e-ISSN: 2582-5208
International Research Journal of Modernization in Engineering Technology and Science
(Peer-Reviewed, Open Access, Fully Refereed International Journal)
Volume:06/Issue:09/September-2024 Impact Factor- 7.868 www.irjmets.com

BEHAVIOR OF DETERMINISTIC EPIDEMIOLOGICAL MODELS UNDER CERTAIN CONDITIONS

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DOI : https://www.doi.org/10.56726/IRJMETS61396

ABSTRACT

Covid-19, given its media coverage, has emphasized the need to predict the evolution of virus contagion. Hence the need to study some epidemiological models, analyzing their behavior over a period of time.

The SI, SIS and SIR models, although basic models, are models that still explain many epidemic evolutions.

Most authors report that these models behave well for populations of constant size. In this study, the models will be modified in order to analyze whether the entry and exit of elements of the population affect the normal evolution of the epidemic over time. Simulation using Python and Euler's method for solving differential equations.

Keywords: Epidemic, SI Model, SIS Model And SIR Model.

I. INTRODUCTION

Since 2019, the words epidemiology and epidemic have been part of every individual's vocabulary, given the Covid-19 pandemic. Epidemiology can be defined as the study of how diseases spread through populations and the factors that can influence or determine this spread [1]. While, for Chasnov [2], an epidemic occurs when a small number of infected individuals are introduced into a susceptible population and results in an increasing number of infected people. On the other hand, Dobson [3] writes that a disease becomes an epidemic when it infects a substantial number of elements of a population in a short space of time.

According to Ledder [4], theory without observation is a myth, and the opposite is nothing more than a collection of disconnected facts. This implies that the evolution of science is only possible with the combination of the two. Ledder [4] also writes that the link between theory and observation/practice is the domain of mathematical modeling. On the other hand, Allman and Rhodes [5] point out that mathematical language is designed for precise descriptions, which allows describing complicated systems that often require a mathematical model.

To study the impact and spread of diseases in a population, models based on mathematical and statistical techniques are used. Second, Torres and Santos [6] write that a model is a conceptual or mathematical representation of a system that serves to understand it and quantify the evolution of an epidemic. The best-known types of models are: behavioral and stochastic models.

According to Allen [7], one of the most important differences between deterministic and stochastic epidemic models is their asymptotic dynamics; whereas, stochastic solutions converge to a disease-free state, while the corresponding deterministic solution converges to an endemic equilibrium.

Behavioral or deterministic models are models that represent the spread of a disease through differential equations that describe how infection, recovery and mortality rates change as a function of time and other factors. These are useful for understanding general trends and making long-term predictions. On the other hand, Torres and Santos [6] write that in deterministic models, velocities depend only on the concentration of elements and model parameters.

Stochastic models, unlike deterministic models, take into account the randomness and uncertainty associated with the spread of a disease. These use probabilistic methods to simulate multiple possible scenarios and assess the likelihood of different outcomes. Torres and Santos [6] state that in stochastic models the velocities also depend on the random noise of the system, due to the uncertainty present in systems containing statistically non-abundant elements.

These models can be used for a variety of purposes, such as predicting the spread of disease, assessing the impact of public health interventions (such as vaccinations or social distancing measures), identifying at-risk populations, and designing disease control strategies. However, it is important to remember that all

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epidemiological models are subject to certain limitations and assumptions and should be interpreted with caution. In this study the main objective is to just study some behavioral models.

II. DETERMINISTIC EPIDEMIOLOGICAL MODELS

Deterministic models are a category of mathematical models that describe the behavior of a system over time using certain deterministic rules and equations. This means that given the same initial conditions and parameters, the model will always produce the same result. They are widely used in epidemiology to predict the behavior of infectious diseases in a given population.

According to Brauer et al. [8], in these models the study population is divided into compartments and assumptions are made about the nature and rate of transfer from one compartment to another. These different compartments, representing different health states and exposure to disease.

Deterministic models are useful for understanding general disease spread trends and for evaluating the impact of public health interventions. However, they have some limitations, such as assuming homogeneity in the population and not capturing stochastic variability in disease transmission.

The models under study will be the models: SI, SIS and SIR.

SI Model

The SI (Susceptible-Infested) model is one of the simplest models in epidemiology, where there are only susceptible and infested individuals. In this type of model, a healthy (susceptible) person can become infectious, since each individual in the population has the same probability of coming into contact with all other elements of the population, and if an individual in this population becomes infected, the disease it is permanent (there is no recovery). This model is best suited for populations where there is no mobility or change in the number of individuals: no losses or new acquisitions.

According to Chasnov [2], when deriving the base differential equation for this model, considering the number of people who become infected and with the capacity to infest another individual over time Δt and let $\beta\Delta t$ be the probability that a randomly chosen infected individual infects another randomly selected individual susceptible during time Δt. Then, with S susceptible people and I infected people, the expected number of newly infected people in the total population during time Δt is $\beta\Delta tSI$ and β a constant that represents the average contact of an infected individual with a susceptible individual. Therefore, we can define the equilibrium equation as being:

$$I(t + \Delta t) = I(t) + \beta\Delta tS(t)I(t)$$

When $\Delta t \to 0$ and assuming that the population consists of N elements, then the number of infected people can be calculated, by:

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

As the number of infected people increases, the number of susceptible people decreases, so we can represent this action by:

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

Transposing these two differential equations to a system in which the number of susceptible individuals S(t) and the number of infected people I(t) appear as time-dependent variables t. According to the principle of mass action (a mathematical model that explains and predicts behavior of solutions in dynamic equilibrium), the rate of change of an individual from a susceptible state to an infected state is proportional to the product of the size of both populations:

$$\begin{cases} \frac{dS}{dt} = -\beta S(t)I(t) \\ \frac{dI}{dt} = \beta S(t)I(t) \end{cases} \quad (1)$$

Therefore, $\beta > 0$ is the infection rate, then $\beta S(t)I(t)$ is the number of susceptible individuals that are infected per unit time.

Although this model is indicated for populations where there are only infested and susceptible individuals in populations of constant number, as previously mentioned. But assuming that we are in a real population where there are entries and exits of elements from the population. These entries refer to births and/or individuals

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coming from other populations, while exits refer to individuals who die and/or leave this population to go and live in another. If we take μ and α to be the rates of entry and exit of individuals, respectively. Adding the inputs and outputs to equation 1, we obtain:

$$\begin{cases} \frac{dS}{dt} = \mu N - \beta S(t)I(t) - \alpha S(t) \\ \frac{dI}{dt} = \beta S(t)I(t) - \alpha I(t) \end{cases} \quad (2)$$

SIS Model

The SIS (Susceptible-Infected-Susceptible) model, as its initials indicate, is aimed at infections, to which individuals have no immunity.

In humans, according to López-Flores et al. [9], some infectious diseases spread due to the combination of pathogenic characteristics and human behavior. Pathogenic characteristics determine the circumstances under which a contagious person can infect another, and human behavior determines the frequency with which these circumstances occur.

According to Brauer et al. [8], SIS terminology is used to describe a disease without immunity against reinfection, to indicate that the transition of individuals occurs from the susceptible class to the infectious class and then back to the susceptible class. In other words, as there is no immunity to this type of disease, a susceptible individual can become infected, remaining in this state until cured. When cured, it returns to the susceptible state.

In this model, the total population N (without births or deaths) is studied, divided into two groups, (S)usceptible and (I)infected, which evolve over time t. The variations between them are a consequence of the contagion of susceptible individuals by infected individuals, and of infected individuals who recover and become susceptible again. Contagion occurs through a contagion rate $\beta > 0$ that depends on each disease and both groups, while recovery occurs through a recovery rate $\gamma > 0$ that depends only on the group of infected individuals that exists at any given time.

According to Chasnov [2], the SI model can be extended to the SIS model, where an infected individual can recover and become susceptible again. We assume that the probability of an infected individual recovering during time Δt is given by γΔt. Then the total number of infested individuals that recover during time Δt is given by $I \times \gamma\Delta t$, which can give rise to the equilibrium equation:

$$I(t + \Delta t) = I(t) + \beta\Delta t S(t)I(t) - \gamma\Delta t I(t),$$

and since $\Delta t \to 0$, we obtain:

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

In the same way, we can calculate the validation of those infected, according to:

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

where β and γ are constants of positive proportionality.

Transposing these two equations into a system of two equations and two unknowns:

$$\begin{cases} \frac{dS}{dt} = -\beta S(t)I(t) + \gamma I(t) \\ \frac{dI}{dt} = \beta S(t)I(t) - \gamma I(t) \end{cases} \quad (3)$$

Which, according to Chasnov [2], is based on the assumptions that:

- The rate of new infections is given by the incidence of mass actions.
- Those infected leave the infectious state and are no longer able to infect others at a rate γI per unit of time and are susceptible to becoming infected again.
- No change in the number of members of the population.
- There are no losses of elements and the total population size is constant N.

Taking into account the previous assumptions, we have: $N = S + I$. Então, taking into account Smith [10], that if we add the equations, we have: $\frac{dI}{dt} + \frac{dS}{dt} = \frac{dN}{dt} = 0$, which is in line with the fact that the population size is

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constant. We can try to solve the system of equations 3 directly and find the time series of the temporal evolution of the disease, where the time variable is explicit.

Although the bibliography consulted always mentions that this model is aimed at constant populations, without inputs and outputs of population elements, a small change was made to simulate the SIS model in a variable population. To test an SIS model in a variable population, we took: μ and α as the entry and exit rates of individuals, respectively. Taking equation 3, and adding the entry rates associated with N and the exit rates associated with infected and susceptible people, we obtain:

$$\begin{cases} \frac{dS}{dt} = \mu N - \beta S(t)I(t) + \gamma I(t) - \alpha S(t) \\ \frac{dI}{dt} = \beta S(t)I(t) - \gamma I(t) - \alpha I(t) \end{cases} \quad (4)$$

SIR Model

The SIR (Susceptible-Infected-Recovered) model is one of the most basic and widely used models. In this model, susceptible individuals (S) can contract the disease if they come into contact with infected individuals, infected individuals (I) can transmit the disease to susceptible individuals and eventually recover, and recovered individuals (R) are immune to the disease and cannot be infected again (or there is a very low rate of reinfection). In Chasnov [2], and Smith [10], the authors call those recovered removed. The term "removed" is a general term that allows infected individuals to no longer be infected but also to be no longer susceptible. In practice, this can mean that the person improves: either through treatment, natural immunity or that the person dies.

The SIR model without variation in the number of elements in the population is an extension of the previous SI and SIS models, but with the difference that those who leave the infected class cannot be infected again, passing into the recovered class. Recovered individuals, for the purposes of the model, are considered permanently immune, even those who died as a result of the disease or were isolated until they achieved permanent immunity. In López-Flores et al. [9], the authors consider that the ideal situation to apply this model is to be considered an infectious disease that is not fatal and that provides permanent immunity to people who contract it.

The SIR model is formulated as a set of ordinary differential equations (ODEs), which describe how rates of change in the number of individuals in each compartment depend on transmission and recovery rates. In its simplest model it can be formulated as the next system of differential equations:

$$\begin{cases} \frac{dS}{dt} = -\beta S(t)I(t) \\ \frac{dI}{dt} = \beta S(t)I(t) - \gamma I(t) \\ \frac{dR}{dt} = \gamma I(t) \end{cases} \quad (5)$$

Where, according to Martcheva [11], the number of individuals in each of the classes varies over time, that is: $S(t)$, $I(t)$ and $R(t)$, are the fractions of the population in each compartment at time t. Although, at any time we have: $S(t) \geq 0$, $I(t) \geq 0$ and $R(t) \geq 0$, where: $S(t) + I(t) + R(t) = N$, when of the initial conditions, for $t = 0$ and $R(t) = 0$, given that no one has recovered yet. The rate of change of susceptible individuals is proportional to the SI, with a proportionality constant $\beta > 0$, while individuals are removed from the infectious class at a rate proportional to the size of class I, with a proportionality constant $\gamma > 0$.

When we consider a variable population, the description of the system becomes more complete, and consequently more complex. Since we will have individuals being born, which implies an increase in the number of beings susceptible to the disease and we will have individuals dying, so that when considering a large time interval, the tendency is for the disease to reach a more or less stationary state and not be extinguished. Therefore, the system of equations 5 can be modified to:

$$\begin{cases} \frac{dS}{dt} = \mu N - \beta S(t)I(t) - \alpha S(t) \\ \frac{dI}{dt} = \beta S(t)I(t) - \gamma I(t) - \alpha I(t) \\ \frac{dR}{dt} = \gamma I(t) - \alpha R(t) \end{cases} \quad (6)$$

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Where μ and α represent the entry and exit rates of individuals, prospectively. These modifications are consistent, because for the susceptible group, we are counting new individuals introduced into the system (births and coming from other non-infected populations) μN as well as the death and exit to other populations of individuals susceptible to the $\alpha S(t)$ disease. How should the departures for those infected and those recovered be counted, hence the negative sign in the last two expressions.

The SIR model can also be used when some fraction of the population is not susceptible to the disease for genetic, behavioral, immunological reasons, etc. This fraction of the population is included in the recovered compartment from the beginning.

III. SIMULATION AND RESULTS

Model To better visualize the model, Python was used to simulate the number of individuals at different stages as a function of time. The code is the same for all three models, changing the equations depending on the model and adding the necessary variables for the model simulation.

Mathematical models for biological and medical sciences are based on a variety of forms, such as: difference equations, ordinary or partial differential equations [12]. The models studied in this article use ordinary differential equations (ODEs).

To simulate the models described above, the Euler method will be used to calculate differential equations, with a $h = 0{,}1$.

Usou-se também uma população de tamanho $N = 500$, uma taxa de contágio $\beta = 0{,}002$ numa dimensão temporal de 30 dias. O valor do β foi calculado em função do numero de infetados na população no momento zero sobre o numero total de indivíduos $\beta = \frac{1}{500} = 0{,}002$.

A population of size $N = 500$ was also used, with a contagion rate of $\beta = 0{,}002$ in a temporal dimension of 30 days. The value of β was calculated as a function of the number of infected people in the population at time zero over the total number of individuals: $\beta = \frac{1}{500} = 0{,}002$.

SI Model

As previously mentioned, the SI model is the simplest where the population is either susceptible to contracting the disease or is infected. On the other hand, as none recover, there comes a certain time when all individuals are infected. Hence, only the contagion rate is necessary, in addition to other general variables such as sample size and time.

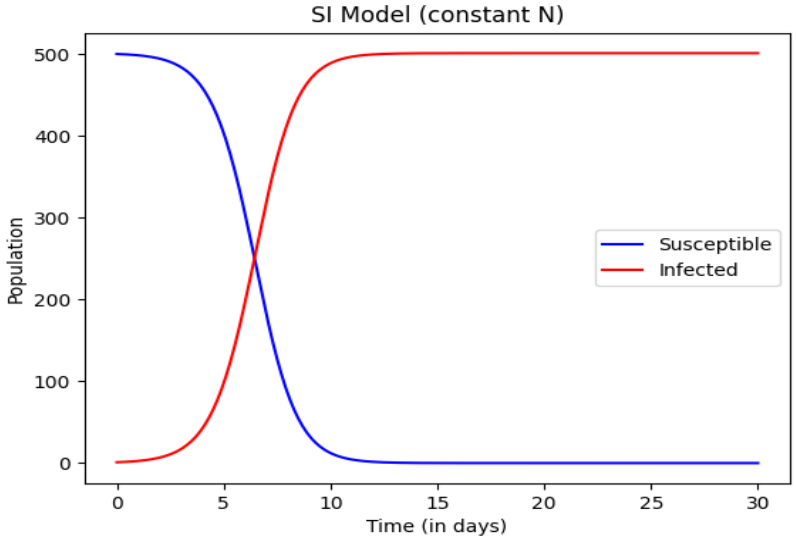

Figure 1 – SI model for populations of constant dimensions

By observing graph number 1, we see that after 6,5 days the same number of infected and susceptible people is reached and after 15 days the population is practically all infected. If we change the value of the contagion rate β, this will change the results. The higher the contagion rate, the faster the population becomes completely infected.

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For the case where there is input and output of elements, the input rates $\mu = 0{,}02$ and output $\alpha = 0.02$ and element output are added. The value of 0,02 comes from having assumed the possibility that 2% of elements of the population could enter/leave the population during the 30 days of the study.

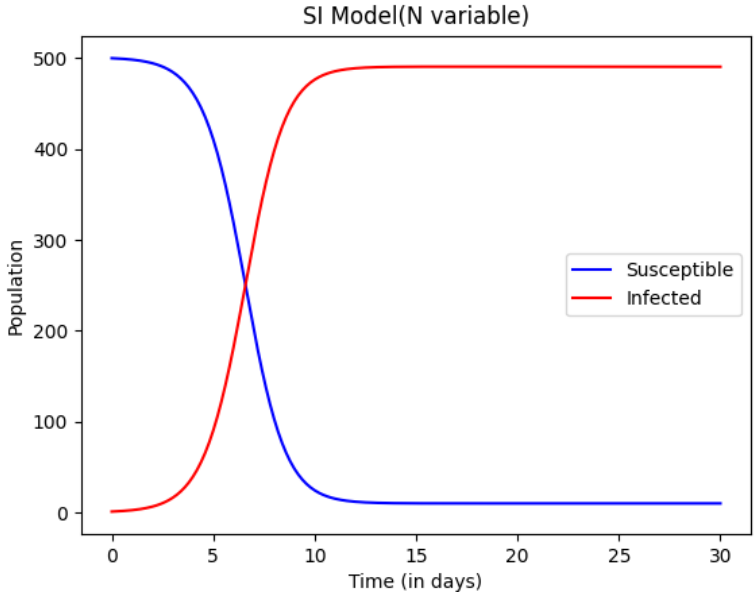

Figure 2 – SI model for populations of varying sizes

As the values of μ and α are the same, it is observed that graphs 2 and graph 1 are apparently very identical. By observing graph 2, it can also be seen that after 6,5 days the same number of infected and susceptible people is reached and at the end of 30 days the population is practically all infected, around 13 non-infected individuals. Also in this case, changing the value of the contagion rate changes the results.

SIS Model

The SIS model is the evolution of the SI, in which in this case after the individual has gone through the disease, already cured of it, he becomes susceptible again. In this case, as a recovery rate $= 0{,}25$ was added, which is the percentage of individuals who go from infected to likely to contract the disease again

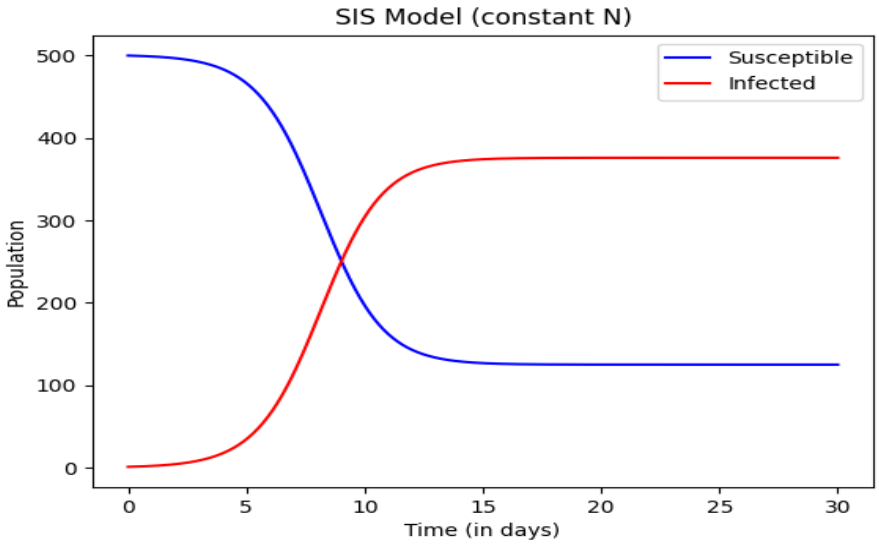

Figure 3 – SIS model for populations of fixed dimensions

By observing graph 3, it can be seen that those infected and susceptible reach the same value after 9 days, and at the end of thirty days there are more infected people (around 375) than susceptible ones (around 125).

To observe whether there are many differences between the SIS model for constant populations and the SIS model for variable populations, with inputs and outputs. To check whether there are differences, the rates of entry and exit of elements are added to the equations.

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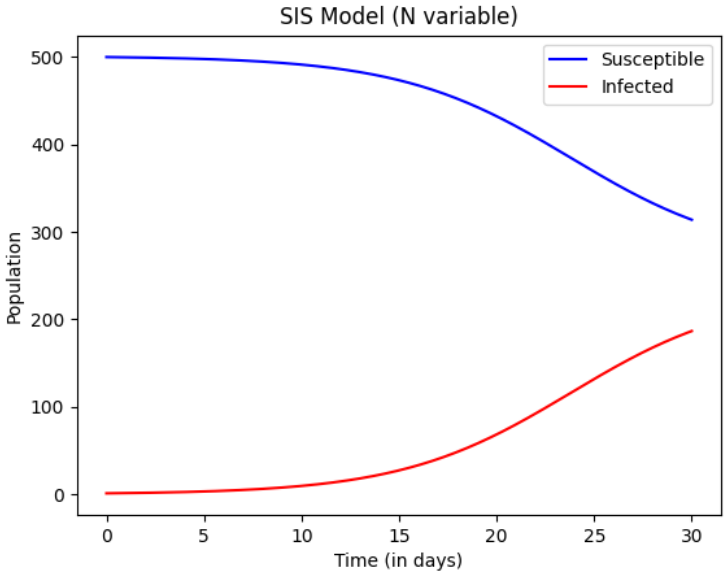

Figure 4 – SIS model for populations of varying sizes

It can be observed that under the same basic conditions, there are differences between graph 3 and graph 4, and in the latter, as there is entry and exit of elements in the population, and given the infection rate, the line of susceptible and non-infested intersect. At the end of the time (30 days) around 315 susceptible and 185 infected were observed. Unlike the graph when studying the constant population in which around 125 susceptible and 375 infected were obtained. In this case, if we lower the infection rate, the two lines end up crossing.

SIR Model

The SIR model is another model that behaves well when dealing with constant populations, that is, without entry or exit of elements in the population as can be seen in graph 5. For the simulation in addition to basic variables such as number of days , the number of individuals and the infection rate, the recovery rate $\gamma = 0{,}25$ was added, for which the same reasoning as the SIS model was followed.

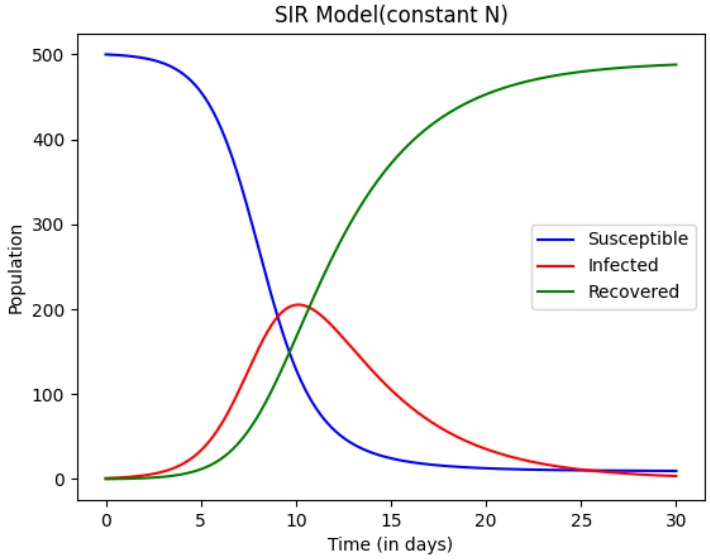

Figure 5 – SI model for populations of fixed dimensions

By observing graph 5, the three states can be identified: susceptible, infected and recovered. Susceptibles start at 499 individuals and decrease exponentially to around 10 at the end of 30 days. The number of infected people increases until the tenth day with around 200 infected individuals and decreasing after this number of days until reaching a value of around 5 individuals at 30 days. On the other hand, those recovered grow from zero on day zero to around 485 individuals on the 30th day.

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When we consider a variable population, it is normal to achieve greater variability than in the case of populations with constant numbers, given their nature. By simulating equations 6, using model conditions, graph 6 is obtained.

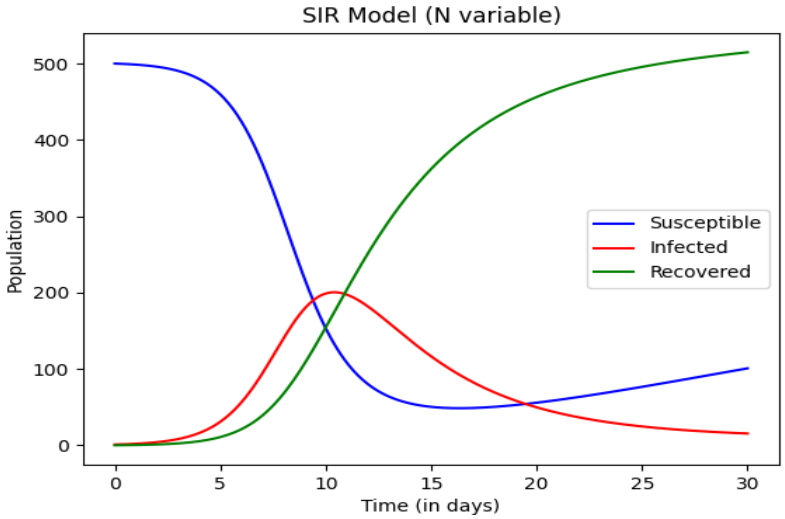

Figure 6 – SIR model for populations of varying sizes

In this graphical representation, differences can be observed in relation to the model with a population of fixed size, with the most significant line of recovered individuals exceeding 500 (515) individuals in the base population, while the lines of infected and susceptible individuals do not reach zero at the end of 30 days. It can also be observed that the number of susceptible people ends up growing (100) and only those infected reach a value above zero (15). As can be seen, this model needed more time to stabilize.

IV. CONCLUSION

According to Allen [13], diseases caused by a virus or bacteria are not modeled directly at the population level, only indirectly through the number of infected individuals. The disease states can be: susceptible, infected and recovered. In the SI and SIS models, only information about those infected and susceptible is treated, while in the SIR model, those who have recovered are added.

After analyzing the simulation of the different models, it is clear that as the complexity of the model increases, there is greater variability in the results. Using the same values for rates, population and infected people at the beginning ended up showing these differences.

During the simulations, care was taken to test with other rate values and it was observed that in the case of the SI and SIS models, there is greater variability than in the case of the SIR.

Although all the models described here are indicated for constant populations, it can be observed that only in the case of the SI model was there no variation, which shows that it must be taken into account in the case of the SIS and SIR models for this issue.

Although in some models the level of infected people even decreases, this does not mean, according to Muller and Kuttler [14] that infectious agents become weaker during an epidemic during the temporal evolution of the observed data. Evolution has to do with the rates involved in the mathematical model that explains the development of pathology.

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