma S(t)(I(t) + J(t))dW(t) \\
dI(t) &= \beta\xi(I(t) + J(t))S(t)dt - \gamma I(t)dt + \sigma S(t)I(t)dW(t) \\
dJ(t) &= \beta(1-\xi)(I(t) + J(t))S(t)dt - \mu J(t)dt + \sigma S(t)J(t)dW(t) \\
dR(t) &= \gamma I(t)dt + \nu\xi S(t)dt \\
dU(t) &= \mu J(t)dt + \nu(1-\xi)S(t)dt
\end{aligned}$$

where W is a real-valued continuous-tme stochastic process defined on a stochastic basis $(\Omega, F, (F)_{t\geq 0}, \mathbf{P})$ and -as referred above- $(\Omega, F, \mathbf{P})$ can be described as probabililty space. Therefore, $Y(t)$ can be considered an adapted non-negative stochastic process with discrete time.

Theorem 5.1 *If $0 < \kappa < \beta - \sigma^2/2$, then the disease-free equilibrium point $E_0 = (1, 0, 0)$ of the stochastic model is globally asymptomatically stable.*

6 Neural Networks

A neural network is a method in artificial inttelligence (AI) that teaches computers to process data in a way that is inspired by the human brain. It is a type of machine learning process, called deep learning, that uses interconnected nodes or neurons in a layered structure that resembles the human brain. It creates an adaptive system that computers use to learn from their mistakes and improve continuously. Thus, artificial neural networks attempt to solve complicated problems, like summarizing documents or recognizing faces, with greater accuracy.

Theorem 6.1 *(Universal Approximation Theorem) A neural network with a hidden layer can approximate any continuous function for inputs within a specific range.*

According to this theorem, if you accept most classes of problems can be reduced to functions, this statement implies a neural network can, in theory, solve any problem. If human intelligence can be modeled with functions (exceedingly complex ones perhaps), then we have the tools to reproduce human intelligence today.

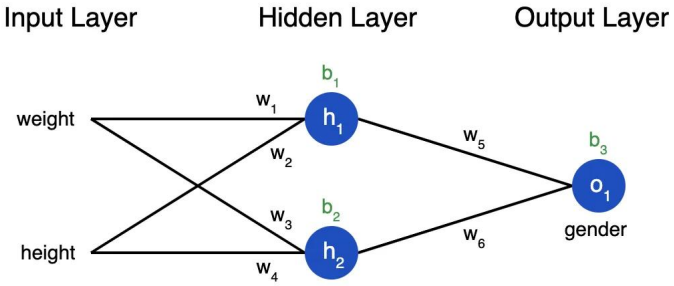

Figure 2: Basic Neural Network

According to Figure 2, if we exclude all the actiovation layers from the above network, we realize that h_1 is a linear function of both weight and height with parameters w_1, w_2 and the bias term b_1. Therefore,

$$h_1 = w_1 \cdot weight + w_2 \cdot height + b_1$$
$$h_2 = w_3 \cdot weight + w_4 \cdot height + b_2$$

So, o_1 is also a linear function of h_1 and h_2, depending on input attributes weight and height as well. This boils down to a linear regression model.

6.1 Activation Functions

However, it is necessary for a neural network to learn, represent and process any data and any arbitary complex function which maps the inpus to the outputs, besides a simple linear model. According to the **Universal Approximation Theorem**, any imaginable process can be presented as a functional computation in neural nets.

Therefore, it is urgent to apply an activation function to create a dynamic network, which is able to extract information, regardless of the given task. An important feature of an activation function is that it must be differentiable so that we can implement back propagation optimization strategy in order to compute the errors or losses with respect to weights and eventually optimize weights using **Gradient Descend** or any other optimization technique to reduce errors.

Since there are many classes that define a neural network, we need to categorize the available activation functions, based on their own properties, thus we get the following :

1. **Binary Step Function**
2. **Linear Function**
3. **Sigmoid Function**
4. **Tanh Function**
5. **ReLU Function**
6. **Leaky ReLU Function**
7. **Parametrized ReLU Function**
8. **Exponential Linear Unit Function**
9. **Swish Function**
10. **SoftMax Function**

However, activation functions can be also divided based on dot products and distance measures. Therefore, we will take a closer look at their characteristics.

6.1.1 Activation Functions-Dot Products

The activation of a static neuron in a network based on the dot product is the weighted sum of inputs x_j multiplied with the weights of each input respectively. In mathematical terms, that mean that

$$f(w_j, x) = \vec{W}_i^T \cdot \vec{X} = net = \sum_{j=1}^{n} w_{ij} \cdot x_j$$

If this equation is equal to zero, it is described as a hyper-plane through the origin of a coordinate system. The activation increases whenever the distance of an input x from the hyperplane increases, otherwise it will decrease. In addition, the sign of the function's value indicates the side of the plane the input is located, hence the sign seperates the input space into two dimensions. A general application of the hyperplane requires that each neuron i has a threshold θ_i, which results in the definition of the hyperplane

$$\sum_{j=1}^{n} w_{ij} \cdot x_j - \theta_i = 0$$

6.1.2 Activation Functions-Distance Measures

There are activation functions based on distance measures, in which the weight vectors of neurons represent the data in the input space. The activation of a neuron i is computed from the distance of the weight w_i from the input x. Determining this distance is possible by applying different measures, such as spatial distance, maximum/minimum distance and manhattan distance.

6.1.3 The choice of an Activation Function

The perfomance of an artificial neural network in a study is key, hence the researchers are constantly looking for the best one, according to the situation. In every case, a lot of things have to be considered like the number of hidden layers in a network, training methods, hyperparameter tuning and activation function is one of the most important parameters to consider.

There isn't a certain rule or a fromula for selecting any activation function but the choice of activation function depends on the task that is to be accomplished. Different Activation Functions have both advantages and disadvantages of their own and it depends on the type of system that we are designing.

6.2 Artificial Neural Network

An Artificial Neural Network model (ANN) is a mathematical evolutionary approach that makes decisions (provides solutions) and a well-documented AI model based on the organization concept of signals transmission in the human nervous system. It is a collection of connected units or nodes called artificial neurons, which loosely model the neurons in a biological brain. Each connection, like the synapses in a biological brain, can transmit a signal to other neurons. An artificial neuron receives signals then processes them and can signal neurons connected to it. The "signal" at a connection is a real number, and the output of each neuron is computed by some non-linear function of the sum of its inputs. The connections are called edges. Neurons and edges typically have a weight that adjusts as learning proceeds. The weight increases or decreases the strength of the signal at a connection. Neurons may have a threshold such that a signal is sent only if the aggregate signal crosses that threshold. Artificial Neural Networks can identify the relationships among dependent and independent variables, which helps in understanding system function.

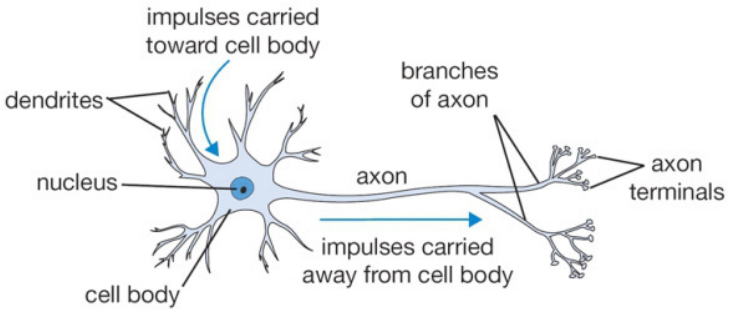

Figure 3: Biological Neuron

The basic model consists of three interconnected layers including the input, hidden, and output layers. Neurons of input and hidden layer connect to all neurons in the subsequent layer. Neurons of input layers take input data-set from the real environment that is modeled. Finding the output of the neuron presupposes the weighted sum of all the inputs, weighted by the weights of the connections from the inputs to the neuron. We add a bias term to this sum and this weighted sum is then passed through a (usually nonlinear) activation function to produce the output.

The input vector x_j is transmitted using a connection that multiplies its strength by a weight w_{ij} to produce the product. Each neuron has a bias θ_j (Figure 3). The output of the neuron is generated by transfer and activation functions.

An activation function consists of linear and non-linear algebraic formulas. These two functions enable the neural network to find the relationships between input and output. The difference between the output and neural network output is considered as an error.

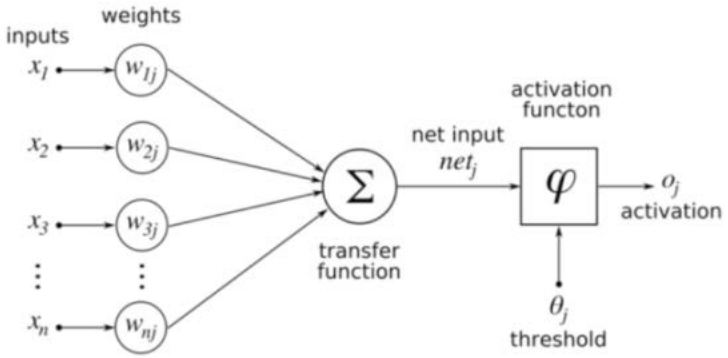

Figure 4: Artificial Neural Network

An Artificial Neural Network can be mathematically shown to be universal function approximators, meaning that they can automatically approximate whatever functional form best characterizes the data.In addtion, it can estimate piece-wise approximations of functions. Although these properties are of little value if the functional form is simple, it allows our model to extract more signal from complex underlying functional forms, plus it is also able to partially transform the input data automatically.

The use of articial neural networks requires the analysts to decide on algorithms and parameters to facilitate effectiveness and efficiency of model solution strategy. An articial neural network is applied in different fields of knowledge to solve different problems of optimization, classification, and prediction. In the medical domain, it has been efficient for clinical diagnosis, image/signal analysis and interpretation and drug development. In epidemiology, it has successfully been used to study dynamics, risk, growth, severity, and control of infectious diseases. In epidemic forecasting network architecture, all layers and nodes are fully connected, that is, each neuron in one layer is connected to all neurons in the following layer.

There is a wide application of neural networks in diagnostic approaches in medicine including image analysis, drug design, biochemical analysis, and diagnostic systems was considered. Different techniques and methods have been developed to investigate epidemics including classification, dynamics, forecast, and intervention or control strategy optimization.

6.3 Epidemic Classification

In a mathematical sense, this involves dividing an n-dimensional space into various regions, with a given point in the space one should tell which region it belongs to using an Artificial Neural Network. Each pattern is transformed into a multi-dimensional point and is classified to a group, each of which represents a known pattern. Clinical observations of diseases tend to be similar at some early stages; further refined characteristics of diseases have to be identified in order to correctly classify a patient or victim of specific infectious diseases. The use of Artificial Neural Network has had tremendous achievements in epidemic classification.

6.4 Epidemic Forecasting

Forecasting prevalence of diseases using an Artificial Neural Network algorithm is a widely accepted approach for epidemiologic investigation of the risk and outbreak size of diseases. The incidence and prevalence of a disease can be predicted in order to assess the impact and requirement of control measures.

Its architecture for epidemic forecast is problem-dependent, based on the decision variables and parameters of the problem investigated. The number of input nodes, hidden layers and nodes, and output nodes would determine the network architecture for forecasting. The mutual information between variables can help inform the choice of structure.

Applying an Artificial Neural Network in epidemic forecast requires analysts' decisions that enhance forecast performance. These decisions required choosing from approaches or techniques of an artificial neural network algorithm, such as decisions on data pre-processing, network architecture or structure, the number of input, hidden layers or output nodes, training algorithms, specification of training parameter, the number of epoch runs, and accuracy measurement tools.

The best-perfoming forecasting model is preferred for any research, therefore forecast consistency measures are needed. The most common measure is the mean square error (MSE),

$$MSE = \frac{1}{n}\sum_{i=1}^{n}(y_i - \hat{y}_i)^2$$

which mesaures the average deviation between the predicted value ($\hat{y}$) and the actual value (y). Comparing our eastimations's MSE, we go for the algorithm with the smaller mean square error, since it ensures us for the best possible result.

6.5 Model Construction

An artificial neural network possesses features analogous to those of a biologic neuron. Neurons are logical structures composed of two parts; the first part receives incoming information (inputs) from possibly many sources, and the second part mathematically transforms the input into output information (outputs). The biological neurons's connections are given by the synapses and the equivalent of that term in artificial neural networks are the weights. Each neuron converts the patterns of inputs that it receives to a single output by multiplying each input with the weight on the connection and summing all these weighted inputs to get a quantity called "net."

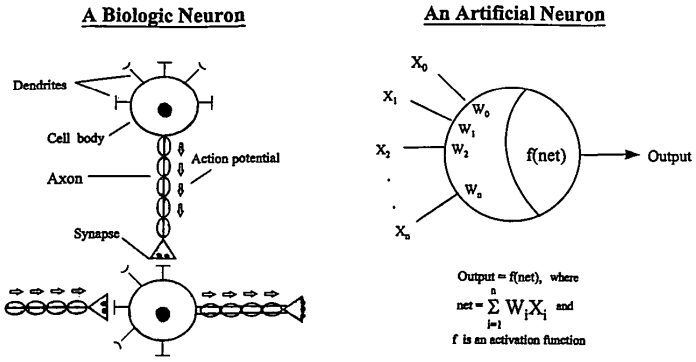

Figure 5: Biological Neuron and Artificial Neuron

We will construct a model, based on mathematical terms,

- **Input vector** : $\vec{X} = (x_1, x_2, \ldots, x_n)$
- **Weight vector** : $\vec{W_i} = (w_{1j}, w_{2j}, \ldots, w_{nj})$
- **Output vector** : $\vec{o_j} = (o_{1j}, o_{2j}, \ldots, o_{nj})$
- **Net** : $\sum_{i=1}^{n} \vec{W_i} \cdot \vec{X} = \vec{W}^T \cdot \vec{X}$
- **Activation function** : $f(\sum_{i=1}^{n} \vec{W_i} \cdot \vec{X})$

According to the information we obtained from the mathematical terms that describe our model, we conclude that $f(net) = \vec{o_j}$. It is clear to see the activation function, $\forall j \in \{1, \ldots, m\}$, results in the output o_j, $\forall j \in \{1, \ldots, m\}$ respectively.

An activation function connects the weights of the neurons to the inputs and determines the activation or the state of the neuron.

6.6 Model Development

The development of a neural net model generally consists of three steps. The first step is to generate the data for the training. The second step is the training of the neural net with the selected data. The third step (testing) involves exposure of the trained network to unfamiliar dataset and consequently the accuracy in the predicted pattern is adjudged. The training process is commonly conducted by the flexible architecture of the network, including three layers,

1. **An input layer**
2. **A hidden layer**
3. **An output layer**

The first and the third ones contain neurons associated with the input and output vectors, respectively. On the other hand, neurons in the hidden layer, which are connected with the neurons of the input and output layers, are basically responsible for turning the input data into the corresponding output data. Additionally, they transfer a weighted summation of the input data using a transfer function.

Generally, inside a layer of an artificial neural network connections between layers are allowed, while inter-layered connections are prohibited. The data flow through the network continues until a relation with a desired precision is obtained. Finally, the better our model is trained, the more accurate results may be achieved.

In our case, the asymptomatic SIR model is our priority, thus we will be using the variables,

- Susceptible (S)
- Symptomatic Infectives (I)
- Symptomatic Recovered (R)

as the input layer, since these groups's numbers are reported, and

- Asymptomatic Infectives (J)
- Asymptomatic Recovered (U)

as the hidden layer, because the asymptomatics are not reported and we can only make assumptions about those groups.

7 Herd Immunity

The term "Herd immunity", also known as population immunity, is the indirect protection from an infectious disease that happens when a population is immune either through vaccination or immunity developed through previous infection. The **World Health Organisation** (WHO) supports achieving 'herd immunity' through vaccination, not by allowing a disease to spread through any segment of the population, as this would result in unnecessary cases and deaths.

A population is said to have herd immunity for a disease if enough people are immune so that the disease would not spread if it were suddenly introduced somewhere in the population. If the population is homogeneously mixing and the immune people are distributed uniformly in the population, then herd immunity will be obtained if a large enough uniformly distributed fraction is immune.

In mathematical terms, herd immunity is achieved when $\sigma(1 - V) < 1$. This equations refers to the vaccinated part of the total population (V) and the basic reproduction ratio (σ) as key factors of herd immunity. Basically, if the virus cannot spread among the group of unvaccinated people expontentailly, then the disease will either become **endemic** or **cease to exist**. We consider that the total infectives (sympotmatic or not) are null ($I = 0$), therefore the susceptibles are equivalent to the unvaccinated group ($S = 1 - V$).

8 Appendix

8.1 Epidemiology Models's Guidelines

The need of understanding the spreading patern of a virus brings us to the epidemiology models. Since those models are best described as compartmental, we divide the population into compartments and there are rules set in order to classify the progress of every person between them.

The choosing of the compartments is usually crucial to the final result, hence we have to evaluate the population groups and create a model efficent enough for our case. Therefore, we start with the simpliest one, the SIR model, which can provide valuable results and we can add up to it. According to the resources and the data given for the reasearch, the appropriate model has to be picked or "constructed" in order to accomplish our goal. The methodology should be described based on solid criteria, hence the final estimations/results have validation.

8.2 Reminder of σ-algebra

Definition 8.1 *Let Ω a subset of $\mathbb{R}$ and $P(\Omega)$ its power set. A quantity system $A \subseteq P(\Omega)$, which can be described as a set of subsets of Ω, is called a σ-algebra, under the following conditions :*

1. *A contains the base set, thus $\Omega \in A$.*
2. *A is stable with respect to complement formation, meaning that $B \in A$ as well as $B_c = (\Omega \setminus B) \in A$.*
3. *A is stable with respect to countable unions, meaning that for a countable set $A_1, A_2, \ldots \in A \Longrightarrow \cup_{n\in\mathbb{N}} A_n \in A$.*

A σ-algebra F is complete relative to P if $A \in F$, $P(A) = 0$, $B \subseteq A$ given that $B \in F$. Therefore, it is necessary for a stochastic basis to be right-continuous, in order to develop a stochastic model.

8.3 Wiener Process

Definition 8.2 *The Wiener process is a real-valued continuous-time stochastic process, which is often also called **Browian motion**. The Wiener process W_t is characterised by the following properties,*

1. *$W_0 = 0$*
2. *For every $t > 0$, the future increments $W_{t+u} - W_t$, $u \geq 0$ are independent of the past values W_s, $s < t$*
3. *W has Gaussian increments, meaning that $W_{t+u} - W_t$ $N(0, u)$*
4. *W_t is continuous in t*

8.4 Gradient Descent

Definition 8.3 *In mathematics, gradient descent is a first-order iterative optimization algorithm for finding a local minimum of a differentiable function. The idea is to take repeated steps in the opposite direction of the gradient (or approximate gradient) of the function at the current point, because this is the direction of steepest descent.*

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