Mathematical Modeling of Epidemic Diseases; A Case Study of the COVID-19 Coronavirus

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***Abstract*— The outbreak of the coronavirus COVID-19 has taken the lives of several thousands worldwide and locked-out many countries and regions, with yet unpredictable global consequences. In this research we study the epidemic patterns of this virus based on public available data, from a mathematical modeling perspective. The study is based on endemic extensions of the *susceptible-infected-recovered (SIR)* family of compartmental models, and the objective is to provide researchers a better understanding of spreading patterns of such diseases. It is discussed how social measures such as regional lock-downs and global public health vigilance, influence the model parameters, which can eventually change the mortality rates and active contaminated cases over time, in the real world. As with all mathematical models, the predictive ability of the model is limited by the accuracy of the available data and to the so-called *level of abstraction* used for modeling the problem. The Matlab source codes for the simulations of this study are provided online for the interested researchers.**

I. Introduction

Since the outbreak of the coronavirus COVID-19 in January 2020, the virus has affected most countries and taken the lives of several thousands of people worldwide. By March 2020, the World Health Organization (WHO) declared the situation a pandemic, the first of its kind in our generation and many countries and regions have been locked-down to stop the virus propagation. From a strategic and healthcare management perspective, the propagation pattern of the disease and the prediction of its spread over time is of great importance.

The problem can be studied using mathematical epidemiology and biological systems modeling techniques. In this study, the epidemic pattern of the virus is studied by using an extension of the *susceptible-exposed-infected-recovered (SEIR)* model, which is a mathematical compartmental model based on the average behavior of the population under study. The objective of this study is to provide researchers a better understanding of the significance of mathematical modeling for epidemic diseases. It is shown by simulation, how social measures such as regional lock-downs and global public health vigilance, can influence the model parameters, which in turns change the mortality rates and active contaminated cases over time.

It should be highlighted that mathematical models applied to real-world systems (social or biological) are only valid under their assumptions and hypothesis. Therefore, this research—and similar ones— that address epidemic patterns, do not convey direct clinical information and dangers for the public, but should rather be used by healthcare strategists for better planning and decision making. Hence, the study of this work is only recommended for researchers familiar with the strength points and limitations of mathematical modeling of biological systems. The Matlab codes required for reproducing the results of this research are also online available in the Git repository of the *open-source electrophysiological toolbox (OSET)* [1].

In Section II, a brief introduction to mathematical modeling of biological systems is presented, to highlight the scope of the present study and to open perspectives for the interested researchers, who may be less familiar with the context. The proposed model for the outspread of the coronavirus is presented in Section III. The report ends with some concluding remarks and future perspectives.

II. An introduction to compartmental modeling & mathematical epidemiology

A. Mathematical modeling

A model is an entity that resembles a system or object in certain aspects, but is easier to work with as compared to the original system. Models are used for 1) identification and better understanding of systems, 2) simulation of a system's behavior, 3) prediction of its future behavior, and ultimately 4) system control. Apparently, from item 1 to 4, the problem becomes more difficult and although the ultimate objective is to harness or control a system, this objective is not necessarily achievable. While modeling is the first and most important step in this path, it is highly challenging and nontrivial. The various issues that one faces in this regard, include:

- Models are *not unique* and different models can co-exist for a single system.
- A model is only *a slice of reality* and all models have a *scope*, outside of which, they are invalid.
- Modeling can be done in different *levels of abstraction*, which corresponds to the level of simplification and the specific aspects of the system that are considered by the model.

Example 1. The response of global stock markets with numerous economic, political, industrial, social and psychological factors, to a high impact news can in cases be modeled with a second-order differential equation, with an impulsive over-damped behavior that reaches its steady state after a while. Or

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in biology, the response of the body— with more that thirty-seven trillion cells— to medication can in many cases be well-modeled with a first order differential equation.

While various types of models are used for biological systems, we are commonly interested in mathematical models, as they permit the prediction and possible control of biological systems. In choosing among different available models, the widely accepted principle is the model *parsimony*, which simply means that *"the model should be as simple as possible and as complex as necessary!"*

The model parsimony, is also an important factor for estimating the unknown model parameters using real data. A more accurate model with fewer number of parameters is evidently preferred over a less accurate and more complex model. But how should one select between a more accurate complex model and a less accurate simpler one? Measures such as the Akaike information criterion (AIC), the Bayesian information criterion (BIC) and the minimum description length (MDL), address the balance between the number of observations and the model unknown parameters to select between competing models with variable number of parameters and different levels of accuracy. Finally, the physical interpretability of the model parameters and the ability to estimate the parameters such that the model matches real-world data, is what makes the whole modeling framework meaningful.

B. Compartmental modeling

Differential (difference) equations arise in many modeling problems. The major application of these equations is when the rate of change of a variable is related to other variables, as it is so in most physical and biological systems. Many powerful mathematical tools exist for the analysis and (numerical) solution of models based on differential equations. Despite their vast applications, differential equations are difficult to conceive and interpret without visualization. In this context, compartmental models are used as a visual means of representing differential equations of dynamic systems. A compartment is an abstract entity representing the quantity of interest (volume, number, density, etc.). Depending on the level of abstraction, each of the variables of interest (equivalent to system states in dynamic systems) are represented by a single compartment, conceptually represented by a box. Each compartment is assumed to be internally *homogeneous*, which implies that all entities assumed inside the compartment are indistinguishable. For example, depending on the model complexity selected for modeling a certain epidemic disease, men and women at risk can be assumed to conform a single compartment, or may alternatively be considered as different compartments. A similar partitioning may be considered for different age groups, ethnicities, countries, etc., at a cost of a more complex (less parsimonious) model with additional states and parameters to be identified. Apparently, the available real-world data may be insufficient for the parameter identification of a more detailed (complex) model.

The compartments interact with one another through a set of rate equations, visually represented by arrows between the compartments. Therefore, compartmental models can be converted to a set of first order linear or nonlinear equations (and vice versa), by writing the net flow into a compartment. Compartmental modeling is also known as *mass transport* [2], or *mass action* [3], in other contexts. More technically, a compartmental model is a weighted directed graph representation of a dynamic system. From this perspective, for an n compartment system, the compartment variables can be considered as state variables denoted in vector form as $\mathbf{s}(t) = [s_1(t), \ldots, s_n(t)]^T$. Each compartment corresponds to a node of the graph and the linking arrows are the graph edges. The compartmental model provides a graphical representation of the state-space model:

$$\begin{aligned} \dot{\mathbf{s}}(t) &= \mathbf{A}(t)\mathbf{s}(t) + \mathbf{B}(t)\mathbf{u}(t) \\ \mathbf{x}(t) &= \mathbf{C}(t)\mathbf{s}(t) + \mathbf{v}(t) \end{aligned} \tag{1}$$

where $\mathbf{A}(t)$ is state dynamics matrix corresponding to the compartmental model graph (which can be possibly time and state dependent), $\mathbf{B}(t)$ maps the deterministic or stochastic external system inputs $\mathbf{u}(t) = [u_1(t), \ldots, u_p(t)]^T$ to the compartment variables, $\mathbf{x}(t) = [x_1(t), \ldots, x_m(t)]^T$ are the observable variables of the model considered as outputs (the measurements), and $\mathbf{v}(t) = [v_1(t), \ldots, v_m(t)]^T$ are the measurement inaccuracies (considered as observation noise). Researchers familiar with estimation theory, have already guessed that the state-space form of (1), implies that one may eventually be able to estimate and predict the compartment variables from noisy measurements, using state-space estimation techniques, such as the Kalman filter.

With this background, the basic steps of compartmental modeling are:

1) Identifying the quantities of interest as distinct compartments and selecting a variable for each quantity as a function of time. These variables are the *state variables* of the resulting *state-space equations*.
2) Linking the compartments with arrows indicating the *rate* of quantity flow from each compartment to another (visually denoted over the arrows connecting the compartments).
3) Writing the corresponding first-order linear or nonlinear differential equations of the model.
4) Setting initial conditions and solving the system of equations (either analytically or numerically), which is in the form of a first-order state-space model.

A compartmental model is *linear* (*nonlinear*), when its rate flow factors are independent (dependent) of the state variables. A compartmental model is *time-invariant* (*time-variant*), when its rate flow factors are independent (dependent) of time. Compartmental models may be *open* or *closed*. In closed systems, the quantities are only passed between the compartments, while in open systems the quantities may flow into or out of the whole system. In a closed compartmental model, the sum of all the differential equations of the system is zero (for all t).

Example 2. A three compartment model corresponding to the following set of equations is shown in Fig. 1.

$$\begin{aligned} \dot{x}(t) &= \lambda - \gamma x(t)^2 - \alpha x(t) \\ \dot{y}(t) &= \alpha x(t) - \beta y(t) \\ \dot{z}(t) &= \gamma x(t)^2 + \beta y(t) - \rho z(t) \end{aligned} \tag{2}$$

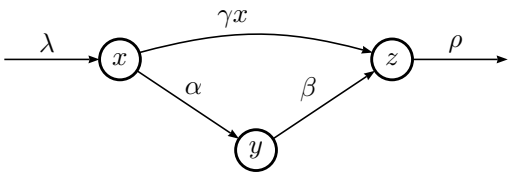


Fig. 1. A sample compartmental model corresponding to the set of dynamic equations in (2)

which can be put in the matrix form of (1). Due to the state-dependency of the rate flow between x and z, the model is nonlinear. It is also an open system, since the sum of rate changes is non-zero, i.e., there is net flow in and out of the whole system (due to λ and ρ).

C. Mathematical epidemiology

In order to model the propagation of epidemic diseases in a population, certain disease- and population-specific assumptions are required. The most common assumptions in this context include:

- The disease is contagious and transfers via contact
- The disease may or may not be mortal
- There may be births during the period of study, and the birth may (or may not) be congenitally transferred from the mother to the baby.
- The disease can have an exposure period, during which the contaminants carry and spread the disease, but do not have visible symptoms.
- Catching the disease may or may not result in short-term or long-term immunity. Depending on the case, the recovered patients can again become susceptible to the disease.
- Items such as medication and vaccination can change the pattern of propagation.

Let us consider an example, which is the basic model that we later extend for the COVIC-19 virus propagation pattern.

1) The susceptible-infected-recovered model: A basic model used for modeling epidemic diseases without lifetime immunity is known as the *susceptible-infected-recovered (SIR)* model. In this model, the total population of N individuals exposed to an epidemic disease at each time instant t is divided into three groups (each represented by a compartment): the susceptible group ratio denoted by $s(t)$, the infected group ratio denoted by $i(t)$, and the recovered group ratio denoted by $r(t)$. Accordingly, the system is closed and we have

$$s(t) + i(t) + r(t) = 1 \tag{3}$$

Note that the compartment variables are in fact the ratio of each group's population divided by N. A compartmental model for the propagation of the disease is shown in Fig. 2.

The compartmental representation of Fig. 2 is equivalent to

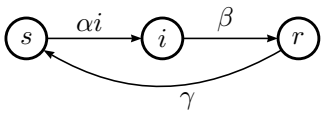


Fig. 2. The basic susceptible-infected-recovered (SIR) model

the following set of differential equations:

$$\begin{aligned}
\frac{ds(t)}{dt} &= -\alpha s(t)i(t) + \gamma r(t) \\
\frac{di(t)}{dt} &= \alpha s(t)i(t) - \beta i(t) \\
\frac{dr(t)}{dt} &= \beta i(t) - \gamma r(t)
\end{aligned} \tag{4}$$

Accordingly, moving from the susceptible group to the infected group takes place at a rate that is proportional to the population of the infected and susceptible groups, with parameter α. At the same time, infected individuals are assumed to recover at a constant rate of β. Finally, considering that the disease is not assumed to result in lifetime immunity of the subjects, the recovered individuals again return to the susceptible group at a fixed rate of γ. From (4), it is evident that

$$\frac{ds(t)}{dt} + \frac{di(t)}{dt} + \frac{dr(t)}{dt} = 0 \tag{5}$$

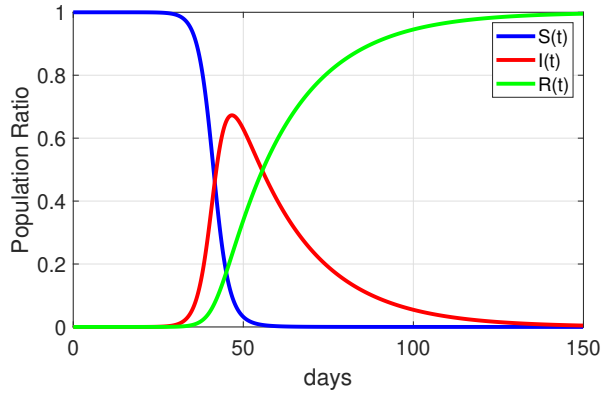
which is in accordance with (3) and the fact that the system is assumed to be closed (no births or deaths have been assumed).

Assuming initial conditions for each group, the set of nonlinear equations (4) can be (numerically) solved to find the evolution of the population of each compartment over time. The numerical solution of a basic (non-endemic) SIR model is shown in Fig. 3, with and without lifetime immunity. The time-step for numerical discretizing of the differential equations of this simulation has been chosen to be Δ=0.1 of a day. Notice how the outbreak of a disease that does not cause lifetime immunity (such as a typical flu), can result in a constant rate of illness throughout time, after its transient period. For widespread epidemic diseases, the healthcare strategists are interested in the slopes of $s(t)$, $i(t)$ and $r(t)$, rather than the total number of infected individuals (as it is currently the case for the COVID-19 coronovirus). The prolongation of the disease spread provides the better management of healthcare resources (hospitalization, medication, healthcare personnel, etc).

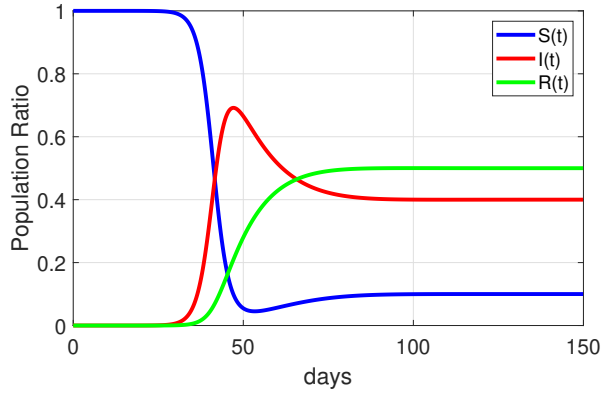
For further reference, it is interesting to study the fixed-point of the SIR model (where $\dot{s}(t) = \dot{i}(t) = \dot{r}(t) = 0$). Equating the left sides of (4) with zero, it can be algebraically shown that if $\alpha, \gamma \neq 0$ (the non-immunizing case), the SIR model has only two fixed-points:

$$\begin{aligned}
(s^*(t), i^*(t), r^*(t)) &= (1, 0, 0) \\
(s^*(t), i^*(t), r^*(t)) &= (\frac{\beta}{\alpha}, I_0, \frac{\beta}{\gamma} I_0)
\end{aligned} \tag{6}$$

where $I_0 \triangleq \dfrac{\gamma(\alpha - \beta)}{\alpha(\gamma + \beta)}$. The first fixed-point corresponds to the lack of any infected cases, and the second corresponds to a



(a) Basic SIR with immunity



(b) Basic SIR without immunity

Fig. 3. Simulation of a basic non-endemic SIR model with α=0.5 and β=0.05 in two cases: a) γ=0.0 (lifetime immunity) and b) γ=0.04

persistent disease in the population, as illustrated in Fig. 3(b). This situation is only reachable if $\beta < \alpha$, i.e., when the infection rate is greater than the recovery rate.

We can also verify whether or not the fixed-points are stable. Various methods can be used for this purpose. Perhaps, the most tangible approach is based on *perturbation theory*. Simply stated, one can add small perturbations to the fixed-points of the system and check whether or not the perturbations are compensated by the system's dynamics by pushing the state vector back to its fixed-point. Accordingly, the first fixed-point in (6) can be perturbed to:

$$(s(t), i(t), r(t)) = (1-\epsilon, \epsilon, 0) \tag{7}$$

where $0 < \epsilon \ll 1$ is the small perturbation (equivalent to a single case of disease outbreak). Now replacing the perturbed point in (4) and neglecting second the higher order terms containing ϵ, we obtain:

$$\begin{aligned} \frac{ds(t)}{dt} &= -\alpha(1-\epsilon)\epsilon \approx -\alpha\epsilon < 0 \\ \frac{di(t)}{dt} &= \alpha(1-\epsilon)\epsilon - \beta\epsilon \approx (\alpha-\beta)\epsilon \\ \frac{dr(t)}{dt} &= \beta\epsilon > 0 \end{aligned} \tag{8}$$

As a result, the first fixed point is unstable, since due to the sign of the derivatives of the perturbed system, the system's dynamics drives the state vector away from the fixed-point (since the population of the susceptible group has a negative derivative). However, depending on whether $\alpha > \beta$ or not, the outbreak may or may not result in an increase in the infected population. Simply put, if the infection rate is greater than the recovery rate ($\alpha > \beta$) the disease would lead into an outspread; but if the recovery rate is faster than the infection rate ($\alpha < \beta$) the percentage of the infected population will remain close to zero. In either case, for a non-endemic non-immunizing disease, all individuals that become infected recover after a while and move to the recovered group and again go back to the susceptible group at a rate of γ.

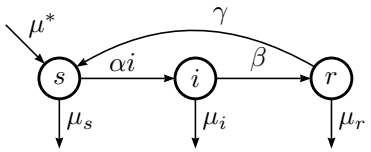


Fig. 4. The susceptible-infected-recovered (SIR) model with birth and death rates

Perturbing the second fixed-point results in

$$\begin{aligned} \frac{ds(t)}{dt} &= -\alpha(\frac{\beta}{\alpha} - \epsilon)(I_0 + \epsilon) + \beta I_0 \approx \epsilon(\alpha I_0 - \beta) \\ \frac{di(t)}{dt} &= \alpha(\frac{\beta}{\alpha} - \epsilon)(I_0 + \epsilon) - \beta I_0 \approx -\epsilon(\alpha I_0 - \beta) \\ \frac{dr(t)}{dt} &= \beta(I_0 + \epsilon) - \beta(I_0 + \epsilon) = 0 \end{aligned} \tag{9}$$

In this case, depending on whether $(\alpha I_0 - \beta) > 0$ or not, the fixed-point may be stable or unstable.

2) The endemic SIR model: An endemic version of the SIR model with rates of birth μ^* and with different death rates from the susceptible, infected and recovered groups is shown in Fig. 4. This system is no longer closed and its state equations can be written as follows:

$$\begin{aligned} \frac{ds(t)}{dt} &= \gamma r(t) - \alpha s(t) i(t) - \mu_s s(t) + \mu^* \\ \frac{di(t)}{dt} &= \alpha s(t) i(t) - \beta i(t) - \mu_i i(t) \\ \frac{dr(t)}{dt} &= \beta i(t) - \gamma r(t) - \mu_r r(t) \end{aligned} \tag{10}$$

With this background, in the sequel, we propose a compartmental model for the modeling of the COVID-19 coronavirus epidemiology.

III. A COMPARTMENTAL MODEL FOR THE CORONAVIRUS EPIDEMIC DISEASE

A. The proposed model

Many infectious diseases are characterized by an incubation period between *exposure* and the outbreak of *clinical symptoms*. Subjects exposed to the infection are much more dangerous for the public as compared to the subjects showing clinical symptoms and the longer the incubation rate. A well-known case is the HIV virus in its *clinical latency* stage. Another example is the recent COVID-19 coronovirus that is

believed to have a two-week clinical latency. For this reason, an additional compartment is added between the susceptibility and infection stages of the SIR model, which accounts for the exposed subjects. Moreover, since we are also interested in minimizing the mortality rate of the disease, a termination compartment is dedicated to the passed-away population. The variables of the model are therefore:

1) $s(t)$: The susceptible population ratio (the number of individuals in danger of being infected, divided by the total population).
2) $e(t)$: The exposed population ratio (the number of individuals exposed to the virus but without having symptoms, divided by the total population).
3) $i(t)$: The infected population ratio (the number of infected individuals with symptoms, divided by the total population).
4) $r(t)$: The recovered population ratio (the number of recovered individuals, divided by the total population).
5) $p(t)$: The number of individuals that pass away due to the disease, divided by the total population).

The model parameters are:

1) α_i: The contagion factor between the infected and susceptible populations (which is related to the contagiousness of the virus and social factors such as personal hygiene, population density and level of human interactions)
2) α_e: The contagion factor between the exposed and susceptible populations. This parameter can even be one or more orders of magnitude greater than α_i, since in ordinary conditions, people may rarely avoid contact with a symptom-less individual.
3) γ: The rate of returning from the recovered group to the susceptible group, for the cases that the subject does not obtain lifetime immunity after recovery. This parameter is the inverse of the immunity rate of the virus.
4) κ: The rate at which symptoms appear in exposed cases (resulting in transition from the exposed to the infected population).
5) β: The recovery rate of the infected cases.
6) ρ: The recovery rate of the exposed cases (the cases that are exposed, but recover without any symptoms).
7) μ: The mortality rate of the infected cases.

The proposed model and its compartmental representation are shown in equations (11) and Fig. 5. As in the classical SIR model, the interpretation of the nonlinear terms including $s(t)e(t)$ and $s(t)i(t)$ is that the rate of exposure to the virus is proportional the population of both the susceptible and exposed/infected subjects.

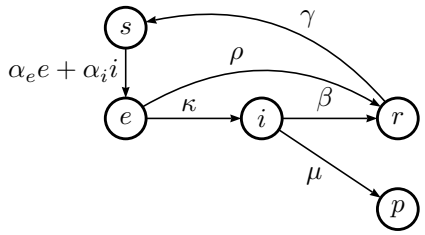


Fig. 5. The endemic susceptible-exposed-infected-recovered (SEIR) model for coronavirus modeling

$$\begin{aligned}
\frac{ds(t)}{dt} &= -\alpha_e s(t)e(t) - \alpha_i s(t)i(t) + \gamma r(t) \\
\frac{de(t)}{dt} &= \alpha_e s(t)e(t) + \alpha_i s(t)i(t) - \kappa e(t) - \rho e(t) \\
\frac{di(t)}{dt} &= \kappa e(t) - \beta i(t) - \mu i(t) \\
\frac{dr(t)}{dt} &= \beta i(t) + \rho e(t) - \gamma r(t) \\
\frac{dp(t)}{dt} &= \mu i(t)
\end{aligned} \tag{11}$$

Note that the simplifying assumptions behind the proposed model are:

1) Natural birth and natural deaths have been neglected. Therefore, other parameters leading to changes in the population are not considered. Neglecting the birth rate is also supported by the current findings that babies are not susceptible to this virus and to the best of our knowledge, no congenital transmissions of the virus from mothers to fetuses have been reported.
2) In the current study, we do not distinguish between male and female subjects; although the current global toll of the virus suggests that men have been more vulnerable to the virus than women.
3) Age ranges have not been considered; although we known that higher aged subjects are more vulnerable to the virus.
4) Moreover, in this primary version, we have not yet considered the effect of factors such as: lockdowns and quarantines, changes in social vigilance and personal hygiene, and possibility of vaccination.
5) Geopolitical factors such as distance, country borders and continental differences have also been ignored. But considering that different countries have adopted customized counter measures against the virus spread, the model parameters should be logically fitted over country-level data.

B. Constant susceptible population assumption (low percentage of infection)

A simplifying assumption that helps the analysis of the model during its rising phase, is that the mortality rate is orders of magnitude smaller than the total population (at least in its primary stages of propagation). For instance, while a death toll of 10,000 cases is indeed significant for any country; but for a country with 10 million people, the death toll is only

0.1% of the total population. Therefore, the analysis can be simplified by assuming that in preliminary phases of the virus spread, $s(t) \approx 1$ regardless of the other parameters of the model. Under this assumption, (11) is simplified to the linear set of equations:

$$\begin{bmatrix} \frac{de(t)}{dt} \\ \frac{di(t)}{dt} \\ \frac{dr(t)}{dt} \\ \frac{dp(t)}{dt} \end{bmatrix} = \begin{bmatrix} (\alpha_e - \kappa - \rho) & \alpha_i & 0 & 0 \\ \kappa & -(\beta+\mu) & 0 & 0 \\ \rho & \beta & -\gamma & 0 \\ 0 & \mu & 0 & 0 \end{bmatrix} \begin{bmatrix} e(t) \\ i(t) \\ r(t) \\ p(t) \end{bmatrix} \tag{12}$$

Defining $\mathbf{x}(t) = (e(t), i(t), r(t), p(t))^T$, (12) can be written in matrix form:

$$\frac{d}{dt}\mathbf{x}(t) = \mathbf{A}\mathbf{x}(t), \quad \mathbf{x}(0) = (e_0, 0, 0, 0) \tag{13}$$

where $\mathbf{A}$ is the 4×4 state evolution matrix on the right hand side of (12). The characteristic function of this linear system is:

$$|\lambda\mathbf{I} - \mathbf{A}| = \lambda(\lambda_+\gamma)(\lambda^2 + \delta\lambda - \kappa\alpha_i) = 0 \tag{14}$$

where $\delta \triangleq (\beta + \mu + \kappa + \rho - \alpha_e)$. Therefore the system's eigenvalues are:

$$\begin{aligned} \lambda_1 &= 0 & \lambda_2 &= -\gamma \\ \lambda_3 &= \frac{-\delta + \sqrt{\delta^2 + 4\kappa\alpha_i}}{2} & \lambda_4 &= \frac{-\delta - \sqrt{\delta^2 + 4\kappa\alpha_i}}{2} \end{aligned} \tag{15}$$

which are all real valued. Moreover, among the four eigenvalues in (15), *one and only one* of the two eigenvalues λ_3 and λ_4 is positive. Therefore, the general form of the solutions of the compartmental variables are summations of exponential terms with the above exponential rates. Specifically, for the infected and passed-away populations we have:

$$i(t) = a + be^{-\gamma t} + ce^{\lambda_3 t} + be^{\lambda_4 t} \tag{16}$$

and the number of infected cases increases exponentially at a rate defined by one of the two parameters λ_3 or λ_4 (whichever is positive). We therefore arrive at the following practical conclusion: with fixed system parameters, the infection rate rises exponentially up to a point at which the linear approximation does no longer hold. This practically translates into:

Result 1. *Without changing the contact patterns in the population, the exponential increase in the number of infected subjects continues to a point where a significant percentage of the population is infected.*

Considering that the death toll $p(t)$ is composed of the same exponential terms as the infected cases in (16), the result is indeed disturbing.

C. Infection peaks in time

The peaks of the infectious group is important from the strategic viewpoint. These points correspond to local or global extremums of $i(t)$, which mathematically correspond to where $\frac{di(t)}{dt} = 0$ in (11), i.e., where $i(t) = \frac{\kappa}{\beta+\mu}e(t)$. It can be shown that this leads to a reduced order set of nonlinear dynamic equations, which can be numerically solved for the remaining variables (all except $i(t)$). The simulations demonstrated in the sequel, show that the infected population can have multiple local peaks over time, with recurrent behaviors, proving that

Result 2. *The epidemic disease can repeat pseudo-periodically over time (in later seasons or years) and turn into a persistent disease in the long term.*

This behavior has been observed in previous pandemics, such as the 1918 influenza pandemic known as the Spanish flu, where three pandemic waves of infection have been observed within an interval of a few months (cf. https://en.wikipedia.org/wiki/Spanish_flu).

D. Fixed-point analysis

As with the basic SIR model presented in Example II-C.1, the fixed-point(s) of the model can be sought by letting the left hand sides of (11) equal to zero. Apparently, the only fixed-point is the no disease case ($i(t) = e(t) = 0$):

$$(s^*(t), e^*(t), i^*(t), r^*(t), p^*(t)) = (1 - p_0, 0, 0, 0, p_0) \tag{17}$$

where $0 \leq p_0 \leq 1$ is the steady-state total death ratio. The stability of this fixed-point can again be addressed by perturbing the fixed point as follows

$$(s(t), e(t), i(t), r(t), p(t)) = (1 - p_0 - \epsilon, \epsilon, 0, 0, p_0) \tag{18}$$

Putting these points in the state dynamics (11), we find:

$$\begin{aligned} \frac{ds(t)}{dt} &= -\alpha_e(1 - p_0 - \epsilon)\epsilon \approx -\alpha_e(1 - p_0)\epsilon < 0 \\ \frac{de(t)}{dt} &= \alpha_e(1 - p_0 - \epsilon)\epsilon - \kappa\epsilon - \rho\epsilon \approx (\alpha_e - \alpha_e p_0 - \kappa - \rho)\epsilon \\ \frac{di(t)}{dt} &= \kappa\epsilon > 0 \\ \frac{dr(t)}{dt} &= \rho\epsilon > 0 \\ \frac{dp(t)}{dt} &= 0 \end{aligned} \tag{19}$$

which is unstable, i.e., the system's dynamics drives it away from the fixed-point resulting in further exposed and infected cases. Therefore:

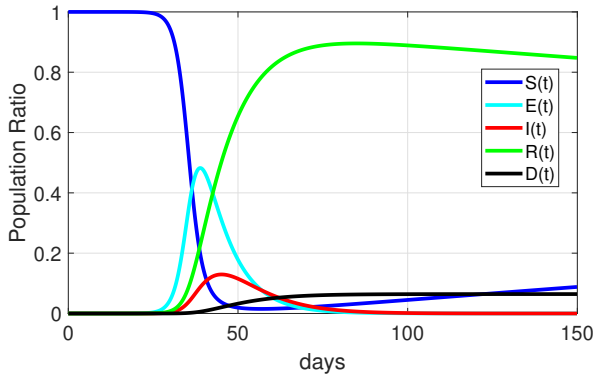
Result 3. *Without intervention in social contact patterns, the non-immunizing endemic SEIR model is unstable and recurrent, with the smallest infection outbreaks.*

Due to the nonlinearity of the model, an analytical study of the system's behavior is highly dependent on the system's parameters. In order to obtain further results we can either add realistic assumptions leading to marginal cases, or we can perform numerical simulations.

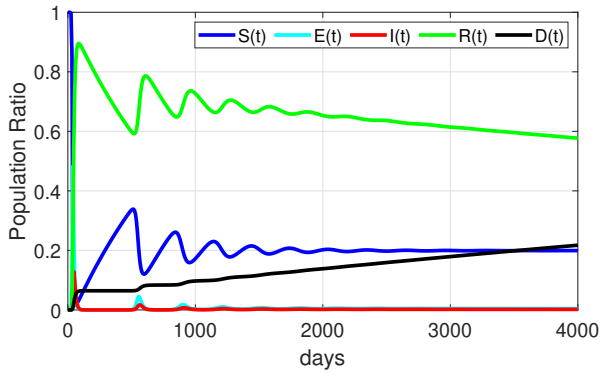
E. Simulations

Important Note: In the current version of the report, the parameters of the simulated results have not been adapted to real-world data of the COVID-19 coronavirus. Therefore, the quantitative infection and death rates reported in the figures of this section are only for studying the model behavior and may not be associated to real-world scenarios.

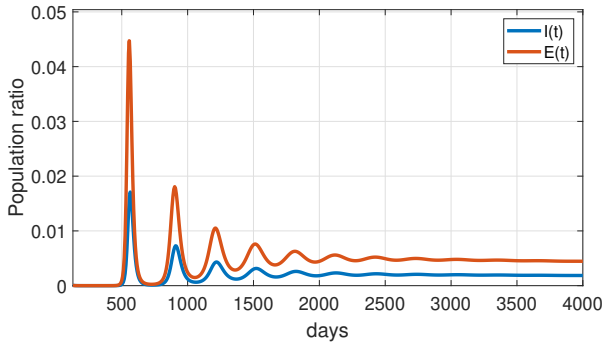
We have carried-out several simulations with different set of parameters, resulting in very different scenarios, illustrated in Fig. 6. It is interesting to see that in one of the scenarios, the model has repetitive patterns over time, similar to the aforementioned Spanish flu.



(a) Scenario A in five months

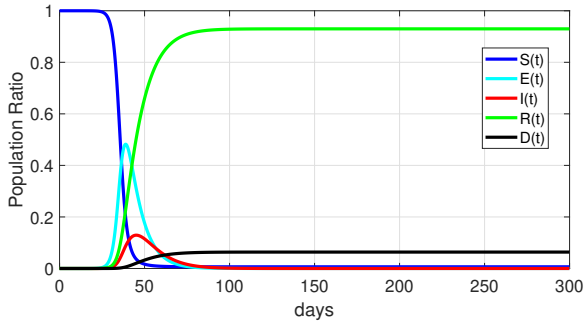


(b) Scenario A in ten years



(c) Scenario A in ten years ($I(t)$ and $E(t)$)

Fig. 6. Simulation of the endemic SEIR model with parameters α_e = 0.65, α_i = 0.005, κ = 0.05, ρ = 0.08, β = 0.1, μ = 0.02, γ = 0.001, a) in one year, b) and c) in ten years



(a) Scenario B

Fig. 7. Simulation of the endemic SEIR model with parameters α_e = 0.65, α_i = 0.005, κ = 0.05, ρ = 0.08, β = 0.1, μ = 0.02, γ = 0, over one year

IV. Model Fitting on the Coronovirus Data of Different Countries

TO BE ADDED

V. Conclusion and Future Work

The research will be continued and extended from various aspects in the future versions. Specifically, by fitting the model over real data, the extension of the assumptions in Section III to more realistic ones, the prediction of infection and mortality rates under quarantine and vaccination conditions, and the study of the recurrent pattern of the epidemic disease over time are of primary interest.

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