UNIVERSIDAD DE INVESTIGACIÓN DE TECNOLOGÍA EXPERIMENTAL YACHAY

Escuela de Ciencias Matemáticas y Computacionales

TÍTULO: Mathematical modeling and simulation of the dynamics of the SARS-Cov-2 virus

Trabajo de integración curricular presentado como requisito para la obtención del título de Matemático

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To Efrain, Esther, Nicole, Sofia, Mia and Holley. Every single good part inside of me is because of you.

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This work ends a journey that began 5 years ago. Going to live away from home for the first time made me realize that the bond between people who really love each other can be stronger than any distance. My friends from my hometown have never missed me, although each one has taken their course after finishing high school, the "crew" has always been present and I hope they will never be absent. No one can achieves anything alone. I am not a person who make too many friends but in Yachay Tech I found a new family, my mathematical family. The career would not have been fun at all without the extraordinary generation that I am part of. The 4G family is made up of talented, hard-working and fun people. I am sure that each of us will imprint our footstep wherever we walk. And what would have happened to me without the teachers who did not let me fall into the dark side of the force? My co-tutor and teacher of so many subjects, that I already lost the number, Juan Mayorga has been a headache, almost all along the ride, because every night that I could not sleep due to his challenging reading controls, but above all he has been an inspiration to continue for this road. It is clear that his thesis-students of my generation consider him as an academic father and we hope to always be at the height of everything he taught us. This work would have been impossible without his mentoring in conjunction with professor Paul Medina, whom I must also thank for all the predisposition and patience that he has had throughout the project. I have left my parents and sisters to the end. Each and every one of my achievements is dedicated to them. We live in a world full of uncertainty but at the end of the day, they are the only safe thing I have in my life. They are the perfect family for me.

.....
Ray Anthony Romero Romero
April, 2021.

Abstract

By the end of 2019 a novel virus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), appeared by the first time in Wuhan, China. Its fast spread around the world led in March 2020 to the World Health Organization (WHO) to declared the SARS-Cov-2 virus a pandemic. Epidemics and pandemics are not new phenomenons in human history, and mathematical models have been used to describe the dynamics of this infectious diseases. In this document we present a SEIR generalized model with no-demographic constraints. In this model we add asymptomatic, asymptomatic recovered, isolated and dead classes. We perform simulations in 4 data-driven contact networks: workplaces, households, general community and agglomeration places. The studies of Cuevas et al., Peng et al. and Quang et al. are relevant for this work. We decided to consider all parameters as constants with exception of the cure and mortality rate, nevertheless in future works this parameters will be modeled as functions of time for more accurate predictions. In particular, we focus in the behaviour of asymptomatic, infected and dead classes in each one of the data-driven networks. We notice that the curves change in terms of which one of them reaches faster the peak of the curve. Finally, we compute the reproduction number, R_0, using the next-generation approach for each one of the networks. Here, for every network we obtain $R_0 > 1$ which agrees with the theory. It is important to mention that the lack of transparency in the data by govern makes hard to built this kind of models.

Keywords— SARS-Cov-2, mathematical models, SEIR, data-driven networks, simulations, basic reproduction number.

Resumen

A finales del 2019 un nuevo virus, síndrome respiratorio agudo 2, SARS-CoV-2 por sus siglas en inglés, apareció por primera vez en Wuhan, China. Su rápida dispersión alrededor del mundo llevó a la Organización Mundial de la Salud (OMS), en Marzo de 2020, a declarar como pandemia al virus SARS-CoV-2. Epidemias y pandemias no son nuevos fenómenos en la historia de los seres humanos, y modelos matemáticos han sido utilizados para describir la dinámica de estas enfermedades infecciosas. En este documento presentamos un modelo SEIR generalizado sin características demográficas. En este modelo añadimos las clases asintomáticos, asintomáticos recuperados, aislados y muertos. Llevamos a cabo simulacions en 4 redes de contactos basadas en datos: lugares de trabajo, hogar, comunidad en general y lugares de aglomeración. Los estudios de Cuevas et al., Peng et al. y Quan et al. son relevantes para este trabajo. Decidimos dejar todos los parámetros como constantes a exepción de la tasa de curación y mortalidad, sin embargo en trabajos futuros estos parámetros serán modelados como funciones del tiempo para predicciones más precisas. En particular, nos enfocamos en el comportamiento de las clases asintomáticos, infectados y muertos en cada una de las redes de contacto. Observamos que las curvas cambian en función de cuál de ellas alcanza más rápido el pico de la curva. Finalmente, calculamos el número de reproducción, R_0, utilizando el enfoque de próxima generación para cada una de las redes. Aquí, para cada red obtenemos que $R_0 > 1$ que concuerda con la teoría. Es importante mencionar que la falta de transparencia en los datos emitidos por el gobierno dificulta la construcción de estos modelos.

Palabras Clave—SARS-Cov-2, modelos matemáticos, SEIR, redes de contacto, simulaciones, número básico de reproducción.

Contents

1 Introduction

Epidemics and pandemics are not a new issue in human history. An epidemic is an outbreak of an infectious disease (an illness caused by an external organism) in a specific area. It becomes a pandemic when the spread of the disease reaches a large part of the world, affecting a significant amount of the population, [16]. Several of these diseases have impact humanity in different levels of severity and, also, they have been the focus of study in various branches of science. In particular, in mathematics there exists a area called *Mathematical Epidemiology* designed to abstract the dynamics of these phenomena through usually compartmental models. There is a current worldwide outbreak caused by the novel coronavirus SARS-CoV-2 and several mathematical models have been developed, e.g. [7] and [12], to help explain, study, and make predictions about its behaviour.

The first epidemiological model dates back to 1776, in which Bernoulli used mathematical methods to analyze the behavior of smallpox. Compartmental models were a significant advance in the study of the spread of infectious diseases; Kermack and McKendick, in 1927, developed a deterministic epidemiological model that distinguishes three classes of individuals: susceptible, infected, and recovered. Since then the development of theory in this area of mathematics has been condensed in a series of books such as [11], [12]. Also, several epidemics and pandemics have been modeled through the use of these tools. Basically, the dynamics of Covid-19 epidemic has been modeled as an extension of the standard SEIR model. The work of [7] and [12] shows different approaches of the same scenario. This last does not implies that one model is wrong, but the authors take into account different intricacies of COVID-19. Although [10] does not study the current outbreak, its focus on how the dynamics of a spread disease changes in different contact networks is relevant for making more accurate predictions.

The studies of the Covid-19 pandemic has been developed from the basic SEIR model. The models made by [7] and [12] have not taken into account demographic characteristics, i.e. births and deaths not-directly caused by the disease in question are not taken into account. In the study of [7], the model consists of describing how the lockdown measures intervened in the dynamics of the pandemic. Unlike other models, as [12], in which the quarantined population is taken as a separate compartment, in this work this characteristic is reflected in the time-dependent factor, β, of contagion. On the other hand, [12] model considers the population subjected to isolation as an individual class. This study is focused on a scenario in which quarantine is only mandatory for the infected population. This study is complemented by [10] approach because the latter analyzes and places a specific weight on the different places of agglomeration: schools, workplaces, households and general community.

This document presents a model derived from the works [7], [12] and [10] that aims to mathematically describe the dynamics of the COVID-19 pandemic in a new scenario that we call a *new normality*. In this scenario, as in [7] and [12], demographic constraints are irrelevant because of the short time period in which the study is developed. Thus, only already infectious individuals must be isolated. We differentiate two infectious classes: asymptomatic and infected. Since asymptomatic individuals are really hard to track, we only consider population in the infected compartment to be isolated. Further studies can consider a vaccinate class as an additional control strategy. All asymptomatic individuals will become asymptomatic recovered ones at an specific rate. Also, we consider social distance and the use of masks effects as a mitigation of the transmission rate, β. Every parameter is obtained as the inverse of average time period that an individual stays in a single class.

A model of 8 time-dependent individual classes, through a system of 8 ordinary differential equations, is presented: susceptible, exposed, asymptomatic, asymptomatic recovered, infected, isolated, recovered and dead. Although in this work we take β as a constant, it is important to emphasize that a more in-depth study of this factor can be carried out and it would lead into a more precisely representation of the current situation. Finally, in this study we show the dynamics of this infectious disease in 4 different layers:general community, households, workplaces and agglomeration places. In each of these places, the infected curve has a different behavior in terms of the speed with which they reach their peak. The computation of the reproduction number, R_0, is obtained as the spectral value of the next-generation matrix. Although R_0 barely differ between layers, this value is greater than 1 in every single one of them which is compatible with the theory.

2 A brief introduction to Mathematical Epidemiology

In this section we will present some rudiments and interesting mathematical tools associated with epidemiology. The mathematical part of this chapter is focused in some basic concepts regarding linear algebra, mathematical modelling and ordinary differential equations. It has as principal references: [3], [9], [14] and [17]. Following this part, some introductory concepts about epidemics and, specially, SARS-Cov-2 virus will be presented. Also, some basic epidemiological models shall be introduced. The main references for this are [2], [8], [4], [11] and [13].

2.1 Some basic concepts

A real matrix is an array of $m \times n$ real numbers arranged in m files and n columns.

$$A = \begin{pmatrix} a_{11} & . & . & . & a_{1n} \\ . & . & & & . \\ . & & . & & . \\ . & & & . & . \\ a_{m1} & . & . & . & a_{mn} \end{pmatrix} = (a_{ij}) \in \mathcal{M}_{mn}(\mathbb{R})$$

If $m = n$, then A is a square matrix. An identity matrix, I, is a square matrix where the elements in the main diagonal are 1 and the remaining elements are equal to 0, i.e.

$$I = (\delta_{ij})$$

where, δ_{ij} denotes Kronecker's delta.

Let $A, B \in \mathcal{M}_n(\mathbb{R})$. Assume that $A \times B = I$. Then B is said to be the inverse of A and it is denoted as A^{-1}. The minor ij of A, M_{ij}, is the $(n-1) \times (n-1)$ matrix obtained from subtracting the n file and m column to A; the ij cofactor of A, A_{ij}, is given by

$$A_{ij} = (-1)^{ij}|M_{ij}|.$$

The determinant of A is given by

$$\det(A) = |A| = \sum_{k=1}^{n} a_{1k}A_{1k}.$$

Thus, λ is an eigenvalue of A iff there exists $v \in \mathbb{R}^n/\{0\}$ such that

$$Av = \lambda v.$$

Proposition 2.1.1. *Let $A \in \mathcal{M}_n(\mathbb{R})$. Then λ is an eigenvalue of A iff*

$$p(\lambda) = \det(A - \lambda I) = 0$$

.

Definition 2.1.2. [Spectral Radius] The spectral radius of A is the maximum absolute eigenvalue of A, that is

$$\rho(A) = \sup\{|\lambda| : \lambda \in \sigma(A)\}$$

where $\sigma(A)$ is the set of all eigenvalues of A.

Definition 2.1.3. **[Jacobian Matrix]** Let $f \in C^1(\mathbb{R}^n)$. Then the Jacobian matrix of f, J, is defined as

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial x_1} & \cdot & \cdot & \cdot & \frac{\partial f_1}{\partial x_n} \\ \cdot & \cdot & & & \cdot \\ \cdot & & \cdot & & \cdot \\ \cdot & & & \cdot & \cdot \\ \frac{\partial f_m}{\partial x_1} & \cdot & \cdot & \cdot & \frac{\partial f_m}{\partial x_n} \end{pmatrix}$$

2.2 Mathematical Modelling

A mathematical model is an abstraction, usually presented as equations or another mathematical relations, of a real life phenomenon. It is important to consider that a model is not exactly, constantly modified, frequently discarded and sometimes used anyway because is better than nothing, [3]. Several disciplines as finance, ecology, epidemiology, medicine, among others are heavily affected by mathematical modelling, [14]. There are three important aspects in modelling: things whose effects are not taken into account, input and output. Clearly if the output is not good the model will be rejected; if the wrong aspects are neglected, the outputs will not give adequate results and finally, if the model tries to handle a lot amount of inputs, it will turn extremely hopeless to compute, [3].

To build a model there is a, commonly accepted, process divided in 4 stages:

- formulate the problem,
- outline the model,
- is it useful?,
- testing the model.

In the first, we consider what we want to explain with the model; the second one is about to identified and classified the different parts of the environment in which the model will be working, here is where the model is formulated; in the third, it is time to evaluate results given by the model, [3]. The model could give results but those differ far away from reality. In the most cases, the phenomenon that is described is leaded to experimentation to evaluate the results of the model, but in the case of epidemics this is not possible because it is unethical carry out this type of experiment on living beings.

2.3 Ordinary Differential Equations

In some mathematical models, hypotheses often involve a ratio of change of one or more of the variables; indeed, the mathematical model can be a differential equation or a system of differential equations, [17].

Let's recall that an ordinary differential equation (ODE) is an equation containing only derivatives of one or more dependent variables with respect to a single independent variable. Then a first order ODE is a relation of the form

$$x'(t) = f(t, x(t)), \tag{1}$$

where $t \in \mathbb{R}$, $D \subset \mathbb{R}^{n+1}$, open and $f : D \to \mathbb{R}^{n+1}$ is usually a continuous function.

An ODE can be classified by,

i) Order: The order of an ODE is the order of the highest derivative in the equation.

ii) Linearity: An ODE is said to be linear when the dependent variable and its derivatives are of the first degree and, also, its coefficients depend at most on the independent variable.

A solution of an ODE is any function ω, defined on an interval $I \subset \mathbb{R}$ such that $\omega \in C^n(I)$, which when substituted into an nth-order ODE reduces the equation to an identity on I. Then, we say that x is a solution of (1) on $I \subset \mathbb{R}$ if $x \in C^1(I)$, for every $t \in I$ and x satisfies (1). In some cases, it is only possible to obtain an implicit solution of (1), that is a relation $G(x, y) = 0$, provided that there exists at least one function ω that satisfies both the relation and the ODE on I.

A *system of ODE's* consists of two or more equations involving the derivatives of two or more unknown functions of a single independent variable, [17]. For example,

$$\begin{array}{c} x_1'(t) = f_1(t, x_1(t), x_2(t), ..., x_n(t)), \\ \vdots \\ x_n'(t) = f_n(t, x_1(t), x_2(t), ..., x_n(t)), \end{array} \tag{2}$$

is a system of n first order ODE's. The solution of (2) is given n differentiable functions, $\omega_1, \omega_2, ..., \omega_n \in C^1(I)$, that satisfy each equation on I.

Definition 2.3.1. [Initial Value Problem (IVP)] It is an ODE where some conditions are imposed over the dependent variable and its derivatives.

Let $I = [t_0, t_f]$. An IVP for (1) has the form

$$\left\{ \begin{array}{rcll} x'(t) & = & f(t, x(t)), & t \in I \\ x(t_0) & = & x_0, & \end{array} \right. \tag{3}$$

and consists in searching for a solution x of (1) satisfying $x(t_0) = x_0$ where $(x_0, t_0) \in D$.

Definition 2.3.2. [Autonomous ODE] It is an ODE in which the independent variable does not appear explicitly.

In the case of (1), if $x'(t) = f(x(t))$, i.e. f does not depend of t, then (1) is an autonomous ODE.

Sometimes the analytic, explicit or implicitly, solutions of systems of ODE's are really hard and, even not possible, to find. When this happens, numerical tools are used to obtain a solution. One of the most accurate numerical procedures used to approximate solutions to problems of the form (3) is the fourth-order Runge-Kutta method, [17]. It consists in finding the parameters, to agree with a Taylor polynomial of degree four, of the following formula:

$$y_{n+1} = y_n + h(w_1 k_1 + w_2 k_2 + w_3 k_3 + w_4 k_4),$$

where

$$\begin{array}{rcl} k_1 & = & f(x_n, y_n), \\ k_2 & = & f(x_n + \alpha_1 h, y_n + \beta_1 h k_1), \\ k_3 & = & f(x_n + \alpha_2 h, y_n + \beta_2 h k_1 + \beta_3 h k_2), \\ k_4 & = & f(x_n + \alpha_3 h, y_n + \beta_4 h k_1 + \beta_5 h k_2 + \beta_6 h k_3). \end{array}$$

Definition 2.3.3. [**Equilibrium Points**] A solution $x_1, x_2, ..., x_n$ of (2) is an equilibrium point iff

$$\begin{cases} x_1'(t) &= f_1(t, x_1(t), x_2(t), ..., x_n(t)) = 0, \\ & \cdot \\ & \cdot \\ & \cdot \\ x_n'(t) &= f_n(t, x_1(t), x_2(t), ..., x_n(t)) = 0, \quad t \in I. \end{cases}$$

The stability around an equilibrium point, x_0, is related to the eigenvalues of the Jacobian matrix, $J(x_0)$.

Remark 2.3.4. To study stability we consider that $\sigma(A) \subset \mathbb{C}$, for $A \in \mathcal{M}_n(\mathbb{R})$.

Definition 2.3.5. [**Stability via Jacobian**] Let λ be any eigenvalue of $J(x_0)$. Then if $\mathrm{Re}(\lambda) > 0$, the system is unstable. On the other hand, if for every $\lambda \in \sigma(J(x_0)), \mathrm{Re}(\lambda) < 0$, then the system is stable near x_0.

2.4 Epidemics. SARS-Cov-2.

An epidemic is defined as *"The occurrence in a community or region of cases of an illness, specific health-related behavior, or other health-related events clearly in excess of normal expectancy"*, [13]. There is no need to have a great amount of infected people to consider an illness as an epidemic, it is just enough to have more cases than the normal. For example, in 2018, the County of San Diego, U.S.A. declared an outbreak of meningococcal disease because there were three infected people, i.e. there were more infected than the expected in a determinate amount of time and space.

The usual motivation to study epidemics presents itself when human population is affected. These studies are directed to a population in certain amount of time and space, [2]. The delimitation of the geographical area and the period of time is the first step in the identification of an outbreak. For example, the subject of study could be the spread of a disease in a hospital in 2 weeks. Once identified the target, it is easier to recognize all the variables in its dynamics, specially by its importance.

At the end of 2019 a novel virus Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) was the cause of a series of pneumonia cases in Wuhan, China, [8]. The rapidity of its export to another countries, through a massive contagion, caused that in March 2020 the World Health Organization (WHO) declared to be a pandemic.

Coronaviruses are enveloped RNA viruses. Under the electron microscope, this kind of viruses have a crown like appearance because of the presence of glycoprotein spikes on their envelope, [8], [15]. The clinical spectrum of COVID-19 varies from asymptomatic or paucissymptomatic forms to clinical conditions characterized by severe respiratory failure that necessitates mechanical ventilation and support in an intensive care unit (ICU), to multiorgan and systemic manifestations in terms of sepsis, septic shock, and multiple organ dysfunction syndromes (MODS), [8].

Figure 1: Coronaviruses have a crown-like appearance because of glycoprotein spikes on their envelope. Source: [1].

2.5 Standar Epidemiology Mathematical Models

There exit several basic epidemiological models such as SI, SIS, SIR and SEIR. There will be explained the rudiments of SIR and SEIR models as these are the most relevant in our study. For a more detailed explanation see e.g. [11] and [5].

Remark 2.5.1. Since all epidemiological models are time-dependent, we will take $t \geq 0$ trough all the following sections.

2.5.1 SIR

The SIR epidemic model was proposed by Kermack and McKendrick in 1927 and it was one of the first epidemic models, [11]. This model divides the total population, N, in three no-intersected classes:

i) S denotes the group of people that are *Susceptible*, i.e., the population that are free from the disease but can get infected.
ii) I denotes the population which already got the disease and can spread it to the healthy individuals. This group is called *Infected*.
iii) R denotes the population that was infected and *recovered*. Individuals in this group cannot contract the disease again.

N is the sum of the amount of people in each one of the three classes. Each one of these classes are functions of time: $S(t)$, $I(t)$, $R(t)$:

$$N = S(t) + I(t) + R(t).$$

The SIR model consists of a system of ODE's that describes the dynamics of susceptible, infected and recovered individuals. To derive the system, it is considered how the classes change in time. Susceptible and infected population decreases and increases, respectively, in a unit of time by all the individuals who change from S to I. In epidemiology, the number of individuals who go from susceptible to infected per unit of time is called *incidence*, D, and is given by

$$D = pcN\frac{S(t)}{N}I(t), \tag{4}$$

where,

- cN is the number of contacts that one infectious individual make.
- $S(t)/N$ is the probability that this contact is made with a susceptible individual.
- p is the probability that this transmission actually happens becuase not every contact between an infected and susceptible individual derives in the transmission of the disease.

Then, pcS is the number of susceptible individuals who become infected per unit of time per infectious individual. So, (4) can be written as

$$D = \beta S(t)I(t),$$

with $\beta = pc$, which is known as *transmission rate constant*. Finally, the rate of change of the susceptible class is given by

$$S'(t) = -\beta I(t)S(t). \tag{5}$$

Individuals from the infected class leave, by recovering or dying, at constant α called *recovery rate*. So,

$$I'(t) = \beta I(t)S(t) - \alpha I. \tag{6}$$

Individuals belonging to the recovered class are those who moved from the infected class

$$R'(t) = \alpha I(t). \tag{7}$$

Then, the model is given by the equations (5), (6) and (7):

$$\begin{array}{rcl} S'(t) & = & -\beta I(t)S(t), \\ I'(t) & = & \beta I(t)S(t) - \alpha I(t), \\ R'(t) & = & \alpha I(t), \end{array} \tag{8}$$

with initial conditions

$$S(0) = S_0, I(0) = I_0 \text{ and } R(0) = R_0.$$

Figure 2: Flowchart of SIR model

By adding the equations in (8), we have that

$$\begin{array}{rcl} N'(t) & = & S'(t) + I'(t) + R'(t) \\ & = & -\beta I(t)S(t) + \beta I(t)S(t) - \alpha I(t) + \alpha I(t) \\ & = & 0, \end{array}$$

which allows to conclude that,

$$N(t) = N,$$

i.e., the size of the population is constant in time. SIR model works with the following assumptions, [4]:

i) the size of the population remains constant: there are no births or and deaths in the population;
ii) no one enters or leave the population, it is closed;
iii) recovered individuals obtain immunity, they cannot get infected by the disease again.

These assumptions are enough to solve (8). Since R does not appear in the first two equations of the system (8), it is enough to work with expressions for *susceptible* and *infected* individuals. So,

$$\frac{I'(t)}{S'(t)} = -\frac{\beta I(t)S(t) - \alpha I(t)}{\beta I(t)S(t)}$$
$$I(t)' = \left(-\frac{\beta S(t) - \alpha}{\beta S(t)}\right) S(t)'.$$

By solving the ODE, it follows that

$$I(t) = -S(t) + \alpha \ln\left(S(t)\right) + C.$$

By the initial conditions and,

$$S(t) \to S_\infty \text{ and } I(t) \to 0, \text{ as } t \to +\infty,$$

It follows that

$$I_0 = -S_0 + \frac{\alpha}{\beta}\ln\left(S_0\right) + S_\infty - \frac{\alpha}{\beta}\ln\left(S_\infty\right).$$

Rearrangering the terms,

$$I_0 = \frac{\alpha}{\beta}\ln\left(\frac{S_0}{S_\infty}\right) - S_0 + S_\infty$$
$$\frac{\beta}{\alpha} = \frac{\ln\left(\frac{S_0}{S_\infty}\right)}{I_0 + S_0 - S_\infty}, \tag{9}$$

which clearly is a positive number. When $I'(t) = 0$, the implicit solution allows us to calculate the maximum number of infected people that is reached, that is

$$\hat{S} = \frac{\alpha}{\beta}.$$

By substituying $\hat{S}$ in

$$I(t) = -S(t) - \frac{\alpha}{\beta}\ln\left(S(t)\right) + S_0 + I_0 - \frac{\alpha}{\beta}\ln\left(S_0\right),$$

it follows that

$$I_{\max} = -\frac{\alpha}{\beta} - \frac{\alpha}{\beta}\ln\left(\frac{\alpha}{\beta}\right) + S_0 + I_0 - \frac{\alpha}{\beta}\ln\left(S_0\right).$$

$I_{\max}$ is the maximum number of infected individuals reached in the epidemic, [11].

2.5.2 SIR Model with Demography

In this model, all individuals are born susceptible. Individuals from every stage die at a per capita death rate μ, [5],[11],[4]. Then, the model takes the form

$$\begin{aligned} S'(t) &= B - \beta S(t)I(t) - \mu S(t), \\ I'(t) &= \beta S(t)I(t) - \alpha I(t) - \mu I(t), \\ R'(t) &= \alpha I(t) - \mu R(t). \end{aligned} \tag{10}$$

Again, by adding the three equations in (10), it is obtained that

$$N'(t) = B - \mu N, \qquad N = S(t) + I(t) + R(t).$$

Since

$$N(t) \to \frac{B}{\mu}, \text{ as } t \to \infty,$$

the population size is asymptotically stable, although it is not constant, [5]. The incidence here, where the population is not constant, is called the *law of mass action*,

$$D = \beta S(t)I(t).$$

Analogously as with the SIR model, the first two equations are independent from the third equation; so, we consider the following system

$$\begin{aligned} S'(t) &= B - \beta S(t)I(t) - \mu S(t), \\ I'(t) &= \beta S(t)I(t) - \alpha I(t) - \mu I(t), \end{aligned} \tag{11}$$

and R is obtained as $R = N - S - I$. SIR model can be written as

$$\begin{aligned} S'(t) &= f(S(t), I(t)), \\ I'(t) &= g(S(t), I(t)). \end{aligned} \tag{12}$$

The system (12) is an autonomous ODE since they do not depend explicitly on the time variable, [11].

Let's make a change of variable in order to transform the system in a nondmiensional form. So, let

$$\tau = \frac{\alpha + \mu}{t},$$

then

$$N(t) = N\left(\frac{\tau}{\alpha + \mu}\right) = \hat{N}(\tau).$$

Analogously, $I(t) = \hat{I}(\tau)$ and $S(t) = \hat{S}(\tau)$. By the chain rule

$$\begin{aligned} \frac{d\hat{S}}{d\tau} &= \frac{1}{\alpha + \mu}\frac{dS}{dt}, \\ \frac{d\hat{I}}{d\tau} &= \frac{1}{\alpha + \mu}\frac{dI}{dt}, \end{aligned} \tag{13}$$

also,

$$x(t) = \frac{\mu}{B}\hat{S} \text{ and } y(t) = \frac{\mu}{B}\hat{I},$$

because we have re-scaled $\hat{S}$ and $\hat{I}$ with the total population size, [5]. Now, let's perform some computations

$$\begin{aligned}
x'(\tau) &= \frac{\mu}{B}\frac{1}{\alpha+\mu}\frac{d\hat{S}}{dt}, \\
&= \frac{\mu}{B}\frac{1}{\alpha+\mu}\left(B - \beta\hat{I}\hat{S} - \mu\hat{S}\right), \\
&= \frac{\mu}{\alpha+\mu} - \frac{\mu\beta\hat{I}\hat{S}}{B(\alpha+\mu)} - \frac{\mu^2\hat{S}}{B(\alpha+\mu)}, \\
&= \frac{\mu}{\alpha+\mu}\left(1 - \frac{\mu\hat{S}}{B}\right) - \frac{\mu\beta\hat{I}\hat{S}}{B(\alpha+\mu)}, \\
&= \rho(1-x) - \frac{B\beta}{\mu(\alpha+\mu)}\frac{\mu^2\hat{S}\hat{I}}{B^2}, \\
&= \rho(1-x) - \mathcal{R}_0\frac{\mu\hat{S}}{B}\frac{\mu\hat{I}}{B}, \\
&= \rho(1-x) - \mathcal{R}_0 xy.
\end{aligned}$$

By a similar process, we obtain

$$y'(\tau) = (\mathcal{R}_0 - 1)y.$$

So, a new dimensionless system of (13) is given by

$$\begin{aligned}
x'(\tau) &= \rho(1-x) - \mathcal{R}_0 xy, \\
y'(\tau) &= (\mathcal{R}_0 - 1)y.
\end{aligned}$$

2.5.3 SEIR

The SEIR model is another classical epidemiological model which incorporates the state of exposed individuals, $E(t)$, to the SIR model, [4]. The individuals in this stage are infected but not infectious, i.e. they have the virus in their system, although they cannot transmit it to another person. With this addition, the model takes the form

$$\begin{aligned}
S'(t) &= B - \beta S(t)I(t) - \mu S(t), \\
E'(t) &= \beta S(t)I(t) - (\mu+\gamma)E(t), \\
I'(t) &= \gamma E(t) - (\mu+\alpha)I(t), \\
R'(t) &= \alpha I(t) - \mu R(t).
\end{aligned} \tag{14}$$

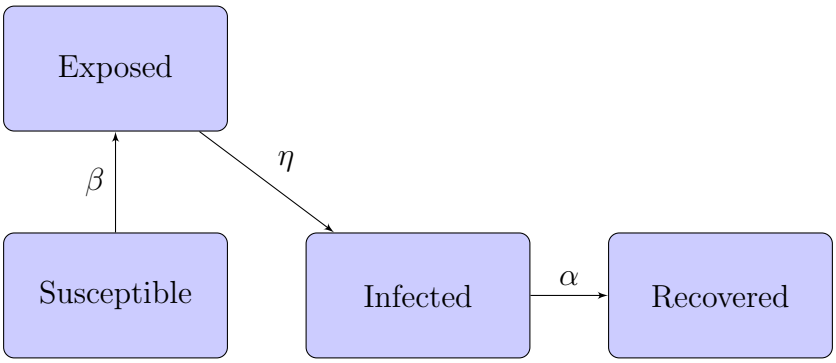

Figure 3: Flowchart of SEIR model

In order to obtain the equilibrium of the system, all the four equations are set to 0 and solved

$$\begin{aligned} 0 &= B - \beta S(t)I(t) - \mu S(t), \\ 0 &= \beta S(t)I(t) - (\mu+\gamma)E(t), \\ 0 &= \gamma E(t) - (\mu+\alpha)I(t), \\ 0 &= \alpha I(t) - \mu R(t). \end{aligned} \tag{15}$$

Let's solve (15); it is quite clear that,

$$\mathcal{E} = \left(\frac{B}{\mu}, 0, 0, 0\right)$$

is a solution of the system; $\mathcal{E}$ is the disease-free equilibrium of the system. In order to find the endemic-free equilibrium, $\mathcal{E}^*$, of (15), we write

$$S(t) = \frac{B}{\beta I(t) + \mu}. \tag{16}$$

By replacing (16) into the second equation of (15), it is obtained

$$E(t) = \frac{\beta I(t)}{\mu+\gamma}\frac{B}{\beta I(t)+\mu}. \tag{17}$$

To compute I, (17) is replaced in the third equation of the system

$$\begin{aligned} I(t) &= \gamma\frac{\beta I(t)}{\mu+\gamma}\cdot\frac{B}{(\beta I(t)+\mu)(\mu+\alpha)} \\ &= \frac{\gamma B}{(\mu+\gamma)(\mu+\alpha)} - \frac{\mu}{\beta} \\ &= \frac{\mu}{\beta}\left(\frac{\beta\gamma B}{(\mu+\gamma)(\mu+\alpha)\mu} - 1\right), \end{aligned}$$

hence,

$$I^*(t) = \frac{\mu}{\beta}(\mathcal{R}_0 - 1), \tag{18}$$

where $\mathcal{R}_0$ is the reproduction number, [11]. Then, by substituting (18) in (16)

$$\begin{aligned} S(t) &= \frac{B}{\mu(\mathcal{R}_0 - 1) + \mu} \\ &= \frac{B}{\mu\mathcal{R}_0} \\ &= \frac{B}{\mu} \cdot \frac{(\mu+\gamma)(\mu+\alpha)\mu}{\beta\gamma B}. \end{aligned}$$

So,

$$S^*(t) = \frac{(\mu+\gamma)(\mu+\alpha)}{\beta\gamma}. \tag{19}$$

To obtain R^*, (18) is replaced in (17)

$$\begin{aligned} E(t) &= \frac{B\mu(\mathcal{R}_0 - 1)}{(\mu+\gamma)(\mu(\mathcal{R}_0 - 1) + \mu)} \\ &= \frac{B\mu(\mathcal{R}_0 - 1)}{(\mu+\gamma)\mu\mathcal{R}_0} \\ &= \frac{B}{\mu+\gamma}\left(1 - \frac{1}{\mathcal{R}_0}\right) \\ E^*(t) &= \frac{\mu+\alpha}{\gamma}\frac{\mu}{\beta}(\mathcal{R}_0 - 1). \end{aligned} \tag{20}$$

Finally,

$$R^*(t) = \frac{\alpha}{\beta}(\mathcal{R}_0 - 1). \tag{21}$$

So, equations (19), (20), (18) and (21) are the components of the endemic-free equilibrium

$$\mathcal{E}^* = (S^*, E^*, I^*, R^*).$$

To analyze the local stability of the system (14), its Jacobian,

$$J = \begin{pmatrix} -\beta I(t) - \mu & 0 & -\beta S(t) & 0 \\ \beta I(t) & -(\mu+\gamma) & \beta S(t) & 0 \\ 0 & \gamma & -(\mu+\alpha) & 0 \\ 0 & 0 & \alpha & -\mu \end{pmatrix},$$

is considered, and to examine the stability of the disease-free equilibrium, the Jacobian is evaluated at $\mathcal{E}$:

$$J(\mathcal{E}) = \begin{pmatrix} -\mu & 0 & -\beta\frac{B}{\mu} & 0 \\ 0 & -(\mu+\gamma) & \beta\frac{B}{\mu} & 0 \\ 0 & \gamma & -(\mu+\alpha) & 0 \\ 0 & 0 & \alpha & -\mu \end{pmatrix}.$$

Let's obtain the roots of the characteristic equation $\det(J(\mathcal{E}) - \lambda Id) = 0$. So,

$$\det(J(\mathcal{E}) - \lambda Id) = (-\mu - \lambda)^2 \begin{vmatrix} -(\mu+\gamma) - \lambda & \beta\frac{B}{\mu} \\ \gamma & -(\mu+\alpha) - \lambda \end{vmatrix}$$

then, the characteristic equation is

$$(-\mu-\lambda)^2\left((\mu+\gamma+\lambda)(\mu+\alpha+\lambda)-\gamma\beta\frac{B}{\mu}\right)=0. \tag{22}$$

So, it is easy to check that (22) has two roots equal to μ and the other roots are the solution of the equation

$$(\mu+\gamma+\lambda)(\mu+\alpha+\lambda)-\gamma\beta\frac{B}{\mu}=0.$$

This equation has one real root if $\mathcal{R}_0>1$ and two conjugate complex roots if $\mathcal{R}_0<1$.

Proposition 2.5.2. *If $\mathcal{R}_0>1$, then the desease-free equilibrium, $\mathcal{E}$, is unstable. If $\mathcal{R}_0<1$, then $\mathcal{E}$ is stable.*

Analogously, to analyze the stability of the endemic-free equilibrium, $\mathcal{E}^*$, the Jacobian is evaluated at $\mathcal{E}^*$ and the roots of the characteristic equation are obtained:

$$J(\mathcal{E}^*)=\begin{pmatrix}-\beta I^*(t)-\mu & 0 & -\beta S^*(t) & 0\\ \beta I^*(t) & -(\mu+\gamma) & \beta S^*(t) & 0\\ 0 & \gamma & -(\mu+\alpha) & 0\\ 0 & 0 & \alpha & -\mu\end{pmatrix}$$

Then,

$$\det(J(\mathcal{E}^*)-\lambda Id)=(-\mu-\lambda)\begin{vmatrix}-\beta I^*(t)-\mu-\lambda & 0 & -\beta S^*(t)\\ \beta I^*(t) & -(\mu+\gamma+\lambda) & \beta S^*(t)\\ 0 & \gamma & -(\mu+\alpha+\lambda)\end{vmatrix}$$

It's quite clear that $-\mu$ is one root and the other roots are obtained from

$$\begin{aligned}-(\beta I^*(t)+\mu+\lambda)(\mu+\gamma+\lambda)(\mu+\alpha+\lambda)-\beta^2 I^*(t)\gamma S^*(t)+\beta S^*(t)\gamma(\beta I^*(t)+\mu+\lambda)=0\\ -(\beta I^*(t)+\mu+\lambda)(\mu+\gamma+\lambda)(\mu+\alpha+\lambda)+\beta\gamma S^*(t)(-\beta I^*(t)+\beta I^*(t)+\mu+\lambda)=0,\end{aligned}$$

thus,

$$(\beta I^*(t)+\mu+\lambda)(\mu+\gamma+\lambda)(\mu+\alpha+\lambda)=\beta\gamma S^*(t)(\mu+\lambda).$$

Since we are interested in solutions with non-negative real part, let's assume that there is λ such that $\mathrm{Re}(\lambda>0)$. Then the following computations are possible

$$\frac{(\beta I^*(t)+\mu+\lambda)(\mu+\gamma+\lambda)(\mu+\alpha+\lambda)}{\mu+\lambda}=\beta\gamma S^*(t)$$

$$\frac{|\beta I^*(t)+\mu+\lambda||\mu+\gamma+\lambda||\mu+\alpha+\lambda|}{|\mu+\lambda|}=\beta\gamma S^*(t). \tag{23}$$

By (19), we have that

$$\frac{|\beta I^*(t)+\mu+\lambda||\mu+\gamma+\lambda||\mu+\alpha+\lambda|}{|\mu+\lambda|}=\beta\gamma\frac{(\mu+\gamma)(\mu+\alpha)}{\beta\gamma}.$$

So,

$$\frac{|\beta I^*(t)+\mu+\lambda|}{|\mu+\lambda|}>1, \qquad \forall\lambda\in\mathbb{C}:\mathrm{Re}(\lambda)>0.$$

Let $\lambda = x+iy$, then

$$\begin{aligned}\frac{|\beta I^*(t)+\mu+\lambda||\mu+\gamma+\lambda||\mu+\alpha+\lambda|}{|\mu+\lambda|} &> |\mu+\gamma+\lambda||\mu+\alpha+\lambda| \\ &\geq |\mu+\gamma+x||\mu+\alpha+x| \\ &\geq (\mu+\gamma)(\mu+\alpha) \\ &= \beta\gamma S^*(t),\end{aligned}$$

which contradicts (23). Thus, the characteristic equation cannot have such kind of solutions, [11]. This leads to the following result.

Proposition 2.5.3. *Assume $\mathcal{R}_0>1$. Then the endemic equilibrium $\mathcal{E}^*$ is locally asymptotically stable.*

2.5.4 SEIAR

In some infectious diseases, there exist individuals who do not experience or show any symptoms but they are infected and contribute to the spread of the disease. The tracking of this individuals is so much harder. Asymptomatic compartment is included as an alternative to the Infected class, [11]. SEIAR model is given by the following system of ODE's:

$$\begin{aligned}S'(t) &= B-\beta S(t)(I(t)+A(t))-\mu S(t),\\ E'(t) &= \beta S(t)(I(t)+A(t))-(\eta+\mu)E(t),\\ I'(t) &= p\eta E(t)-(\alpha+\mu)I(t),\\ A'(t) &= (1-p)\eta E(t)-(\gamma+\mu)A(t),\\ R'(t) &= \alpha I(t)+\gamma A(t)-\mu R(t).\end{aligned} \tag{24}$$

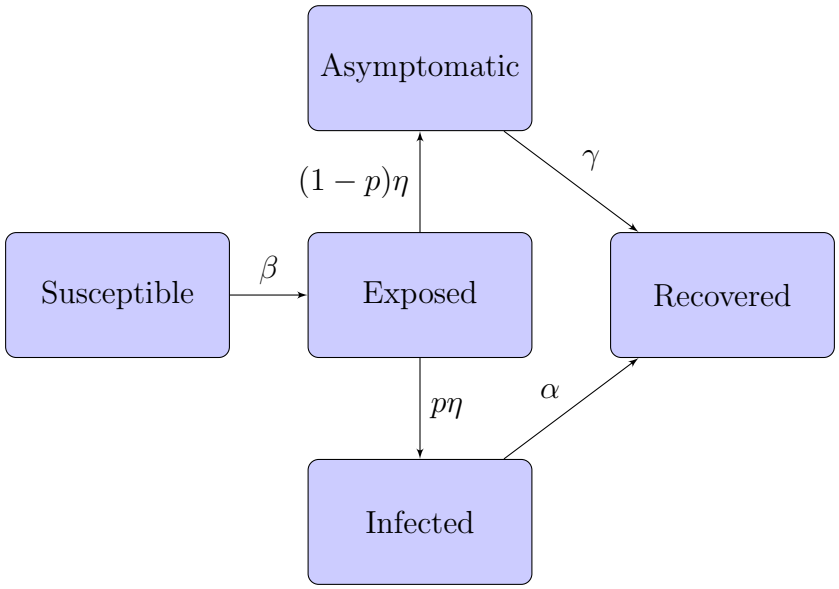

Figure 4: Flowchart of SEIAR model

Here, Exposed individuals move to the Infected class with a probability p, meanwhile they progress to the Asymptomatic compartment with probability $(1-p)$, [5].

2.5.5 Control strategies. Modeling quarantine and isolation

There exist several strategies to control the spread of infectious diseases which include vaccination, treatment, quarantine, isolation and prophylaxis, [11]. Here, we will focus on isolation and quarantine.

In quarantine and isolation measures, exposed or infectious individuals are confined to prevent a more rapid spread of the disease. Isolation is a measure that consists of separating already infected individuals from the population, as opposed to quarantine in which its target is apparently healthy but potentially infected people, [4]. Although both are used to control some many dangerous diseases, isolation is more commonly used. But, in extreme cases, as SARS-Cov-2 epidemic, quarantine is the first response method because it will reduce at the minimum the interaction between susceptible individuals with infectious ones, reducing the velocity of the spread of the disease.

In mathematical models that involve control strategies, the reproduction number, $\mathcal{R}_0$, depends on the method of control. It is called a controlled reproduction number, [11]. Isolation and quarantine models are built by adding separate compartments, Q_1 and Q_2, to a standard model. Consequently, an extension of (14) is given by,

$$\begin{aligned} S'(t) &= B - \beta\tfrac{S(t)(I(t)+qE(t))}{N-Q} - \rho S(t) - \mu S(t) + \eta_1 Q_1, \\ E'(t) &= \beta\tfrac{S(t)(I(t)+qE(t))}{N-Q} - \rho E(t) - (\mu+\gamma)E(t), \\ Q_1'(t) &= \rho S(t) + \rho E(t) - (\mu+\eta_1+\eta_2)Q_1, \\ I'(t) &= \gamma E(t) - (\mu+\alpha+r_2)I(t), \\ Q_2'(t) &= \alpha I(t) + \eta_2 Q_1 - (\mu+r_1)Q_2(t), \\ R'(t) &= r_2 I(t) + r_1 Q_2(t) - \mu R(t). \end{aligned} \tag{25}$$

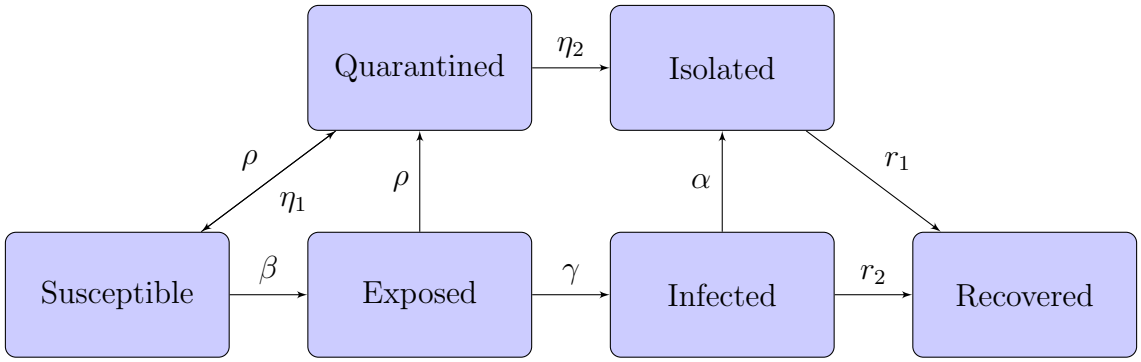

Figure 5: Flowchart of SEIR model with Isolated and Quarantine compartments.

Individuals in these classes do not participate in the total active population dynamics. That is the reason why the total active population in the denominator of the standard incidence is $N-Q$, where $Q = Q_1 + Q_2$.

By following the same procedure as in the SEIR model, it is possible to compute the

controlled reproduction number, $\mathcal{R}_c$:

$$\mathcal{R}_c = \frac{\beta\gamma}{(\gamma+\rho+\mu)(r_2+\sigma+\mu)} + \frac{q\beta}{\gamma+\rho+\mu}. \tag{26}$$

Notice that the first term of (26) is the number of secondary infections generated by the infectious individuals and the remaining term is the number of secondary infections generated by exposed individuals, [4].

2.6 Computation of R_0. The Next Generation Approach

The next-generation approach relies on verifying the R_0 factor that corresponds to how an infected individual gives birth to a new generation of infected ones. This process is characterized by the growth of all infected offspring; if this growing is too fast, it is considered an epidemic. The growth factor from generation to generation is mathematically characterized by the factor R_0. In models with separate classes, a next-generation matrix can be defined. This matrix relates the number of newly infected individuals in the various categories in consecutive generations, [11]. Then R_0 is obtained as the spectral radius of the next-generation matrix.

For the derivation of the next-generation matrix, here will be use the *Van den Driessche and Watmough Approach*. It consists in the division of the model into two non-intersected categories: infected and non-infected individuals. Let's assume that there is $m+n$ compartments; then there is m infected and n non-infected classes. So, the entire ODE system has $m+n$ dependent variables. Let $x \in \mathbb{R}^m$ be the vector of dependent variables in the infected compartments, and $y \in \mathbb{R}^n$ be the vector of dependent variables in the non-infected compartments, [11]. The procedure consider the following steps:

i) We rewrite the ODE system as

$$\begin{aligned} x_i' &= f_i(x,y), && i = 1,...,m, \\ y_j' &= g_j(x,y), && j = 1,...,n. \end{aligned}$$

ii) The first equation is splitted as follows

$$\begin{aligned} x_i' &= \mathcal{F}_i(x,y) - \mathcal{V}_i(x,y), && i = 1,...,m, \\ y_j' &= g_j(x,y), && j = 1,...,n. \end{aligned} \tag{27}$$

where,

- $\mathcal{F}_i(x,y)$ is the rate of emergence of new infectious in i;
- $\mathcal{V}_j(x,y)$ includes the remaining transitional compartments.

From this procedure, it is possible to obtain different decomposition. So, we require that the decomposition satisfy the following conditions:

- $\mathcal{F}_i(0,y) = 0$ and $\mathcal{V}_i(0,y) = 0$ for $y \geq 0$, $i = 1,...,m$;
- $\mathcal{F}_i(x,y)$, for every $x, y \geq 0$;
- If $x_i = 0$, then $\mathcal{V}_i(x,y) \leq 0$ for every $i = 1,...,m$;
- $\sum_{i=1}^{m} \mathcal{V}_i(x,y) \geq 0$, for evey $x, y \geq 0$.

iii) Assume that the disease-free system

$$y' = g(0, y)$$

has a unique disease-free equilibrium $\mathcal{E}_0 = (0, y_0)$.

iv) The matrices F and V, coming from the linearization of the system (27) around the $\mathcal{E}_0$, are determined as

$$F = \left[\frac{\partial \mathcal{F}_i(0, y_0)}{\partial x_j}\right] \qquad \text{and} \qquad V = \left[\frac{\partial \mathcal{V}_i(0, y_0)}{\partial x_j}\right] \tag{28}$$

v) Finally, the next generation matrix is defined as

$$K = FV^{-1}$$

and $\mathcal{R}_0$ is written as the spectral radius of K, i.e.

$$\mathcal{R}_0 = \rho(FV^{-1}).$$

3 Results

In this section it will be presented a model that aims to reproduce the dynamics of the Covid-19 pandemic in a scenario where the quarantine is mandatory only for infected individuals. Here, the virus behaviour will be simulated in 4 different layers: general community, households, workplaces and agglomeration places. Also, the reproduction number, R_0, will be computed for each one of these networks.

3.1 Adaptation of the model

Here we adapt the studies [7] and [12] on the SARS-CoV-2 virus dynamics, by introducing 8 individual classes $\{S(t), E(t), A(t), Ar(t), I(t), Q(t), R(t), D(t)\}$ denoting the number of Susceptible, Exposed, Asymptomatic, Asymptomatic Recovered, Infected, Isolated, Quarantined, Recovered and Dead individuals at time t.

To begin with, as our period time is short, we discard demographic constraints, i.e., we consider that the size of the population, $N(t)$, is constant in time:

$$N(t) = S(t) + E(t) + A(t) + Ar(t) + I(t) + Q(t) + R(t) + D(t).$$

The model begins with a fully susceptible population that can change to the exposed class by the interaction with two compartments of already infectious individuals: asymptomatic and infected. A latent period, around 5 days, denoted by σ^{-1} follows after an individual becomes exposed, [7]. After this period, the individual can become either asymptomatic or infected. The asymptomatic path is much simpler, since they will not show any symptoms. The asymptomatic infectious period is typically of 7 days, and it is represented as M_{Ar}^{-1}. Population recovered from A is denoted as A_r. As a control strategy, infected individuals become isolated ones at rate δ. This isolation usually consists of 14 days, [15]. The recovery rate will be denoted as λ. Finally, an isolated individual dies at rate κ. Transmission rate, β, is the interaction factor that leads to new infectious. There is an specific β for each interaction, β_{SA} and β_{SI} for the interaction between S and I, as well as I and S, respectively. Cure, $\lambda(t)$, and mortality, $\kappa(t)$ rates are taken as was proposed in [12]; the remaining parameters are considered as constants. Then the model is given by

$$\begin{aligned}
S'(t) &= -\beta_{SA}\frac{S(t)A(t)}{N-Q} - \beta_{SI}\frac{S(t)I(t)}{N-Q} \\
E'(t) &= \beta_{SA}\frac{S(t)A(t)}{N-Q} + \beta_{SI}\frac{S(t)I(t)}{N-Q} - (\gamma+\sigma)E(t) \\
A(t)' &= \sigma E(t) - M_{Ar}A(t) \\
Ar'(t) &= M_{Ar}A(t) \\
I'(t) &= \gamma E(t) - \delta I(t) \\
Q'(t) &= \delta I(t) - (\lambda+\kappa)Q(t) \\
R'(t) &= \kappa(t)Q(t) \\
D'(t) &= \lambda(t)Q(t)
\end{aligned}$$

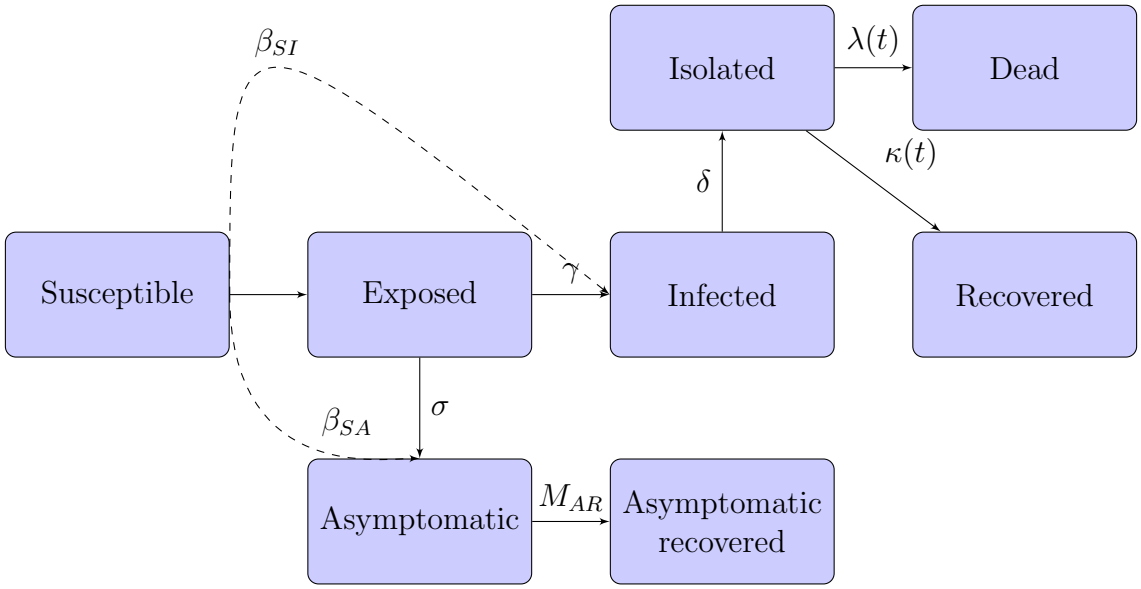

Figure 6: Flowchart of SEAArIQRD model.

3.2 Simulations

To perform the simulations, we fix the parameters as follows. For the transmission rate we consider

$$p_{SI} = 0.95 \text{ and } p_{SA} = 0.9.$$

The latent period is found to be about 2-3 days, so

$$\sigma = 0.29.$$

The total incubation period is of 4-7 days,

$$\gamma = 0.19.$$

The time scale of recovery for asymptomatic is 7 days,

$$M_{Ar} = 0.14.$$

Time scale of going from infected class to isolated compartment

$$\delta = 0.101.$$

Isolated individuals are expected to recover at rate $\lambda(t)$ and die at rate $\kappa(t)$. These are time-dependent factors, [1], [8], [15], [12], [7]. The system is rewritten as

$$Y'(t) = AY(t) + F + G,$$

where

$$Y'(t) = \begin{pmatrix} S'(t) \\ E'(t) \\ Ar'(t) \\ I'(t) \\ Q'(t) \\ R'(t) \\ D'(t) \end{pmatrix}, \qquad A = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\gamma-\sigma & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma & -M_{Ar} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & M_{Ar} & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma & 0 & 0 & -\delta & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \delta & -\kappa(t)-\lambda(t) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \kappa(t) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \lambda(t) & 0 \end{pmatrix},$$

$$Y(t) = \begin{pmatrix} S(t) \\ E(t) \\ A(t) \\ Ar(t) \\ I(t) \\ Q(t) \\ R(t) \\ D(t) \end{pmatrix}, \qquad F = S(t)A(t) \begin{pmatrix} -\beta_{SA} \\ \beta_{SA} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \qquad G = S(t)I(t) \begin{pmatrix} -\beta_{SI} \\ \beta_{SI} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

and solved by the fourth-order Runge-Kutta method, [6]. For the simulations that we are going to present, we used MATLAB R2020a under a PC Toshiba Intel (R) Core(TM) i5-5200U CPU @ 2.20GHz 2.20GHz, 64 bits.

Matlab Code

The following code is adapted from the code used in [6] by changing the initial values and the choise of matrices A and G so to be able to implement the fourth-order Runge-Kutta method since the dimension of the model is bigger than the model proposed in [6]. We consider a population

$$N = 17 * 10^6$$

and initial conditions

$$\begin{aligned} I(0) &= 325000, \\ Q(0) &= I(0), \\ A(0) &= 0.7 * I(0), \\ Ar(0) &= 0.4 * I(0), \\ E(0) &= 1000, \\ R(0) &= 270000, \\ D(0) &= 16746. \end{aligned}$$

```
function [S,E,A,Ar,I,Q,R,D] = SEAArIQRD(wg,ba,bi,gamma,delta,
    lambda0,sigma1,Mar,kappa0,Npop,E0,A0,Ar0,I0,Q0,R0,D0,t)
```

```
% [S,E,A,Ar,I,Q,R,D] = SEAArIQRD(wg,alpha,ba,bi,gamma,delta,
   lambda0,sigma1,Mar,kappa0,Npop,E0,A0,Ar0,I0,Q0,R0,D0,t)
   simulate the time-histories of an epidemic outbreak using a
   generalized SEIR model.

% Initial conditions
N = numel(t);
Y = zeros(8,N);
Y(1,1) = Npop-A0-Q0-E0-R0-D0-I0;
Y(2,1) = E0;
Y(3,1) = A0;
Y(4,1) = Ar0;
Y(5,1) = I0;
Y(6,1) = Q0;
Y(7,1) = R0;
Y(8,1) = D0;

if round(sum(Y(:,1))-Npop)~=0
    error('the sum must be zero because the total population (
       including the deads) is assumed constant');
end

modelFun = @(Y,A,F,G) A*Y + F + G;
dt = median(diff(t));

% ODE resolution

kappa = kappa0(1)*(1-exp(-kappa0(2).*t));
lambda = lambda0(1)*exp(-lambda0(2).*2*t);

for ii=1:N-1
    A = getA(gamma,delta,lambda(ii),kappa(ii),sigma1,Mar);
    SI = Y(1,ii)*Y(5,ii);
    SA = Y(1,ii)*Y(3,ii);
    F = zeros(8,1);
    G = zeros(8,1);
    F(1:2,1) = [-wg*bi/(Npop-Q0);wg*bi/(Npop-Q0)].*SI;
    G(1:2,1) = [-wg*ba/(Npop-Q0);wg*ba/(Npop-Q0)].*SA;
    Y(:,ii+1) = RK4(modelFun,Y(:,ii),A,F,G,dt);
end

S = Y(1,1:N);
E = Y(2,1:N);
A = Y(3,1:N);
Ar =Y(4,1:N);
```

```
I = Y(5,1:N);
Q = Y(6,1:N);
R = Y(7,1:N);
D = Y(8,1:N);

    function [A] = getA(gamma,delta,lambda,kappa,sigma,Mar)
        A = zeros(8);
        % E
        A(2,2) = -gamma-sigma;
        % A
        A(3,2:3) = [sigma,-Mar];
        % Ar
        A(4,3) = Mar;
        % I
        A(5,2:5) = [gamma,0,0,-delta];
        % Q
        A(6,5:6) = [delta,-kappa-lambda];
        % R
        A(7,6) = kappa;
        % D
        A(8,6) = lambda;
    end
    function [Y] = RK4(Fun,Y,A,F,G,dt)
        % Runge-Kutta of order 4
        k_1 = Fun(Y,A,F,G);
        k_2 = Fun(Y+0.5*dt*k_1,A,F,G);
        k_3 = Fun(Y+0.5*dt*k_2,A,F,G);
        k_4 = Fun(Y+k_3*dt,A,F,G);
        % Output
        Y = Y + (1/6)*(k_1+2*k_2+2*k_3+k_4)*dt;
    end

end
```

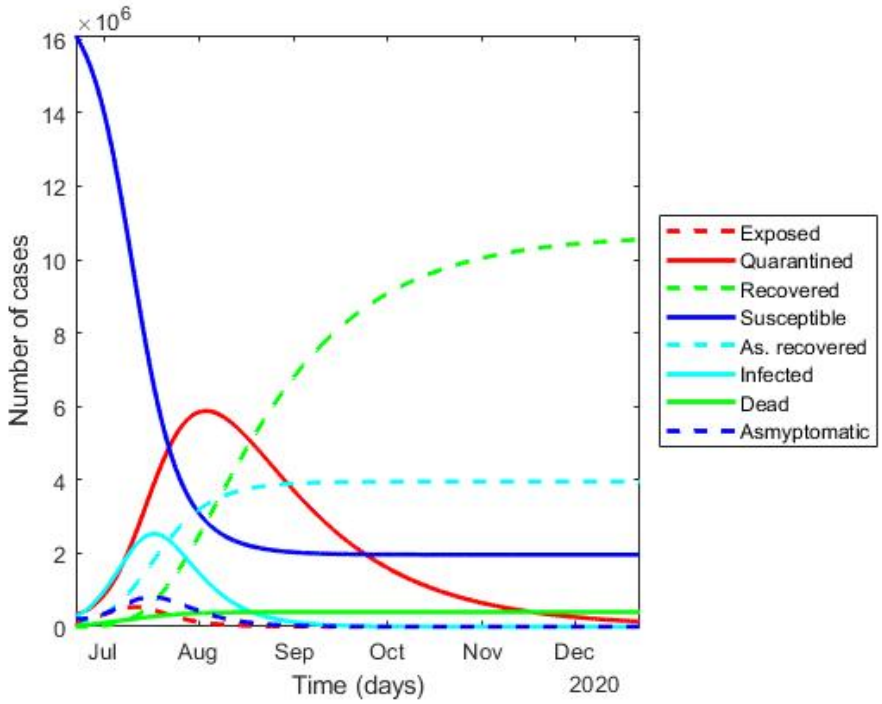

Figure 7: Dynamics of the SEAArIQRD model within a population of 17 million people.

In Figure 7 we see the expected dynamics of the SARS-CoV-2 virus simulated by the modeled presented. The susceptible curve decreases as fast as the asymptomatic and symptomatic curves reach their peak. The model predicts a scenario where the last isolated individuals will be free of the disease in about 5 months. This clearly is not accurate with the current situation, this is because of the lack of transparency in the data given by the Ecuador government, the disobedience in bio-safety issues by the citizens and, also, we are not taken into account the variants of the SARS-Cov-2 virus. This model works for the first wave of contagion after the mandatory lockdown for all population.

We fix the contact weights as in [10]. Then we put 0.18, 0.33. 0.3, 0.19 for general community, households, workplaces, and agglomeration places, respectively. It is important to mention that we consider the same pack of initial values in every network.

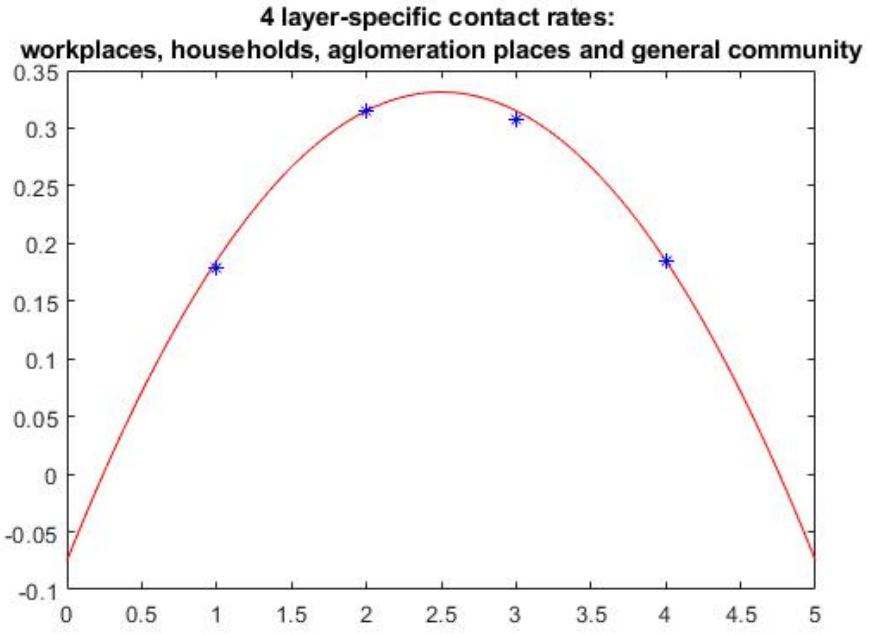

Figure 8: 4 layer-specific contact weights. The contacts rates are fixed to 0.18, 0.33, 0.3, 0.19 for general community, households, workplaces and agglomeration places, respectively.

This contact weight influences the transmission rates, β_{SA} and β_{SI} which are computed as

$$\beta_{SA} = \omega_l p_{SA} \text{ and } \beta_{SI} = \omega_l p_{SI}$$

where p is the probability of transmission per contact, 1/days, and ω_l is the specific contact weight in layer l, $l \in \{c, h, w, a\}$. Here the set $\{c, h, w, a\}$ is the set of layers: c, w, h, a; that represents general community, households, workplaces and agglomeration places, respectively.

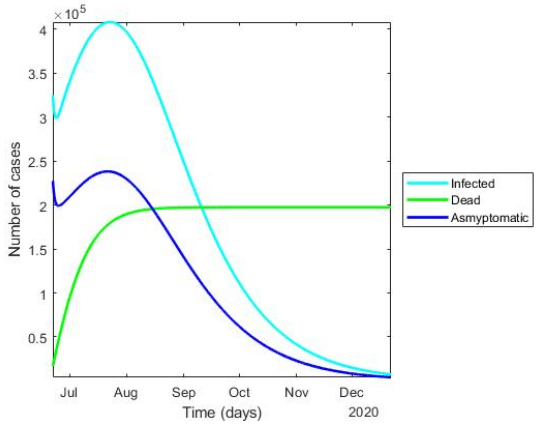

Figure 9: Amount of infected, I(t), asymptomatic, A(t) and dead, D(t) cases in general community network from July to December 2020.

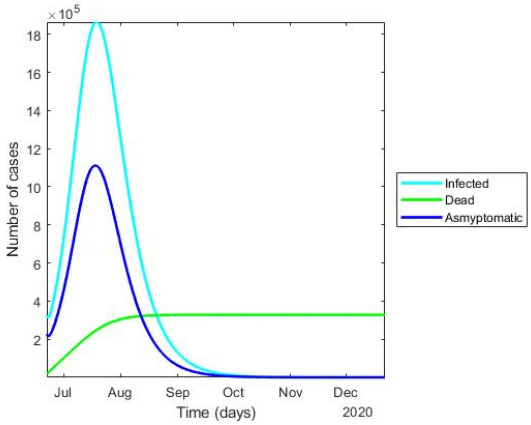

Figure 10: Amount of infected, I(t), asymptomatic, A(t) and dead, D(t) cases in households network from July to December 2020.

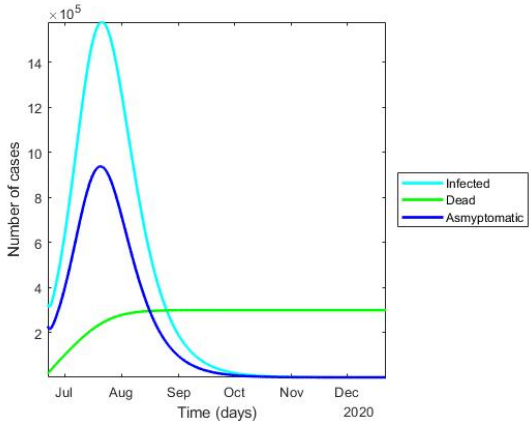

Figure 11: Amount of infected, I(t), asymptomatic, A(t) and dead, D(t) cases in workplaces network from July to December 2020.

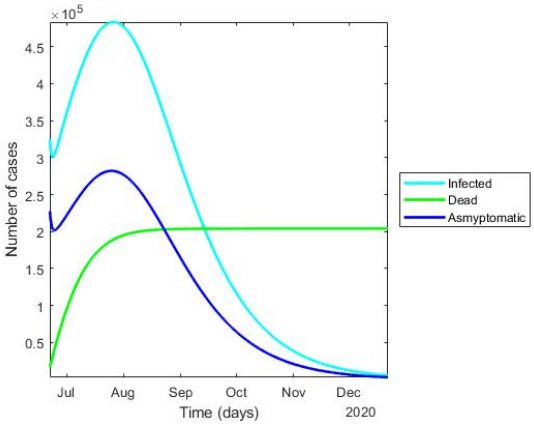

Figure 12: Amount of infected, I(t), asymptomatic, A(t) and dead, D(t) cases in places of agglomeration from July to December 2020.

In Figures 9 and 12, the three classes of interest present a similar dynamic. In community places as neighborhoods the contact between individuals is low because of the social distancing measures. This shows how the peak of infectious (infected and asymptomatic) individuals is relatively low. Also, the dead class stabilizes as the infected curve falls, this is accurate since only infected individuals can die. Here we consider agglomeration places as supermarkets where some normative rules must be obeyed to be in operation. Although the contact weight in agglomeration places is lightly higher than in general community, Figure 12 shows how the amount of infected, asymptomatic and dead individuals increases in 15% with respect to Figure 9.

General Community	Infected	Asymptomatic	Recovered
Workplaces	72%	75%	34%
Households	78%	78%	44%
Agglomaration places	15%	13%	10%

Table 1: Comparison of the increase in the number of Infected, Asymptomatic and Dead individuals in each layer with respect to the General Community network.

The model shows that in every layer, the curves reach their pick at the same moment. In Figures 11 and 10, the amount of individuals in every single class drastically increases in approximately 72% with respect to Figures 9 and 12. Notice that the household infected curve increases near 18% with respect to the workplace curve. The contact weight in workplaces and households is higher because the dynamic within this networks social distancing or the use of masks (only in households) does not intervene.

3.3 R_0 computation

To compute the basic reproduction number we will use the next-generation approach. Although we have 8 classes, we only consider the infected and infectious ones. Also, notice that at

infection-free steady $E = A = I = Q = 0$, then $S = N$. Then we have the following linear system:

$$\begin{aligned}
E'(t) &= \beta_{SA}A(t) + \beta_{SI}I(t) - (\gamma + \sigma)E(t) \\
A(t)' &= \sigma E(t) - M_{Ar}A(t) \\
I'(t) &= \gamma E(t) - \delta I(t) \\
Q'(t) &= \delta I(t) - (\lambda(t) + \kappa(t))Q(t).
\end{aligned}$$

Now, we define the vectors $\mathcal{F}$ and $\mathcal{V}$ as,

$$\mathcal{F} = \begin{pmatrix} \beta_{SA}A + \beta_{SI}I \\ 0 \\ 0 \\ 0 \end{pmatrix}, \qquad \mathcal{V} = \begin{pmatrix} (\gamma + \sigma)E \\ -\sigma E + M_{Ar}A \\ -\gamma E + \delta I \\ -\delta I + (\lambda(t) + \kappa(t))Q \end{pmatrix}.$$

It follows that,

$$F = \begin{pmatrix} 0 & \beta_{SA} & \beta_{SI} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \gamma + \sigma & 0 & 0 & 0 \\ -\sigma & M_{Ar} & 0 & 0 \\ -\gamma & 0 & \delta & 0 \\ 0 & 0 & \delta & \lambda(t) + \kappa(t) \end{pmatrix}$$

Finally the reproduction number is obtained as the spectral value of FV^{-1},

$$\begin{aligned}
R_0 &= \rho(FV^{-1}) \\
&= \rho\left(\begin{pmatrix} 0 & \beta_{SA} & \beta_{SI} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}\begin{pmatrix} \gamma + \sigma & 0 & 0 & 0 \\ -\sigma & M_{Ar} & 0 & 0 \\ -\gamma & 0 & \delta & 0 \\ 0 & 0 & \delta & \lambda(t) + \kappa(t) \end{pmatrix}^{-1}\right) \\
&= \rho\left(\begin{pmatrix} 0 & \beta_{SA} & \beta_{SI} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}\begin{pmatrix} -\frac{1}{\gamma+\sigma} & 0 & 0 & 0 \\ 0 & -\frac{1}{M_{Ar}} & 0 & 0 \\ \frac{\gamma}{(-\sigma-\gamma)\delta} & 0 & \frac{1}{\delta} & 0 \\ -\frac{\gamma}{(-\sigma-\gamma)(-\kappa(t)-\lambda(t))} & 0 & \frac{1}{-\lambda(t)-\kappa(t)} & \frac{1}{-\lambda(t)-\kappa(t)} \end{pmatrix}\right) \\
&= \rho\begin{pmatrix} -\frac{\beta_{SI}\gamma}{(\sigma+\gamma)} & -\frac{\beta_{SA}}{M_{Ar}} & -\frac{\beta_{SI}}{\delta} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \\
R_0 &= \frac{\beta_{SI}\gamma}{(\sigma + \gamma)\delta}.
\end{aligned}$$

Contact Networks	Basic Reproduction Number, R_0
General community	1.01
Workplaces	1.68
Households	1.85
Agglomaration places	1.07

Table 2: Basic Reproduction Number, R_0, in 4 contact network layers: General community, Workplaces, Households and Agglomeration places.

Then the basic reproduction number for general community, households, workplaces and agglomeration places are 1.01, 1.68, 1.85, and 1.07. Then $R_0 > 1$ for every layer which represents an outbreak in every single network. The expected number of cases directly generated by one infected case is clearly higher when the individuals are in closed space with no distancing measures as in the households. In workplaces as offices, the contact rate is also high and that is why the home office is so important. Here, agglomeration places are considered sites where all bio-security measures are fully complied with. Consequently, the reproduction number is relatively low. Finally, in the general community network we have the lowest R_0 because of the poor contact rate.

4 Conclusions and recommendations

4.1 Conclusions

The whole world is being affected by the pandemic caused by the SARS-CoV-2 virus. Ever since the WHO declared it as such, it has been the subject of both clinical and mathematical studies. In this document we developed a generalized SEIR epidemiological model that describes the dynamics of the virus in a short period of time. The model consists of 8 non-intersected classes: susceptible, exposed, asymptomatic, asymptomatic recovered, infected, isolated, recovered, and dead. All the parameters were obtained from clinical observations made previously in [1], [8] and [15] and we took the cure and mortality rate as [12] proposed in its study. Our focus was on the dynamics of the infected, asymptomatic and dead classes in 4 different networks: households, workplaces, agglomeration places and general community. The dynamics of the 3 compartments have the same nature in each layer, as expected. In this work it was observed how the speed of the disease considerably increase in the networks with the highest weight of contacts. This is because in closed spaces such as a home or workplaces, the probability of get infected with the disease is quite high. On the other hand, in crowded places or common squares, this speed is diminished thanks to social distancing and the use of masks. This is evidenced by the reproduction number, R_0, which was greater than 1 in all networks, which is a necessary constraint for an infectious disease to be considered a pandemic.

This work presented difficulties when we compared it with the current situation. This is because we discard several variables of the SARS-Cov-2 pandemic in order to make a-not-so-hard-to-compute model. It is also assumed that the population follows the bio-security norms. Modeling a spread disease is hard but it is even harder to model the human behaviour. Also, recently has been discovered new strains of this virus. This model only takes into account the original spread. Finally, we have presented the first wave of cases after the mandatory lockdown.

4.2 Recommendations

- We invite more students of the mathematics major at Yachay Tech to follow this line of study since, thanks to all our previous interdisciplinary training, nice results can be obtained. Mathematics is everywhere, it is our job to open the eyes to the world.
- The proposal of a university like Yachay Tech undoubtedly attracts the attention of many of the most talented and potential students in this country, but this must be accompanied by institutional stability. The students here deserve more, we are just asking for the basics.
- Transparency of the data provided by the government is recommended in this health crisis. This is a huge obstacle to the development of this kind of research. The joint work of the academy and the government is essential for a prosperous future for Ecuador.

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