CHAPTER
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Modeling and understanding social isolation in the evolution of the COVID-19 pandemic

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ABSTRACT
In December 2019, an outbreak that began in the city of Wuhan, China, sparked what would become a pandemic characterized by an infectious disease caused by SARS-CoV-2. Already at the beginning of 2020, the scientific community had been involved, along with political decisions, in a profound and protagonistic way in the development of measures to combat and contain the evolution of the pandemic. Among the issues discussed, social distancing gained prominence due to the uncertainty between the effectiveness and the economic and social impact generated by its implementation. In this work, we evaluated some epidemiological models that emerged because of the need to insert social distancing in the evolution of the number of infected. We discuss how such models help in understanding social isolation as a combat measure and conclude that such measures, when expressive, can help reduce the number of infections, especially when implemented at the beginning of the epidemic due to the low geographical spread.

Keywords: Modeling, Social Isolation, Epidemiology.

1 INTRODUCTION

Throughout history, humanity has lived and recorded major epidemics arising from contact with animals, sanitary conditions, and lack of etiological knowledge. Rezende (2009, p. 74) points out, as the largest recorded epidemics, the plague of Athens, the plague of Syracuse, the Antonine plague, the plague of the third century, the Justinian plague, and the Black Death of the fourteenth century.

In his book *History of the Peloponnesian War,* Thucydides relates the story of the plague of Athens, which occurred in 428 B.C. The author states that, a few days after the invasion of the Peloponnese's territory of Attica, an epidemic that began in the city of Lemnos attacked countless victims with disconcerting violence, leaving the population with little or no medical support, since "[...] the doctors could do nothing, because at first they did not know the nature of the disease and moreover they were the first to have contact with the sick and died first" (Tucídides, apud Rezende, 2009, p. 75).

Already in the second century AD, the Antonine plague was characterized by the great devastation of Rome and its extension throughout Italy, in addition to having two waves of growth, the first in 166 AD and the second in 189 AD (Rezende, 2009, p. 76)).

Later, in the fourteenth century, humanity experienced the most tragic epidemic in history, called the *Black Death* due to the dark spots that characterized the sick (REZENDE, 2009). The Black Death had large-scale economic and social impacts. The Archbishop of Winchester, for example, charged a rise in the rate of *heriots (*paid to the lord in case of transfer of lieutenants, in general, by death) from an average of 52 per 1000 to 500 per 1000 during the epidemic (SILVA, 1973, p. 388). Its spread began in the East, but reached France in 1348, by sea, followed by Italy in 1349 and later throughout Europe (REZENDE, 2009, p. 78).

After more than a century without experiencing this type of phenomenon, in December 2019, a pandemic caused by the SARS-CoV-2 virus began in the city of Wuhan, China. The high rate of transmission and spread across several countries, in addition to the first deaths recorded in January 2020 in China and in February 2020 in France, led the WHO to officially treat the problem as a global health emergency (OLIVEIRA; JANUARY; SALLES et al., 2021).

Thus, unlike previous epidemics, this was the first time that identification and combat could already be initiated in the first months of spread. The fight against the pandemic began with the use of mandatory sanitary measures in public settings, such as the use of masks and alcohol gel, as well as an intense scientific "race" for the development of vaccines, as an immunization mechanism, and epidemiological models as tools to assist in making decisions of social impact, such as isolation measures.

One year after the beginning of the pandemic, 261 vaccines were being developed worldwide, 79 of which were in human trials (phase III), and 9 with approval for emergency use (OLIVEIRA; JANUARY; SALLES et al., 2021, p. 26). In addition, several epidemiological models were developed to predict the progress of the pandemic, taking into account the scenarios of social isolation, geographical evolution, application of vaccines, among others. Much of this rapid development took place through *preprints* that allowed for rapid communication between the scientific community. Rocha and Araújo (2021), in a survey on the online attention of preprints, pointed out that until September 2021 56,690 preprints on the theme "covid-19" were shared in the *Dimensions database*.

In this paper, we discuss some epidemiological models that have emerged throughout and for the analysis of the Covid-19 pandemic. We extract theoretical consequences from the implementation of parameters for the evaluation of social isolation in these models. Finally, we have gathered the main results that can be used in the future in the fight against epidemics.

2 EPIDEMIOLOGIC MODELS

In an attempt to understand the mechanisms by which epidemics gain strength, mathematical models were created from various methodologies that aim to explore their dynamics, prediction and

control. Stochastic models, for example, analyze the probabilistic aspect of the spread of epidemics (ALLEN, 2008). Network models or agents, on the other hand, explore the interactions between individuals in the population analyzed to simulate the spread of the epidemic and can be very realistic, especially by employing a large amount of data, such as population density, age structure and social groupings in schools, businesses and the labor market (FERGUSON; CUMMINGS; FRASER et al., 2006). Finally, dynamic models based on differential equations and on the division of the population into compartments related to each stage of infection (e.g. susceptible, exposed, asymptomatic, infected, recovered, hospitalized) constitute a simpler alternative, both from a numerical and theoretical point of view, regarding the interpretation of the consequences of each simulated scenario for the end of the epidemic (LÓPEZ-FLORES; MARCHESIN; MATOS & SCHECTER, 2021).

The models we will discuss here originate from the 1927 work published by A. G. McKendrick and W. O. Kermack, who provided the first set of differential equations based on the compartmental methodology of populations (KERMACK & MCKENDRICK, 1927)). The SIR (Susceptible-Infected-Removed) model they created models a non-fatal epidemic, where individuals become infected from a susceptible population and recover from this infection, gaining immunity.

The set of equations of the SIR model is written as follows:

$$\begin{cases} \dot{S} = -\beta SI \\ \dot{I} = \beta SI - \gamma I \\ \dot{R} = \gamma I \end{cases} \qquad (I)$$

where β is the infection rate and the γ recovery rate of individuals. In this set of equations$S = S(t)$, is the fraction of the susceptible population, the fraction of the infected population, and the fraction $I = I(t)$ of the recovered population, so that $R = R(t)$ and $S(t) + I(t) + R(t) = 1. \dot{S}(t) + \dot{I}(t) + \dot{R}(t) = 0$

Other epidemiological models can be constructed from system-like structures by adding or removing new compartments for different populations. Table 1 presents other epidemiological models and their main characteristics:

Table 1 – Compartmental epidemiological models

Acronym	Equations	Populations	Constants
SIS	$\begin{cases} \dot{S} = -\beta SI + \gamma I \\ \dot{I} = \beta SI - \gamma I \end{cases}$	Susceptible Infected	Infection rate (β) Recovery rate (γ)
SIRS	$\begin{cases} \dot{S} = \delta R - \beta SI \\ \dot{I} = \beta SI - \gamma I \\ \dot{R} = \gamma I - \delta R \end{cases}$	Susceptible Infected Retrieved	Infection rate (β) Recovery rate (γ) Rate of immunity loss (δ)
SEIR	$\begin{cases} \dot{S} = -\alpha SI \\ \dot{E} = \alpha SI - \beta E \\ \dot{I} = \beta E - \gamma I \\ \dot{R} = \gamma I \end{cases}$	Susceptible Exposed Infected Retrieved	Exposure rate (α) Infection rate (β) Recovery rate (γ)

Source: the authors.

Each of these models presents different etiological characteristics since different causes of the epidemic are associated with different biological consequences. In the SIS model, for example, the main feature that differentiates it from the SIR model is the fact that recovered individuals lose immunity and return to the susceptible population. In the SIRS model, the same occurs, but with only a part of the population recovered. Finally, the SEIR model is characterized by the distinction between the population exposed to contact with infected individuals and the infected population, if a fraction of the interactions between susceptible and infected do not produce infections.

3 SIR MODEL WITH GEOGRAPHIC AND SOCIAL ISOLATION

Throughout the Covid-19 pandemic, one of the main issues raised in parallel with vaccine development has been the implementation and consequences of social isolation measures. In April 2020, we shared a preprint analyzing a model that aimed to adapt the system's equations to include the isolation effects associated with the geographic spread of Covid-19 across Brazil and social distancing measures (SCHULZ; COIMBRA-ARAÚJO & COSTICHE, 2020).

These adaptations arose, first, because it should be noted that the SIR model supposes, as well as the others presented in Table 1, that initially the entire uninfected population is susceptible to contagion, that is, that $S(0) = 1 - I(0)$. However, in a pandemic context like Covid-19, this assumption can lead to an exaggerated estimate of the number of contagions, especially in the initial modeling stage. For example, the first case of Covid-19 in Brazil was recorded on February 26, 2020, in the state of São Paulo. From this fact, it does not follow that the population of distant states such as Amazonas, Sergipe and Rio Grande do Sul can be considered as susceptible in February, since it is not reasonable to assume that individuals can spread the infection throughout the country immediately.

Thus, one way to alleviate this hypothesis is to assume that the *potentially* susceptible population $S(t)$ becomes susceptible to the extent that *natural isolation*, due to geographical separation, is reduced. Thus, if it is $\rho(t)$ the percentage of the population that is potentially susceptible, but that is not effectively susceptible due to geographic isolation, it is possible to assume that:

$$\rho(t) = \rho_0(1 - \Delta\rho)^t \quad (1)$$

where ρ_0 is the population initially geographically isolated, and the $\Delta\rho$ percentage reduction of the population in geographic isolation.

Secondly, the Covid-19 pandemic was marked by social distancing and isolation measures, characterized by policies of reducing/banning public events and gatherings such as schools, universities, concerts, sporting events, commerce, among others (PIGLET; FERREIRA; DE ABREU et al., 2021). Just like geographic isolation, social isolation measures act by reducing the potentially susceptible population, so that if it $\varphi(t)$ is the percentage of the population socially isolated, then the effectively susceptible population $S_e(t)$ can be obtained by taking:

$$S_e(t) = S(t)\big(1 - \rho(t) - \varphi(t)\big) \quad (2)$$

From the equation , the SIR system with geographic and social isolation becomes:

$$\begin{cases} \dot{S} = -\beta S_e I \\ \dot{I} = \beta S_e I - \gamma I \\ \dot{R} = \gamma I \end{cases} \quad (II)$$

which bears a natural resemblance to the system . In general, therefore, isolation measures, whether natural or intentional, constitute ways to reduce the susceptible population to only part of the total population and, consequently, to reduce the proportion of the infected population.

Also in 2020, other work addressing social isolation emerged based on the SIR or on more robust models, such as modified SEIR. Gomes, Monteiro and Rocha (2020), for example, used the SIR to predict the evolution of the Covid-19 pandemic in some Brazilian cities and proposed to model the isolation of the susceptible population by considering an effective infection rate$\beta_e = \beta p$, where $0 < p < 1$ is the percentage of the population in circulation. In addition, they suggested an effective recovery rate, $\gamma_e = \gamma + k$where k it is the rate of isolation of the infected population that goes into isolation and stops infecting new individuals. Using a modified SEIR model, Cardoso, Sousa, Cardoso and Utsumi (2020) modeled social isolation measures for the state of Minas Gerais and Brazil by considering a variable infection rate as follows:

$$\beta(t) = \begin{cases} \beta_0, & t < t_0 \\ \beta_{min} + (\beta_0 - \beta_{min})^{-r(t-t_0)} & t \geq t_0 \end{cases} \quad (3)$$

where is the minimum infection rate and β_{min} is the rate of decrease in the infection rate$r.\beta_0$

Each modified model allows an adaptation to the context from which the actual data is obtained. The equation, for example, can be useful when the actual scenario to be modeled is a country or continent, but it won't be as relevant to modeling a statewide or city-wide epidemic. The equation may not be satisfactory if the changes in the infection rate are abrupt, and do not decline continuously, as in the case of a significant and abrupt lockdown.

4 DYNAMICS IN ISOLATION

To understand how social isolation measures impact the dynamics of epidemiological systems, we must first analyze their natural dynamics, that is, without isolation measures. To do so, we will obtain the fundamental characteristics of the system and then we will go on to check how these characteristics change in the system. The SIR model was chosen for this analysis due to its simplicity, however, similar approaches may lead to obtaining equivalent results for the other systems presented in Table 1 (LÓPEZ-FLORES; MARCHESIN; MATOS & SCHECTER, 2021).

Therefore, note that the system can be rewritten as:

$$\frac{dS}{dt} = -\beta SI \tag{4}$$

$$\frac{dI}{dt} = \beta SI - \gamma I \tag{5}$$

taking $R = 1 - S - I$. So, dividing by :

$$\frac{dI}{dS} = -1 + \frac{\gamma}{\beta S} \tag{6}$$

which has as a solution the family of curves:

$$I = -S + \frac{\gamma}{\beta} lnS + C. \tag{7}$$

and the maximization condition of leads, by the equation , to:

$$S = \frac{\gamma}{\beta} = \frac{1}{R_0} \tag{8}$$

which implies two possible cases:

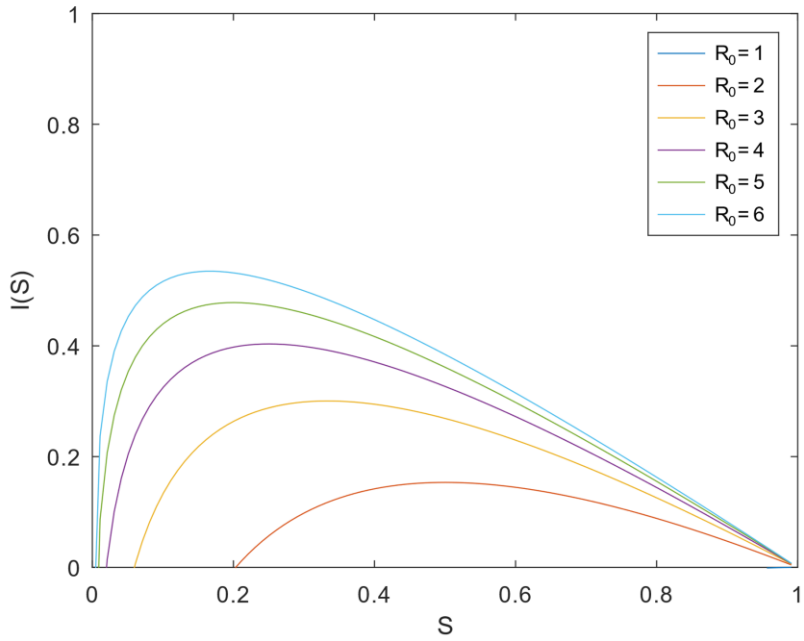
- If $R_0 > 1$, the maximum of I happens to some $S < 1$ and therefore a part of the susceptible population becomes infected.

- If $R_0 \leq 1$, the maximum I happens with $S \geq 1$ and therefore no susceptible individual becomes infected.

ThereforeR_0, the value of establishes a natural condition for the occurrence of an epidemic, namely, that the rate of infection must be strictly higher than the rate of recovery because, otherwise, any initially infected individual will recover before infecting new individuals.

Figure 1 shows the function $I(S)$ given by the equation for $C = 1$ arbitrarily chosen and different values of R_0:

Figure 1 – Function $I(S)$ with $C = 1$



Source: the authors

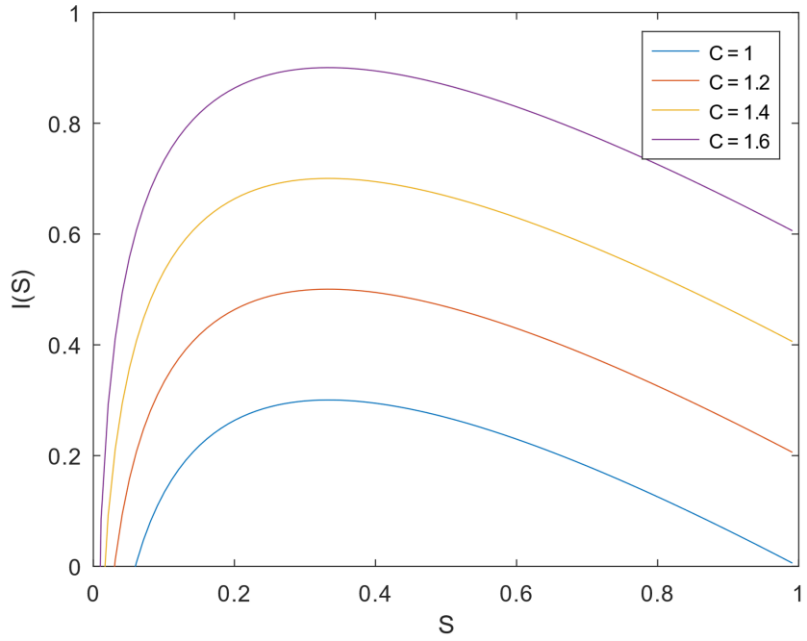
It is easy to notice that larger R_0 values of imply maxima for the function $I(S)$ happening for smaller values of S, as the equation predicts. The for curve $R_0 = 1$ is not displayed due to $I(S)$ being null in almost the entire domain.

Another naturally important characteristic that must be considered is the initial amount of infected individuals, which can be analyzed by the constant in the C equation , since as $S(t = 0) \approx 1$:

$$C \approx 1 + I\big(S(t = 0)\big)$$

and, in this case, the higher the value of , $I\big(S(t = 0)\big)$the higher the value of C and the curve $I(S)$ will be shifted vertically indicating a greater number of contaminations, as shown in Figure 2:

Figure 2 - $I(S)$ with $R_0 = 3$



Source: the authors

Briefly, therefore, these two characteristics determine the dynamics of the system, so that the modeled epidemic will be more expressive as much as the higher the values of R_0 and $I\big(S(t=0)\big)$.

For the system, the meaning and importance of the values of and R_0 $I\big(S(t=0)\big)$ remain the same. The difference, however, lies in the fact that any epidemic modeled by the system will have a lower number of infections due to . $S_e(t) \leq S(t)$ This difference is determined by the functions $\rho(t)$ and $\varphi(t)$ therefore it is necessary to analyze how each of them influences the susceptible population to understand the effects of social isolation on the system.

Let's start by noting that, unlike the system, the system can be controlled through the functions and , that $\rho(t)$ $\varphi(t)$is, we can find functions such that the number of new infections is null and the epidemic is eliminated. That's because the number of new infections (NI) can be determined by doing:

$$= \dot{I} - \gamma I = \beta S_e I$$

Therefore, if we want there to be no new infections, we must have:

$$= \beta S(t)\big(1 - \rho(t) - \varphi(t)\big)I = 0$$

which implies:

$$1 - \rho(t) - \varphi(t) = 0$$

or:

$$\varphi(t) = 1 - \rho(t) \qquad (9)$$

Thus, it follows that social isolation must be as comprehensive as the fraction of the population that is not geographically isolated. That is, especially in the early period of the epidemic, imposing a comprehensive social isolation on the susceptible population geographically located in the vicinity of the infected population is sufficient to mitigate the evolution of the epidemic. On the other hand, the longer the time interval between the beginning of the epidemic and the implementation of social isolation measures, the greater the fraction of the isolated population should be, since, by the equations and :

$$\lim_{t\to\infty} 1-\rho(t) = \lim_{t\to\infty} 1-\rho_0(1-\Delta\rho)^t = 1$$

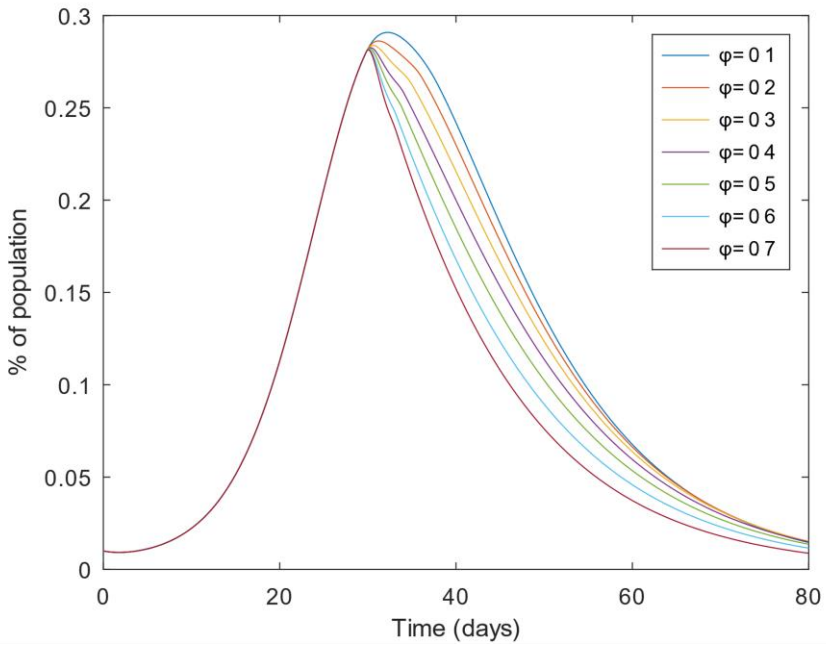
well $\Delta\rho \geq 0$.

To visualize this effect, consider an initial value problem for the system with , with$\big(S(0), I(0), R(0)\big) = (0.99, 0.01, 0)$, $R_0 = 3$ $\beta = 0.3$ and $\rho(t) = 0.8(1-0.01)^t$. In this case, after 30 days we will have $\rho(30) \approx 0.592$ and therefore we can define $\varphi(t)$ as:

$$\varphi(t) = \begin{cases} 0, t < 30 \\ \varphi_0, t \geq 30 \end{cases}.$$

Figure 3 shows the evolution of the number of infected for different values of φ_0:

Figure 3 - $I(t)$ for different values of φ_0



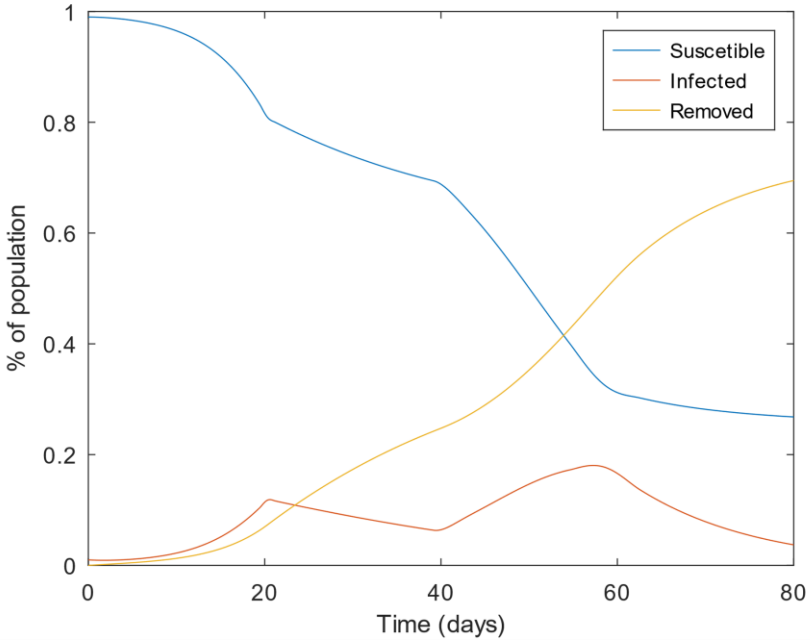
Source: the authors

It is possible to verify that the slope of the graph in Figure 3 becomes negative for values of $\varphi_0 > 0.4$, which agrees with the result predicted by the equation . In addition, the definition by parts of the function $\varphi(t)$ can lead to the creation of more than one wave of infection. For example, if:

$$\varphi(t) = \begin{cases} 0, t \leq 20 \\ 0.7, 20 < t \leq 40 \\ 0, 40 < t \leq 60 \\ 0.7, t > 60 \end{cases}$$

then, for the same initial value problem as before, the system dynamics are shown in Figure 4:

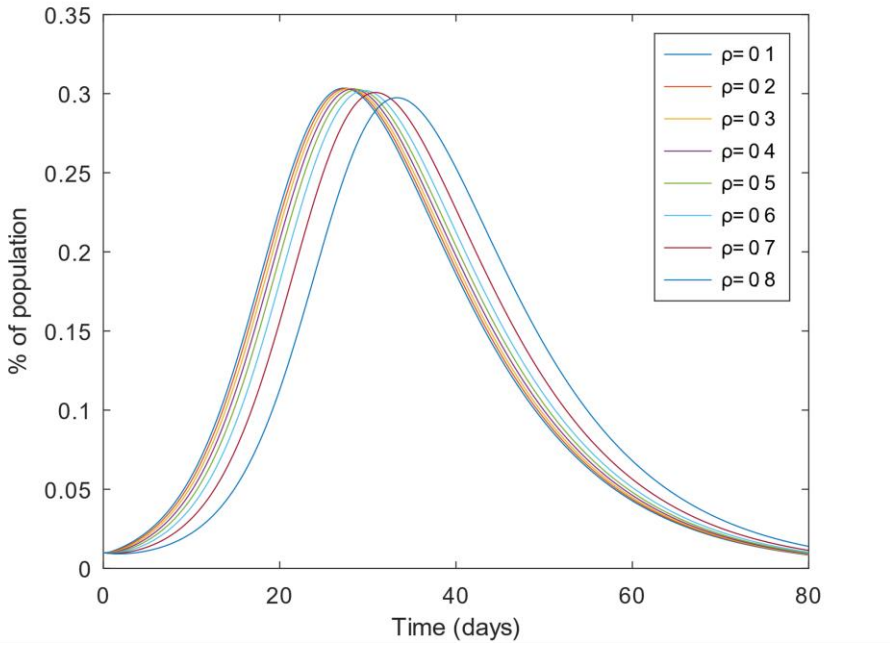
Figure 4 – SIR model with isolation and two waves of infection



Source: the authors

In addition, we can see how geographic isolation acts on system dynamics by delaying the peak of the curve. That is, higher values for the fraction of the geographically isolated population ρ_0 imply a horizontal displacement of the curve $I(t)$, as shown in Figure 5:

Figure 5 – Curve $I(t)$ for different values of ρ_0



Source: the authors

Therefore, while R_0 the values of e determine the criteria for the epidemic to evolve$I\big(S(t=0)\big)$, the values of e $\rho(t)$ $\varphi(t)$ determine the conditions of delay, reduction or creation of multiple waves in the infection curve $I(t)$. However, reproducing the conditions of $\rho(t)$ and , $\varphi(t)$in real epidemics, is a difficult task. This is because the implementation of isolation measures for the population can give rise to the appearance of mental disorders, such as anxiety and depression (FARO; BAHIANO; NAKANO et al., 2020), as well as economically harm the population (PORSSE; SOUZA; OAK & VALE, 2020).

Although the models exhibit objective conditions for an epidemic to be controlled, its real phenomenon is shown as a multifaceted problem that is beyond the hypotheses of the models, interfering in several of the life and health of individuals. However, since social isolation measures can be and have been adopted throughout the Covid-19 pandemic, for example, these models are useful tools for drawing comparisons between the effectiveness of these measures and the cost-effectiveness in relation to their adverse consequences.

5 FINAL CONSIDERATIONS

Throughout the Covid-19 pandemic, which began in late 2019, several epidemiological models have been proposed and modified to meet the need to understand the effectiveness of social isolation measures implemented in several countries.

In this paper, we present some of these models and discuss how measures of geographic and social isolation can be inserted into a SIR model, and from this we extract theoretical consequences that establish conditions of increase or reduction of the infection curve.

We conclude that the construction and use of these models allows us to understand, in an objective way, the effects of delay or reduction of the infection curve and, therefore, constitute useful tools for decision making in epidemic scenarios.

The algorithms used for the production of the figures and for the other models presented in Table 1 were built in the GNU Octave software, mostly using the ode45 function, and will be available to the interested reader through a repository[1] or request to the authors.

[1] Link to access: https://github.com/samuelcostiche/Covid-Algorithms.git.

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