ΔΙΑΤΜΗΜΑΤΙΚΟ ΠΡΟΓΡΑΜΜΑ ΜΕΤΑΠΤΥΧΙΑΚΩΝ ΣΠΟΥΔΩΝ στα ΔΙΚΤΥΑ και ΠΟΛΥΠΛΟΚΟΤΗΤΑ
ΤΜΗΜΑ ΟΙΚΟΝΟΜΙΚΩΝ ΕΠΙΣΤΗΜΩΝ
ΤΜΗΜΑ ΜΑΘΗΜΑΤΙΚΩΝ
ΤΜΗΜΑ ΒΙΟΛΟΓΙΑΣ
ΤΜΗΜΑ ΓΕΩΛΟΓΙΑΣ
ΑΡΙΣΤΟΤΕΛΕΙΟ ΠΑΝΕΠΙΣΤΗΜΙΟ ΘΕΣΣΑΛΟΝΙΚΗΣ

INTER-FACULTY MASTER PROGRAM on NETWORKS and COMPLEXITY
SCHOOL of ECONOMICS
SCHOOL of MATHEMATICS
SCHOOL of BIOLOGY
SCHOOL of GEOLOGY
ARISTOTLE UNIVERSITY of THESSALONIKI

Master Thesis

A novel approach to the spreading of SARS-COV2 with the use of networks.

Vasileios Vachtsevanos

Supervisor: *Meletlidou Efthymia, Assistant Professor, AUTH*

Thessaloniki, August ,2021

ΔΙΑΤΜΗΜΑΤΙΚΟ ΠΡΟΓΡΑΜΜΑ ΜΕΤΑΠΤΥΧΙΑΚΩΝ ΣΠΟΥΔΩΝ στα ΔΙΚΤΥΑ και ΠΟΛΥΠΛΟΚΟΤΗΤΑ
INTER-FACULTY MASTER PROGRAM on NETWORKS and COMPLEXITY

ΤΜΗΜΑ ΟΙΚΟΝΟΜΙΚΩΝ ΕΠΙΣΤΗΜΩΝ SCHOOL of ECONOMICS
ΤΜΗΜΑ ΜΑΘΗΜΑΤΙΚΩΝ SCHOOL of MATHEMATICS
ΤΜΗΜΑ ΒΙΟΛΟΓΙΑΣ SCHOOL of BIOLOGY
ΤΜΗΜΑ ΓΕΩΛΟΓΙΑΣ SCHOOL of GEOLOGY
ΑΡΙΣΤΟΤΕΛΕΙΟ ΠΑΝΕΠΙΣΤΗΜΙΟ ΘΕΣΣΑΛΟΝΙΚΗΣ ARISTOTLE UNIVERSITY of THESSALONIKI

□□□□π□□□□□□□ □□□□□□□□

Μια πρωτότυπη προσέγγιση στην εξάπλωση του SARS-COV2 με τη χρήση δικτύων

Βασίλειος Βαχτσεβάνος

Επιβλέπουσα : *Μελετλίδου Ευθυμία, Αναπληρώτρια Καθηγήτρια, Α.Π.Θ*

*Τριμελής Επιτροπή (Εγκρίθηκε στις *Ημερομηνία*):*

Ε. Μελετλίδου	*Σ.Σγαρδέλης*	*Χ.Πολάτογλου*
Αν.Καθηγήτρια, Α.Π.Θ	*Καθηγητής, Α.Π.Θ*	*Καθηγητής, Α.Π.Θ*

Θεσσαλονίκη, Αύγουστος, 2021

Βασίλειος Βαχτσεβάνος,

Πτυχιούχος Φυσικός Α.Π.Θ

Abstract

The COVID-19 epidemic has troubled both medicinal and STEM personnel for more than one year. This epidemic has proven most resilient to medicinal and social measures undertaken worldwide. Trying to model this epidemic is an arduous task, both because of the virus' mutability and due to the fact that each government trying different measures with a varying degree of success. In this work, we will try to achieve two things. The first one is to create an "umbrella" dynamic system, which can be adjusted to make predictions for all cases, while the second is to transfer this model to a mesoscopic equivalent, in order to make predictions that fit the specificities of smaller populations more accurately. Finally, there will be presented a novel idea for examining whether or not an epidemiological system is closed or open, i.e., the epidemic will end or not.

Key Words

COVID-19, Epidemiology, Epidemiological Networks, Dynamical Systems, Complexity

Σύνοψη

Η επιδημία COVID-19 έχει προβληματίσει ιατρικό και επιστημονικό προσωπικό για παραπάνω από ένα έτος . Αυτή η επιδημία αποδείχθηκε πολύ ανθεκτική στα ιατρικά και κοινωνικά μέρα πρόληψης που έχουν ληφθεί διεθνώς. Η προσπάθεια να γίνει μοντελοποίηση της επιδημίας είναι ένας απαιτητικός άθλος, τόσο λόγω της γρήγορης τάσης ιού να μεταλλάσεται αλλά και του γεγονότος ότι κάθε κυβέρνηση παγκοσμίως έχει λάβει διαφορετικά μέτρα , με διαφορετικό βαθμό επιτυχίας. Στη παρούσα διατριβή, θα γίνει μια απόπειρα να επιτευχθούν δυο στόχοι. Ο πρώτος είναι να σχεδιαστεί ένα δυναμικό σύστημα «πασπαρτού», το οποίο μπορεί να προσαρμοστεί για εκάστοτε περίπτωση, ενώ ο δεύτερος είναι η μεταφορά του μοντέλου στο μεσοσκοπικό ανάλογο του, ώστε να γίνονται πιο ακριβείς προβλέψεις για περιπτώσεις μικρών πλυθησμών βάση των ιδιαιτεροτήτων τους . Τέλος, θα παρουσιαστεί μια νεα ιδέα, η οποία μας επιτρέπει να εξετάζουμε εαν ένα επιδημιολογικό σύστημα είναι ανοιχτό ή κλειστό. Αυτό σημαίνει αν προβλέπεται λήξη μιας επιδημίας ή όχι

Λέξεις Κλειδιά

COVID-19, Επιδημιολογία, Επιδημιολογικά Δίκτυα, Δυναμικά Συστήματα, Πολυπλοκότητα

Contents

Summary in Greek (Περίληψη)

Στη παρούσα εργασία θα επιχειρήσουμε να μοντελοποιήσουμε την επιδημία COVID-19 που έχει τεράστιο κόστος σε ανθρώπινες ζωές , από το Δεκέμβρη του 2019 εώς και σήμερα (Αύγουστος 2021). Με τη χρήση στοιχειώδων γνώσεων θεωρίας δικτύων, δυναμικών συστημάτων και στατιστικής θα προσπαθήσουμε να μοντελοποιήσουμε την επιδημία με τη χρήση εργαλείων που μέχρι πρόσφατα δεν είχαν εφαρμοστεί με το τρόπο που θα παρουσιάσουμε ή δεν είχαν εφαρμοστεί καθόλου.

Το πρώτο κεφάλαιο μας προσφέρει μια συνοπτική ιστορική διαδρομή μέσα από την επιδημιολογία, τόσο τη μαθηματικοποιημένη και φορμαλισμένη εκδοχή της , αλλά και την ερευνητική ιατρική σκοπιά. Εξοικειωνόμαστε με τις τεχνικές της επιδημιολογίας και το τρόπο που πραγματοποιούνται έρευνες πάνω στο αντικείμενο.

Το δευτερο κεφάλαιο είναι μια συμπυκνωμένη ανάλυση των βασικ΄ν ενοιών των δυναμικών συστημάτων και των ιδιοτήτων τους που θα μας απασχολήσουν κατά την υπόλοιπη εργασία.

Το τρίτο κεφάλαιο είναι ομοίως με το δεύτερο, μια συμπυκνωμένη εισαγωγή στα δίκτυα και περιγράφει κάποια δίκτυα που θα χρειαστούμε αργότερα στη μελέτη μας.

Το τέταρτο κεφάλαιο είναι μια συνοπτική περιγραφική αναφορά στην έννοια της εντροπίας και κάποιες χρήσεις της, για να διαχωρίσει την εντροπία που ήδη μελετάτε από το τρόπου θα την εφαρμόσουμε εμείς.

Το πέμπτο κεφάλαιο είναι μια εις βάθος ανάλυση της μεθοδολογίας μας . Πως σχεδιάσαμε το σύστημα βασιζόμενοι μονάχα στις βασικές μαθηματικές αρχές και σταδιακά το εμπλουτίσαμε με τη χρήση υπάρχωντων γνώσεων στο αντικείμενο, πραγματικών δεδομένων αλλά και με προτωτυπες και ασυνήθιστες λύσεις στα προβλήματα που εμφανίστηκαν. Ολο το πρώτο μέρος του κεφαλαίου ασχολείται αποκλειστικά με τη διαδικασία αυτή, την παρουσίαση των ευρυμλατων κάθε μεθόδου και αξιολόγηση τους. Τέλος αναφέρονται τρόποι βελτίωσης του ίδιου του μοντέλου, μιας και η επιδημιολογία είναι δυναμική και πρέπει να προσαρμόζεται στο περιβάλλον.

Το δέυτερο μέρος του πέμπτου κεφαλαίου είναι η εφαρμογή του δυναμικού συστήματος σε 3 διαφορετικά αρχετυπικά δίκτυα. Συγηρίνονται τα αποτελέσματα αυτά με τα αποτλέσματα ενός toy model του τελικού μοντέλου από το 5.1. και αξιολογούνται τα αποτελέσματα. Πρατηρούμε πως το μέγεθος του δικτύου παίζει καθοριστικό ρόλο στη μελέτη, μιας και η μελέτη με τη χρήση προσομοιώσεων έχει διαφορετικούς χρόνους ολοκλήρωσης. Στο τέλος του αναλύουμε μερικούς λόγους που οδηγολύν στην απόκλιση των αποτελεσμάτων.

Το τρίτο και τελευταίο μέρος του πέμπτου κεφαλαίου είναι αρχικά μια συζήτηση για την χρήση διαγνωστικών μεθόδων ου μας επιτρέπουν να βρούμε εαν ένα επιδημιολογικό σύστημα θα οδηγηθεί σε μια κατάσταση ηρεμίας ή όχι, καθώς επίσης πως ο ρυθμός μόλυνσης είναι αναξιόπιστος για συστήμα περίπλοκα όπως το δικό μας. Προτείνουμε μια νεα χρήση της εντροπίας ως διαγνωστικό εργαλείο για το ευρύτερο σύνολο των επιδημιολογικών μοντέλων, μιας και εξαρτάται μόνο από τα αποτελέσματα της λύσης του συστήματος (ή δικτύου) που έχουμε αναλύσει. Αυτή η χρήση της εντροπίας μπορεί να βοηθήσει να βρεθεί αν υπάρχει κάποιο τέλος στην επιδημία ή όχι.

Στο τέλος της εργασίας υπάρχει μια σύνοψη των αποτελεσμάτων μας, αναφορές και μερικοί από τους κώσικες μας , ώστε να μπορούν να επαναλγφθούν τα αποτελέσματα μας.

Acknowledgements

For this present thesis I would like to thank my academic guides, my supervisor Assistant Professor Meletlidou Efthymia, as well as Professor Chariton Polatoglou (who is my supervisor for another academic dissertation). Those two people have guided me from my freshman years and I hope they will continue to offer me their knowledge and help as mentors and as part of my "academic family".

I would also like to thank my parents for the support they have provided me during this hard period of the epidemic, but also all the years that have passed, culminating to this work. In the same spirit I would like to thank my grandparents for instilling me with strong values and a love for academic knowledge.

Results presented in this work have been produced using the Aristotle University of Thessaloniki (AUTh) High Performance Computing Infrastructure and Resources.

Finally, I would like to extend a special thank you, to the Director of the program, Professor Ioannis Antoniou, whose faith in me was a factor that enabled me to join this academic program and learn so much.

Purpose

The purpose of this dissertation is to design a mathematical model which has high predictive capabilities and is easily adjustable to any specific case of an epidemic similar to the COVID-19 pandemic, that currently has engulfed the world. This means that we have to create a flexible model of equations that can be adjusted accordingly.

We are also going to expand the model to a network equivalent, in order to simulate more intricate cases. The pandemic of COVID-19 has cost a lot of lives and resources to all the countries around the globe and the humanitarian efforts were never enough. Any classical predictive tool, based only on the available statistical data was not very successful at helping governments prevent the spread of the disease.

The final step of this work is to try to find a tool that will help us predict whether or not the disease reaches an end or if the virus is here to stay. This is tricky for a virus that mutates this quickly, therefore a proper tool would solve the problem.

1. History of Epidemiology

Epidemiology through the lens of mathematics can be traced back to Daniel Bernoulli and d'Alambert[1]. Bernoulli was curious about how effective the process of inoculation was, when used to fight smallpox. In order to model it properly he created a set of equations that describe the survival of an individual from a sickness. The model is one of two populations, the susceptible S and the immune I. One susceptible that lives long enough is transferred to the group I. The age of a person is one of the two variables that affect the evolution of the system. Both groups have a mortality rate $\mu(\alpha)$- where α the age of a person- that is independent of the disease that is studied, and the susceptible group has an additional parameter $\lambda(\alpha)$ that is the force of infection. The transition from susceptible to immune is based on a probability $w(\alpha)$ which is found from the equation:

$$\frac{dw}{da} = s(\alpha)\lambda(\alpha)u(\alpha) - \mu(\alpha)w \tag{1.1}$$

$u(\alpha)$ is the probability that a newborn individual is alive and susceptible at the age α.

This model was highly opposed by d'Alambert, who published a critique to Bernoulli's model before it was even officially published by the royal academy of France, in 1760. His model was quite different than Bernoulli's. He approached the problem using function analysis. Like Bernoulli he also modeled his functions based on the age of the individual. He is considering that the death rate for each disease, event or natural causes can be deduced as a different function, and with a formula similar to signal theory derives the form of the mortality rates.

However, those models are too general and complex. In the early 20th century two English colonial officers, a physician and a mathematician[2], tried to model the spreading of an infectious disease at a local population. Their work was later adapted and formalized by Kermack and McKendrick. They studied the spread of an infectious disease by separating the populace into susceptible and infected. They then expanded on their model, which like Bernoulli's and d'Alambert's , was dependent on the age of each individual . However in this model, integrating the equations we can have a set of equations that are only time dependent and the parameters (mortality rate, recovery rate, etc) are a real number.

This first model was a simple SIS model, in which a person of age α, had a possibility to become infected by the disease rampant. The individual was then transferred in the infected group from where he would either die or be cured and return to the main populace. The general mortality rate and birth rate could also be taken into account in order to be able to more accurately find the expected number of deaths caused by the disease. However , being age specific would mean that for each group, a number of subgroups equal to the age existed, which would mean that instead of a 2x2 system, we would be faced with a 2*n x 2*n system of equations. For that purpose, modern epidemiology avoids modeling age specifically and prefers the macroscopic approach.

1.1. Non Mathematical Epidemiology

1.1.1. John Snow, the father of epidemiology

In Victorian England , there was the first case of pathological and investigative research about the source and transmission of a disease[3]. During that era, London was plagued by cholera outbreaks. Some prominent physicians of that time supported the theory that "miasma" – a form of bad air was behind the epidemic. During this period, the germ theory of disease had not yet been developed, therefore a new way to study the source of this disease should be found. With the help of local authorities, John Snow managed to map the household inflicted by the disease and time stamp them. He then found out that the common link between the houses was the fresh water source, that they shared, in the form of a public water pump. Using statistics and appropriate sampling techniques, John Snow made it clear that water quality and cholera were connected.

The idea of mapping and tracing the spread of an epidemic disease (whether viral or not) was at a time an innovation, one that influenced later scientists to incorporate the mathematical methods proposed by the previously mentioned scientists.

1.1.2. Variables

When managing data of epidemics, some variables appear to play important roles on the way a disease is spread. Those factors can be biological like sex, genetics, other infections or conditions etc, or environmental, like habits, nutrition, climate etc. Those factors are important when we try to identify and understand the way a virus works. Unfortunately, those factors cannot be easily inserted into our generalized mathematical models. They increase the complexity of the systems. Unlike age, they are usually binary, if one person belong to category A or B (high or low risk), and instead of creating a two variables system, they are better represented by splitting a dimension (ie group of people in the competitive population model) into 2 other subspaces. In the case of networks, they add more information that must be dealt and makes any process more complicated, increasing both mathematical and computational complexity.

When trying to map a disease or the characteristics of the germ [4]behind it , those variables are extremely useful and vital. Physicians and health scientists not only require those information , but actively seek them. This is clear distinction between natural scientists and health scientists when approaching an epidemic.

1.2. Types of epidemiological research

There are 5 prevalent types of epidemiological research[5] :

Table 1 : Epidemiological research methods

Name	Characteristics
Case series and population case series	The researcher counts cases and relate to population data in order to produce rates and find patterns.
Cross-sectional	The researcher studies the state of the population's health and disease rate at a defined place and time and then measures the burden of disease.
Case–control	The researcher compares the data in a series of cases and a control group.
Cohort	The researcher is trying to relate information on risk factor patterns and health states.
Trial	The researcher intervenes with some measure designed to improve health, then collects data to see the effect.

Our research is a cross-sectional one, as we only use data from one country and try to model the behaviour of the virus.

One important factor we must take into considerations is researcher bias. Selective sampling, biased interpretation of the data are some of the ways the researcher might be mislead into a biased result and erroneously downgrade or emphasize some aspects of the epidemic.

1.3. Infection Rate

An infection rate [6] is the probability or risk of an infection spreading out of control in a population. It is used to measure the frequency with which new instances of the infection appear within a selected population during the time period under study. This number is equal to:

$$(1.1)\ R = ct\frac{I}{N}$$

Where ct is a constant, I is the number of infected people and N the total population or the total number of susceptible. This formula adapts to the equations used. One common change made to the above equation is :

$$(1.2)\ R = \frac{\beta}{\rho}$$

Where β is the spreading rate and ρ is the recovery rate. This version of the infection rate calculates whether the infection spreads faster than people are healed. If this number is greater than 1, then we might be unable to control the pandemic.

For our model we will calculate the values of R for some model, since it is a stiff tool and has accuracy only for a short time.

One more important information is that the infection rate can be equivalently be described as :

$$(1.3)\ R = \frac{rate\ of\ new\ infections}{rate\ of\ people\ re-entering\ the\ main\ populace}$$

This formulation enables to calculate the effects of mortality rate as well as other external factors, like prevention measures, vaccines or quarantines.

2. Dynamical Systems

A dynamical system is a mathematical construct that allows us to study natural systems, whose state can be described by a countable set of discrete variables. Those time dependent variables are called dynamic variables. A dynamical system could then be described [7] as a map in the phase space of a point x, from time t to time t'.

If the system is free of random parameters, it is known as a deterministic system. Deterministic systems appear to have the same evolution every time. This means that for every starting point, for a time t_0, the coordinates of the point at the time t' are always the same, if we do not change the initial conditions of the system. On the contrary, a system where a parameter has random values, means that a point will not always reach the same destination after every run, even if the starting conditions are the same. Systems like this are called stochastic.

Dynamic systems can also be described by whether or not its parameters are static or time dependent. Although a system with time dependent parameters is mostly predictable, it is quite complex and hard to map. Systems that ae not explicitly dependent on time are known as autonomous.

The most common type of dynamical systems are the ones where we use differential equations to map describe the process. This allows us to study a space of N+n dimensions, where N is the number of different variables and n are the independent variables. For a dynamical system , n usually equal 1, and represents time.

The equations describing the behaviour of the system, as an interaction between the different variables and not the time, is known as a vector field. By examining the divergence of the vector field f, we can examine the behaviour of the system.

- For a divergence equal to 0, the system is area preserving (does not expand).
- For a divergence less than 0, the system is area is dissipative (the system collapses within some boundaries)
- For a divergence greater than 0, the system is explosive (expands outside towards infinity)

2.1. System Stability

When studying time evolving systems, we usually need to find whether or not an equilibrium point exists, that remains stable. This however is not always achievable. Only a small portion of systems (always autonomous, most of the times linear) can be studied in this manner. Thankfully, the polyonymic nature of most epidemiology models means that for autonomous cases a solution can be reached eventually. A solution found this way can describe the system's stability [8] [9] [10]. In order for us to find those solutions and their stability, we first assume that any derivative of the vector field has no dynamical effect in the equations ($\frac{df}{dt} = 0 => f = 0$). Then we linearize the equations and solve the system. After doing so, with use of a Jacobian matrix, we find the eigenvalues corresponding for each solution. Notice that for many systems we have to deal with an unknown number of variables n, where $n > 2$. W will use the eigenvalues to determine the stability of the system.

The stability can be :

- Stable sink (all perturbation will lead back to the point, dissipative system)
- Unstable source (all perturbation will sent the point into a trajectory away from this point, explosive system)
- Spiral Sink
- Spiral Source
- Center (all perturbation lead to closed trajectories- Metastable)
- Saddle (unstable)

2.2. Chaos

Sometimes a system is so complex that is very hard to predict its behaviour. Sometimes the trajectory a point follows is based heavily on the initial conditions of the system. Stochastic parameters or big distances between two consecutive time points are also sign of chaotic behaviour.

3. Network Theory[11]

The combination of discrete objects and their relations, represented through a matrix or graphic representation is a rough definition for a graph. When those objects (both the nodes and/or the edges) have additional information (names, conditions, weight etc) we have a network. When those information are changing through time we have a dynamic network.

The way a network is wired , ie the nodes and edges are connected, is called network topology. The density of the edges or the grand total of connected nodes can affect the properties of a network. A human society is a complex social network that is hard to properly mirror. Therefore we will present 3 different models for random network models.

1. Barabasi- Albert

Networks of this type are scale free (follow a power law when it comes to degree distribution) that use preferential attachment. This type of random network creates a number of socially "rich" and "poor" nodes.

2. Erdos- Renyi

The typical random network, where the edges are randomly determined. The number of nodes and edges per node are predetermined.

3. Watts-Strogatz

This model creates a random network that has the small world property. This property means that for the network the typical distance L between two randomly selected nodes grows proportionally to the logarithm of the number of nodes N in the network. This type of network has a short average path length and high clustering.

3.1. Epidemiology and networks

In recent times, networks are used for a plethora of sciences. Biology, sociology, physics etc. The use of networks is also prevalent in epidemiology. Networks are used in 2 ways [12]in this case. Either to map the viral mass travelling through geographic locations (cities, countries, airports, etc) or to find patient zero. The first type uses well defined networks, made with data from flights, trains and generally people using transportation crossing borders or buying tickets. The second type requires to find the interactions between a specific person and its inner circle, and then expand it up to the nth neigbhour, depending on the researcher's precaution.

Using networks that describe a society as a whole is a challenge of increased complexity, since we neither have a preferable network type to describe human societies in big scale, neither the amount of information is easily manageable.

4. Entropy

Entropy is a concept in natural and information science, that is most commonly associated with a state of disorder, randomness, system complexity, or uncertainty. Entropy can be used as property of any system and be used for studying a system's predictability, excess of energy or internal disorder.

Entropy arose from Clausius' effort to formalize the intuition of when a process is possible or not, due to energy conservation, this however meant that entropy as not a well-defined concept. Entropy's ability to describe transitions as possible or not, and find the preferable processes led to entropy being used outside thermodynamical systems or even be extended mathematically.

The first well defined statistical entropy (ie a statistical property of a system) was defined by Boltzmann , through the use of combinatronics. This model was expanded by Gibbs and from that time it is mostly unchanged.[1]

Entropy is extensively used in other fields of physics and in information science, as a way to calculate the complexity created by the information within a system. There are many types of entropy used for that purpose, all stemming from Shannon's [13][14] entropy for studying information transferred from a message.

Entropy is a malleable term, therefore many times it is used interdisciplinary[15] to describe other properties, outside thermodynamics and information theory.

[1] There exist some generalized versions of entropy, from which the Boltzmann -Gibbs model arises, like Tsallis entropy, but it is of no concern to our current work.

5. Methodology

5.1. Modelling the epidemic using dynamical systems

Modeling the epidemic is not an easy task. The process that we will demonstrate starts from the initial stages of more simplistic approaches to our extended model. The process was arduous and time consuming, especially when we consider the fact that the epidemic's characteristics were not fully understood until the 12-month threshold. Mirroring closely this lack of knowledge, our model adapts to the environmental conditions and new information per version of it.

5.1.1. Basic Models (SIR/SEIR/SEIRS)

Based on the basic theory and mathematics presented in the previous chapters, the process of modeling an epidemic using those tools will be described below. The first assumption was that we have a SEIR model where a patient that falls ill cannot be infected again. The SEIR model was also the preferred model used at the start of the pandemic [16] [17] [18]. The carriers either fall sick or have a little to no symptoms. This would mean that we either have to create 3 groups or assume that the "carriers" group $e[t]$ includes those who get over the disease without any obvious symptom. The simple solution would be to avoid using more groups, as it increases the dimensions of the system increasing its complexity.

The system will create is not linear, however all its constituents are of first degree power only, based on systems that show bifurcations.[19][20]

The first step was determining the value of some parameters. The SEIR model used was the following:

(5.1)
$$s' = P - m\,s - T\frac{b_1\,e + b_2\,i}{s+e+i+r}\,s[t]$$
$$e' = -(m+\sigma+k_1)e + T\frac{b_1\,e + b_2\,i}{s+e+i+r}\,s$$
$$i' = \sigma e - (m+a)i - k_2 i$$
$$r' = k_1 e' + k_2 i - m\,r$$

Where:

P: is the population increase rate per day.

m: is the mortality rate based on effects non related to the epidemic.

T: is a dampening effect, which we will see in detail later.

b_1 & b_2: is the spreading rate of the infection, we theorize that for infected and carriers do not have the same parameter, but they can be assigned an equal value, This will be examined later in more depth.

σ: is the rate that carrier falls sick.

k_1 & k_2: is the of inverse of approximate time a person is a carrier or sick.

a : is the mortality rate of the epidemic .

Here we used two methods to calculate the values of parameters, the first was for localized values, like spreading rate and mortality rate, where we estimated the value based on statistical data [21] [22] [23], while the second was bibliographical.

The following table shows the initially assigned value for each parameter:

Table 2: SEIR model parameter values

Parameter	**value**	**source**
P	0.00002925	Hellenic Statistical Authority
m	0.00003047	Hellenic Statistical Authority
T	0.01	Estimation based on NPHO[2] advertisements
σ	0.03333	Estimation based on Worldmeters Data
b_1	66.6667	Estimation based on NPHO data
b_2	66.6667	Estimation based on NPHO data
k_1	0.333333	Based on other coronaviruses epidemics (SARS, MERS)[24] [25] [26]
k_2	0.99	Estimation based on NPHO data
a	0.01	Estimation based on NPHO data

For this simulation we used as initial condition the total population of Greece and we considered that there is only one infected person at the start of the epidemic.

Now a comparison between estimated infected cases and data will be assessed.

[2] National Public Health Organization (Greece)

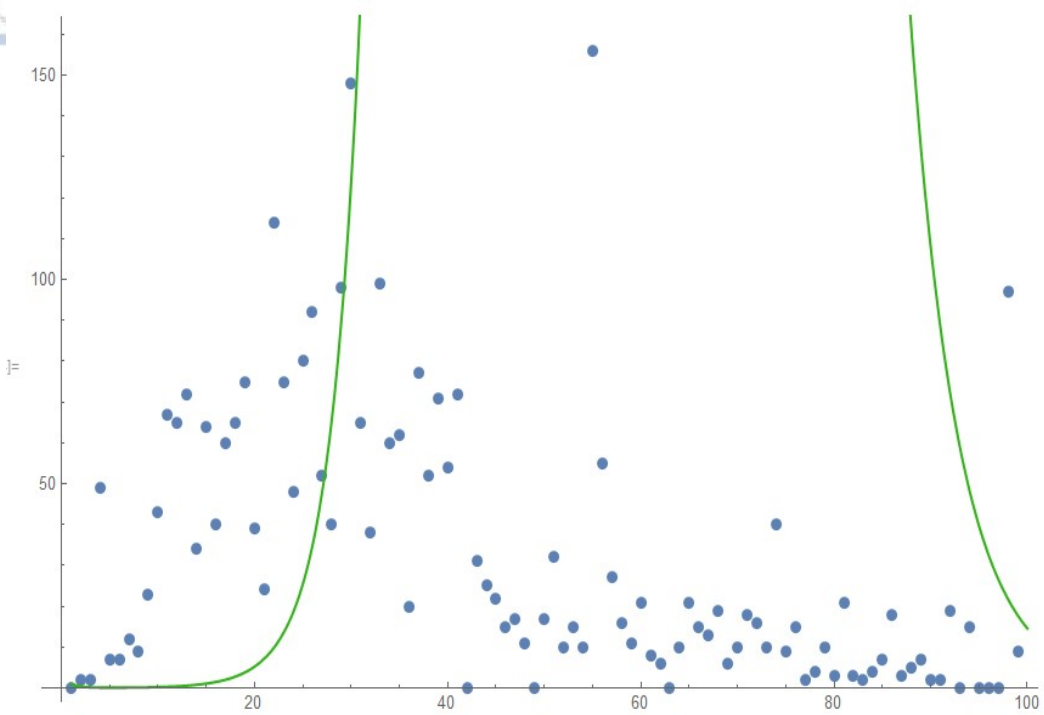

Figure 1 : Comparison for data and initial SEIR model

We can immediately recon that the duration of the epidemic and the magnitude is quite obviously in disagreement with the actual data. This calls for improvements on the initial model.

One information that changed the model's structure was the revelation that people who have undergone the disease can fall sick again. This led to changing the model to SEIR-S type of model, where there is a "loop", resupplying the populace with susceptible people.

One important parameter was the dampening effect T. Initially the T parameter was aimed to describe the reduced effectiveness of the virus and would affect the infection rate R. However, no matter the value of R resulting from the simple SEIR model, it appeared that T was not completely describing the effectiveness of the measures undertaken.

This led into two options, the examination of other models where quarantine was implemented or the modelling of a time dependent T.

For the time dependent dampening parameter T, a number of different models we used, harmonic equations, reverse gaussians or step functions. The results were unfortunately not very realistic and this lead to the implementation of quarantine.

Based on work for past epidemics, most models use the following logic:

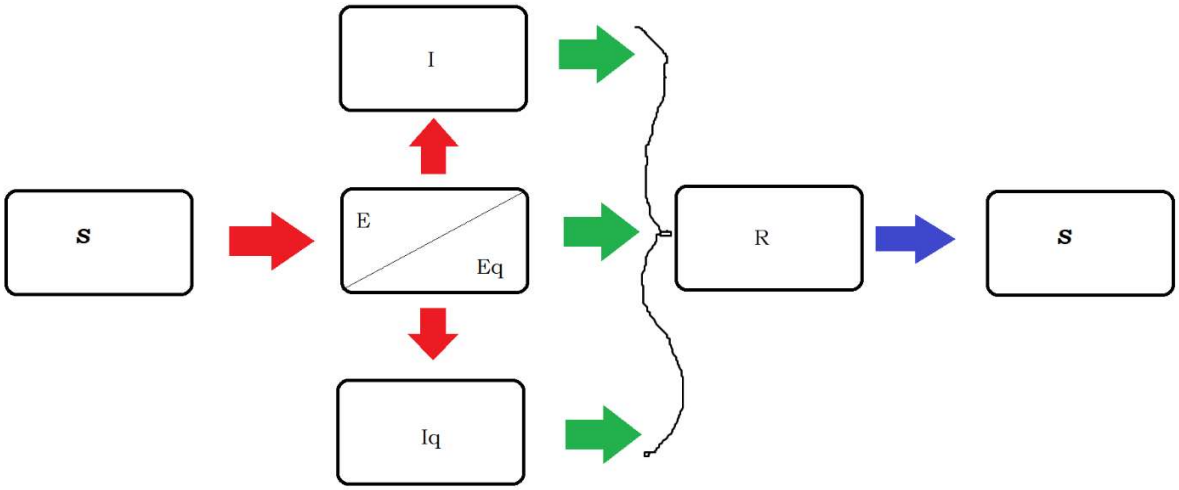

Figure 2: Classical Quarantine in SEIR models

The figure above shows a simplified version of the SEIR-S models used so far when quarantine is included. Iq and Eq represent quarantined infected and carriers. [27]It shows that we will need at least one additional dimension (usually two, since E and Eq are not the same group but are closely related[28], since a carrier usually transfers from E to Eq.

This model however does not describe the social distancing measures, since a quarantine is only used on people who exhibit or are suspect of being infected. For that reason a new model was required from us.

5.1.2. Advanced Models (SEIDR/S)

The problem of properly simulating the effectiveness of the countermeasures taken by the Greek government appears to lead into an unavoidable fifth dimension (ie a new group of people) added in the system. This is a problem since:

A. It increases the complexity[29]
B. We must define its properties accurately
C. It adds internal dissonance in the system's topology, making harder for the simulation to be replicated in a micro/mesoscopic level (for examples through dynamic networks)

We went forth with the creation of a super group known as Distanced (D). This supergroup includes three subgroups, the aforementioned Iq and Eq , as well a new group, that is prevalent in the

measure taken by most countries, [30]limited interaction and mobility for susceptibles, people who are practicing social distancing (Sd).

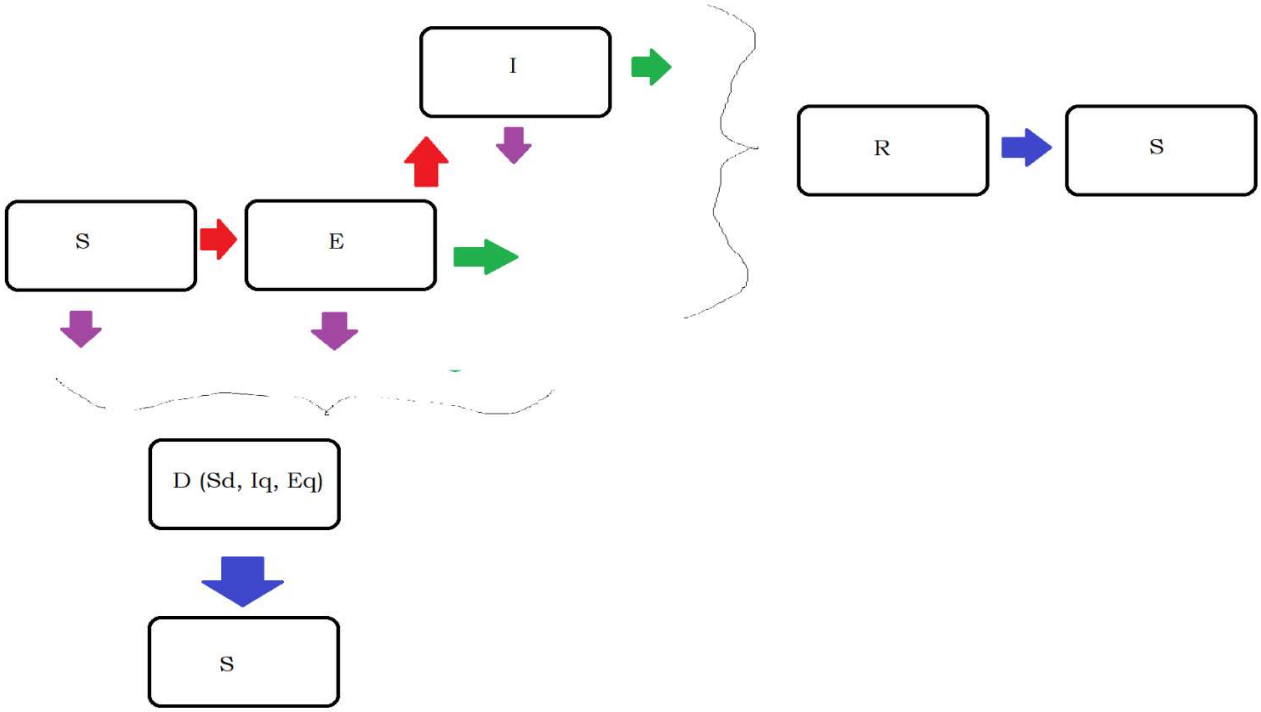

Figure 3 :SEIDR-S model logic

As we can see, the D super group absorbs people and by putting them through a process of social isolation [31]reduces their interactions to the least amount possible. For this reason, even though carriers and infected are absorbed, the mean time required for them to be reintroduced to the main population is longer than the process of recuperation, therefore the D group is not transferring people to R but instead they are sent directly back to S.

Based on the above thoughts the group D interacts as following with the other groups:

A. From S and E it randomly drains people, since for most carriers, they are usually not discovered in order to be lead to a quarantine.
B. From I it drains a big number of people who are either in hospitals or in home isolation.
C. It has no interaction with R
D. At a specific rate, people form D, are reintroduced to S

Having made those observations, we made the first models.

$$(5.2)\quad s' = P - m\,s - T\frac{b_1\,e + b_2\,i}{s+e+i+r}\,s\; - \mathrm{dis}\,s\frac{i}{s+i+e+r+d} + \mathrm{ttq}\,d$$

$$e' = -(m + \sigma + \mathrm{k}_1)\, e + T\frac{b_1\, e + b_2\, i}{s + e + i + r}\, s - \mathrm{dis}\, e\frac{i}{s + i + e + r + d}$$

$$i' = \sigma\, e - (m + a)i - \mathrm{k}_2\, i - \mathrm{qu}\, i$$

$$r' = \mathrm{k}_1\, e + \mathrm{k}_2 i - m\, r$$

$$d' = \mathrm{dis}\, \frac{(s + e)i}{s + i + e + r + d} + \mathrm{qu}\, i - \mathrm{ttq}\, d$$

This is a crude model and the parameters used are not optimized, since the effect of any process has a different result from the traditional model, due to the different effects on the population density and cohesion. The return rate from R to S is still small relative to the duration of the epidemic in Greece (~60 days since day 1) and is not taken into consideration, although it was used for modeling initially.

The new parameters are :

dis : the rate though which people are slowly removing themselves from the society, practicing social distancing.

Ttq: the rate that approximately a person returns to the society actively or has an interaction without safety.

T: here this parameter describes the safety from using masks, gloves and other measures for prevention.

qu: the rate percentage of people who are certainly infected and properly isolated.

The simulation was repeated, and the new results were still too overwhelming. For this reason, we made a comparison for approximate characteristics of the epidemic. As we could easily extrapolate, the duration of the epidemic lasts approximately ~60 days for the initial model (we are only using the data of March to June 2020, therefore a second or third wave are still speculations).

We have assigned the following values:

- σ is now equal to 1/28 which equates to a carrier having a 50% possibility per 14 days to fall ill (The cycle of the COVID-19 virus was estimated to last 10-14 days at that point, therefore we used the upper limit)
- T is now 0.025, since we have a relative 75 % chance to prevent the virus from entering our bodies if we take proper prevention measures and the infection process is reduced by 1/10 due to reduced social transport.
- dis is equal to 0.9, as we consider that the social distancing was imposed our voluntarily followed by 90% of the population in relation with the percentage of the infected.
- ttq is equal to 1/60, since the initial duration of the measures was expected to last approximately 60 days.

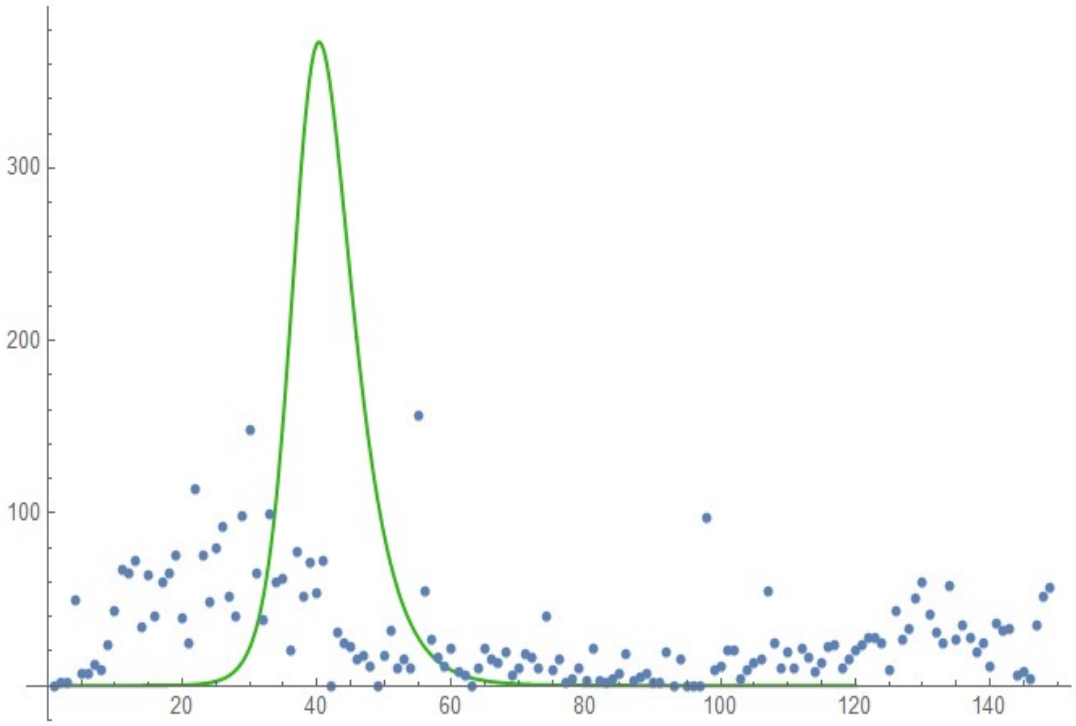

Figure 4 : Comparison of normalized number of infected with real data

We can see that there is still a temporal displacement present (~7 days), however the behaviour is much smoother, as the main body of the epidemic lasts approximately as long as the real data show (~40 days). This means that we must adapt the values assigned to the parameters, since social distancing appears to have an effect on the length of the epidemic.

After some pondering the following assumptions were made:

- b_1 and b_2 are not longer equal. Since an infected person is avoided by others, we assigned to the infected a smaller spreading rate equal to 1/5 of the one assigned to the carrier. The new values are $b_1 = 2.415$ & $b_2 = 0.483$.
- qu is increased to 0.95 .
- dis is adjusted to 1000.
- T is increased to 0.3, since the social transport is part of the distancing and not prevention and the prevention effectiveness we re-evaluated to 70%.
- σ is increased to 1/14

One more interesting fact is that we start simulations from day 10, in order to reduce the time displacement, using as initial condition the existence of 10 (based on available data) infected people instead of 1.

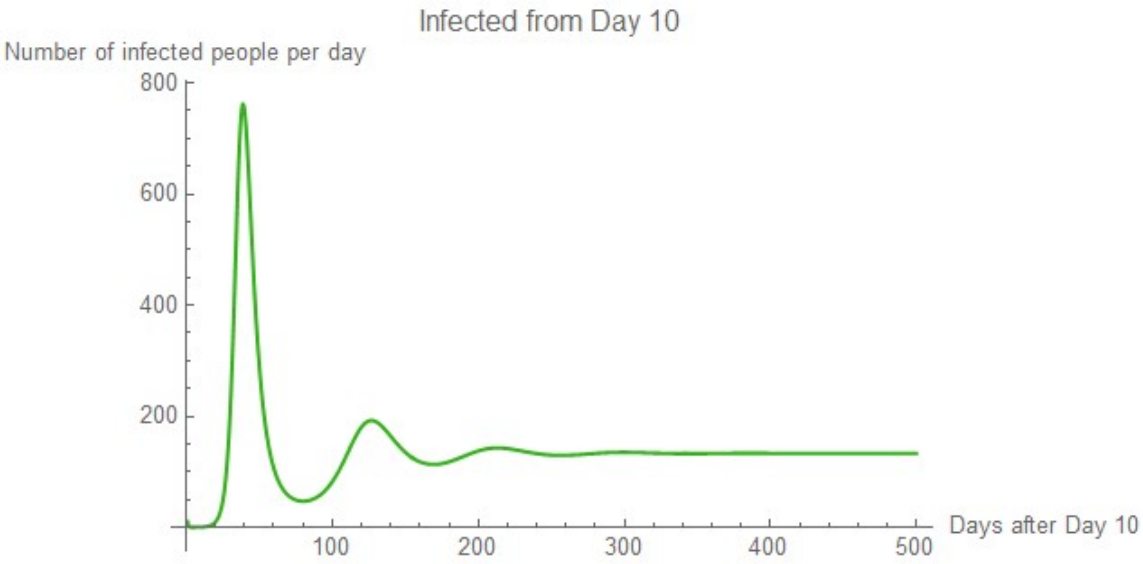

Figure 5: Expected number of infected people after improving the model

After the new simulation we notice the following characteristics:

- The number of total deaths for the duration of 6 month is ~270 which at the time was realistic.
- There is a second wave, starting at June.
- The epidemic flat lines, which is worrisome.
- The summer would include incoming tourists or returning locals from other infected countries, meaning there would be a "kick" in the system.

Taking those problems under consideration some new ideas were implemented and lead to a new model which will be described in the next part.

We should mention that when we make the move from static parameters to dynamic ones, we cannot study the stability of the system. However, the stability of the system will be seen in more depth in part 5.3. The same is true to with the infection rate derived from each version of our model. Those infection rates unfortunately have no use, since those models failed to predict the epidemic properly.

5.1.3. Dynamically evolving parameters

Most parameters were turned into functions with time and location localization. This can explain why the pandemic spreads with different rate for most countries.

The first change made was the adjustment of the virus characteristics. The rate that a carrier falls sick was adjusted. The same has happened to the social distancing factor, since it is not static and changes depending on how severely the measures were imposed. We cannot predict how long the measures are imposed, therefore we initially tried to replicate the period that has passed (start of epidemic up until September's 30th ,2020). Finally, we added 2 new functions. The influx of tourists or outside carriers in the system (tt(t)) [32]and the seasonal effectiveness of the virus (tp(t)) , since the virus appears to prefer the winter period and is less effective during summertime. This might be due to difference in population density, but we should use other countries, that are not major holiday destination in order to test this hypothesis. We have also incorporated the rate through which recovered individuals are reintroduced to the main populace, as rec.

The new model therefore is :

(5.3)

$$s' = P + 1000\,tt(t) - ms - T\,tp(t)\frac{b_1 e + b_2 i}{s+e+i+r}\,s - \mathrm{dis}\,s\frac{i}{s+i+e+r+d} + rec\,r + ttq\,d,$$

$$e' = 50\,tt(t) - (m + k_1 + \sigma(t))e + T\,tp(t)\frac{b_1 e + b_2 i}{s+e+i+r}\,s - \mathrm{dis}\,e\frac{i}{s+i+e+r+d}$$

$$i' = \sigma(t)e - (m + a)i - k_2\,i - qu\,i$$

$$r' = k_1 e + k_2\,i - mr - rec\,r$$

$$d' = \mathrm{dis}(s + e)\frac{i}{s+i+e+r+d} + qu\,i - ttq\,d$$

This new model is quite more complex and has many more details that we should properly model. In the appendix A.5. one can see the initial form of the functions. They were shaped based on the current events, severity of measures, public transportation, and internal population migration. We will see in depth the final functions; however, some intermediate forms can be visited in the Appendix.

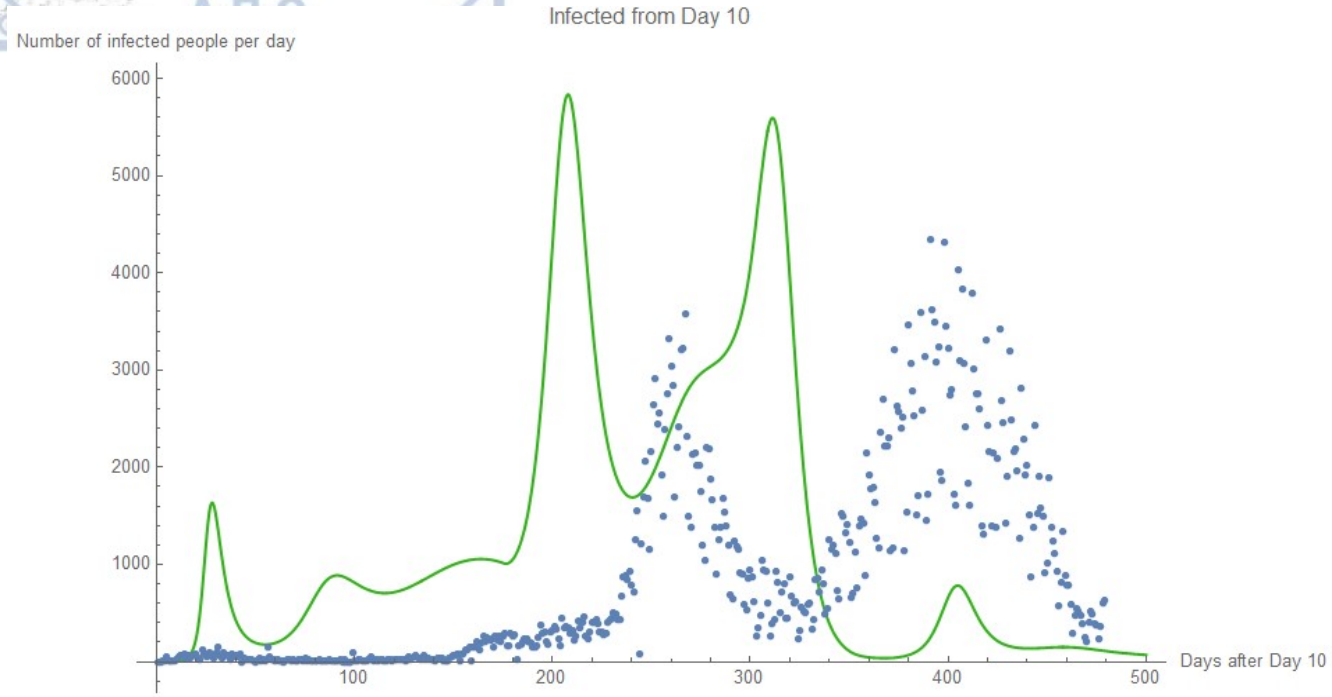

Figure 7 : First model with time dependent parameters

As we can see from the above figure, this new model looks more like the real data, but is compact. This led into the introduction of some new ideas.

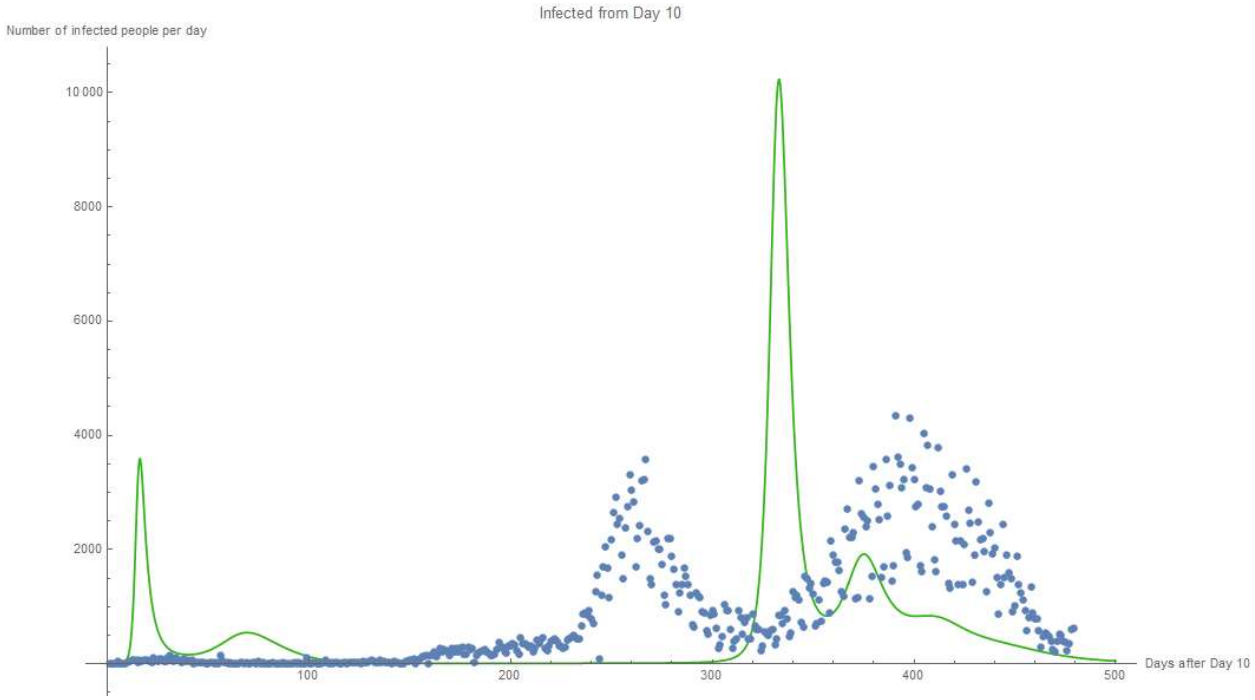

Figure 6 : Adjusted model for mortality and internal migration

For the second iteration two changes were made. The first was the change of the mortality rate into a function as well, since the most lethal strains survived during the appearance of the British and South African strains. We also tried to adjust the mutation of the virus, so that it spread more as time goes by, since the most infectious strains survived. However, there is an imbalance in the aggressiveness of the virus and the parameter function were reevaluated.

After a revaluation of those functions, we decided to add a factor describing the reduced effectiveness of the measure of social distances due to riots and civil unrest during the February of 2021.

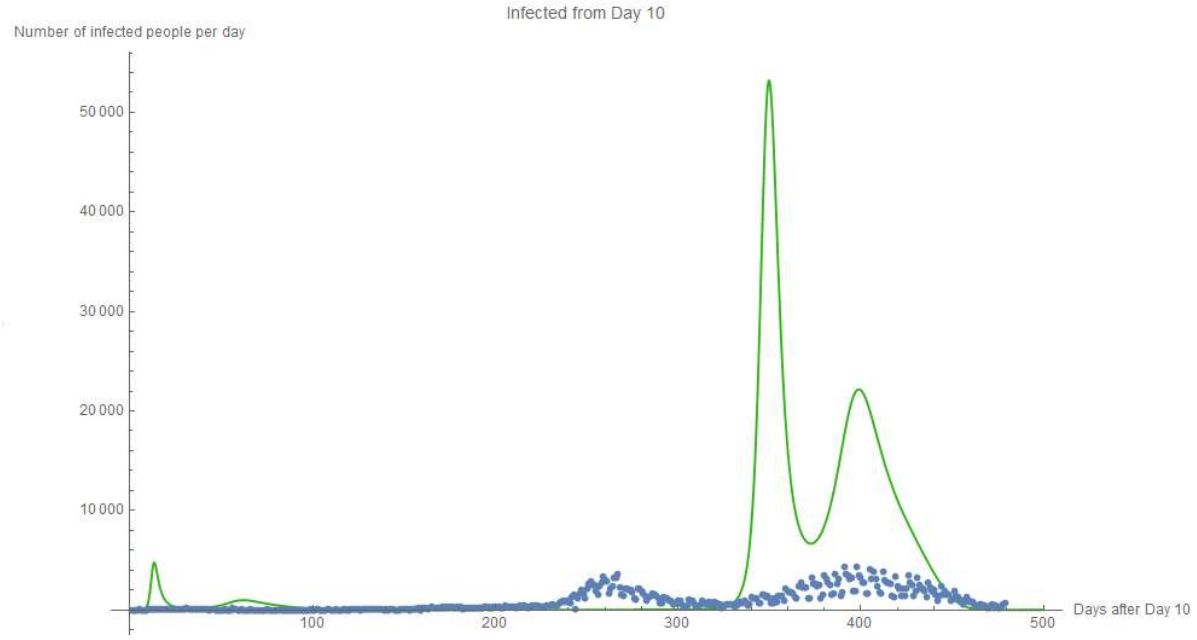

Figure 8 : Rios including model initial version

From what we can see the new social distancing parameter creates a problem with the intensity and the duration of each wave. We also have a problem with the fact that although the third wave should be during the peak of the virus's spreading rate, it is shorter. Therefore, the model was readjusted.

The final adjustments lead to the final model which will be presented later in this work.

5.1.4. Final Model

For the final model we have made one interesting change from (5.3). The rate of mutation was made a different function, which can be separately studied. Therefore, the seasonality function describes the difference in population density related to the time of the year. It is a repetitive process with a one-year period. In depth:

$$(5.4)\ atr[t] = (0.215)\left(\frac{1 + \left(\frac{t}{1825}\right)}{10}\right)$$

The mortality rate slowly increases from 2.15% and doubles within 6 years.

$$(5.5)\ ttq[t] = \left(\frac{1}{tquar}\right) + \frac{\left(Exp\left[\frac{t - 210}{60}\right]\right)}{10^9}$$

The rate that people return from the distanced group to the susceptible populace. It is initially $\frac{1}{60}$ people per day (rough estimation) and slowly increases, to show that people got tired of staying inside.

$$(5.6)\ dis[t] = 1000\left(Abs\left[Cos\left[\left(\frac{t}{900}\right) * 2 * Pi\right]\right] * \left(\frac{1}{Exp\left[\frac{t - 275}{30}\right] + 1}\right)\right)$$

The social distancing parameter function is quite a trickier one. The first part (blue) describes a reduction of measures severity during summertime while the second part (green) shows the gradual non conformity by the people, starting during the end of January 2021, right before the first riots. This however can be adjusted because it creates a small knee, as we see later.

$$(5.7)\ tt[t] = Abs[Sin[Pi * \left(\frac{t}{365}\right)]] * \left(\frac{1}{Exp\left[\frac{t - 450}{15}\right] + 1}\right)$$

The tourist and invading carriers parameter is a crude numerical estimation, which describes the rate with which new people enter the country (some may be carriers). The exponential in the last part serves as a dampening effect, in order to study the behavior of the system until the next October and not further. The issue with the dampening factor is that by this September (2021) the vaccinated group will have complete or partial immunity and be a new factor to add. Thusly any long-time prediction should include the vaccinated group.

$$(5.8)\ mt[t] = Exp\left[\frac{t}{2500}\right]$$

The mutation function is a slow (relatively) exponential process. Most biological processes follow power laws and for that reason we decided to adapt a simple version of it.

$$(5.9)\ tp[t] = mt[t] * \left(1 + Cos\left[2 * Pi * \left(\frac{t + 106}{455}\right)\right]\right)$$

The seasonality parameter includes the mutation strength and has a cosine to describe the approximately yearly changes in population density. Due to the quarantine these changes where shifted, due to people returning to their family houses for financial and personal reasons.

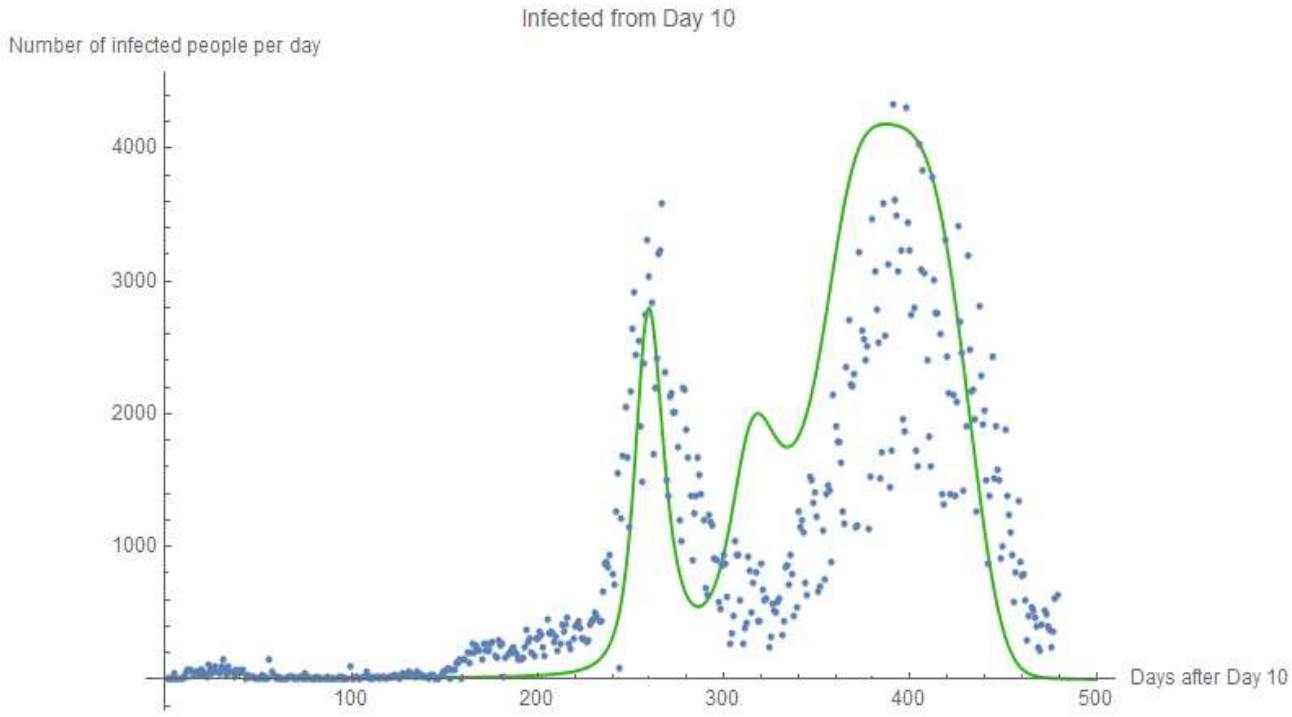

Figure 9: Final model comparison with real data

We can see that this model is quite a close fit to the actual data. There is the exception of the knee, which as we discussed was caused due to the complexity of properly including the civil unrest [3] alongside the distancing parameter.

For the initial days of the pandemic the model is quite accurate as well.

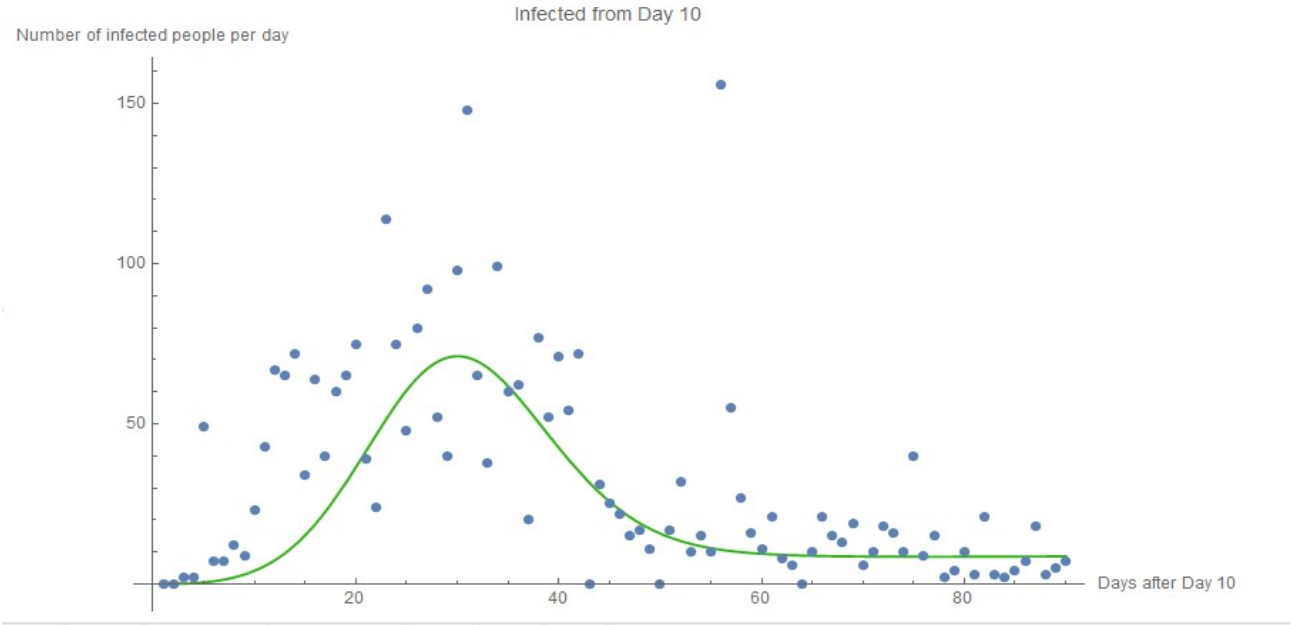

Figure 10: Final Model initial wave

As we can see the only issue is the severity of the epidemic seems undermined, however properly adjusting the exponential part of $dis[t]$, would increase the initial aggressiveness of the virus.

As a last fact we should mention that as of the 5th of July 2021 the death toll in Greece was 12716 people. Our model predicted 12496. This is a divergence of 1.7%. The model as it is (not adjusted to increased tourism of 2021, starting July and not including vaccinated) caps approximately at 12700 deaths by September, due to design limitations.

5.1.5. The Vaccinated Group and expansions on the model

The model we have presented (5.3), is quite an accurate approach to the COVID-19 epidemic. However, as the process of expounding its final form showed, there are many changes and outside factors that require special attention. Some of those include:

- Civil Unrest and rioting or protests, which work as superspreading events.

[3] This includes all acts that go against the measures. From non-compliance to rioting. This is corrective post script note, in order to ensure that this work is purely scientific and not politically coloured,

- Uncontrolled or limited control of people entering the country.
- The introduction of vaccines and new mutations.

We would like to improve our model to better simulate the above issues, however this process requires a lot of time and resources. We have made however the following observations:

A. All social unrest and disobedience can be simulated with trigonometric or harmonic functions, in order to simulate peaks and valleys.
B. Tourism influx can have a stochastic parameter to simulate random superspreading events at airports, tolls, and ports.
C. The vaccines races against mutations, therefore complete immunity is hard to come by, we can test if the vaccination can be used as a parameter function instead of an additional group, which would inevitably increase complexity of the system.

5.2. Modelling the epidemic using networks

For the next part of our work, we will compare the results from a dynamical network following the process described in our dynamical system model.

5.2.1. Model properties

For our comparison between networks and the dynamical system, we will use 3 types of random networks. The Erdos- Renyi, Basarabi -Albert and small world (Watts -Strogatz) random networks. They were all set up using commands in the igraph library in R.

We did decide to use the following characteristics:

- There is no influx of new people in the network (either births or tourists) in order to try to keep the network's topology stable.
- The network's topology should remain stable, therefore any person that is isolated, is not removed, but is inactive, i.e., does not allow viral transference through them, whether they are infected or carriers.
- The network is not dense, meaning that people interact with 3-4 other people daily. We will try to keep this as a stable and not interchangeable element.

The biggest problem in the network approach lies in the fact that people are malleable to social interactions, which means that a person interacts with many people with different frequency. This would then mean, we must create weighted networks or networks that rewire with preferences, which would increase the complexity to a level of non-computability.

For the initial comparison we chose the simplest possible structure for our networks. As we will see in the next chapter, size and network density play quite an important role when it comes to calculation time.

One last interesting note is that in our model, since everything happens in steps, we first added the death step and then the spreading. This might affect the way the network behaves, but we will talk about it more when we compare the toy models.

5.2.2. Network size and Properties

For the simulations we will make, we used networks with the sizes 100 and 1000 people. The more populous a network the more it increases the complexity therefore the time and resources required to solve it. The more the complexity increases the problem tends to a NP problem. This led to us using smaller size networks. This might be because our algorithm uses a lot of nested functions in order to keep track of different groups (Isolated, Carriers and Infected) during each step. The algorithm also makes an ego network for each carrier and slowly spreads the virus. Based on the density of the networks (low density) the time required differed for each network type depending on their density. For 50 runs of maximum 730 steps, we approximately had:

Table 3 : required time for each network type

Network Type/ Number of people	**100**	**1000**	**10000**
Erdos-Renyi	~5hours	>168 h	>168 h
Barabasi-Albert	~1 hour	~24 hours	~130 h
Watts-Strogatz	~30 min	~1 hour	~48 h

As we can see the time required for a complete simulation is extremely demanding for one core and in order for us to complete it , we should use parallel programming for networks of greater size.

The characteristics of each network are:

- Watts-Strogatz: dimensions of the starting lattice= 1, the neighborhood within which the vertices of the lattice will be connected =1 and the rewiring probability =0.1
- Erdos -Renyi: density=0.35
- Barabasi-Albert: mean number of connections=5

5.2.3. Comparison of toy models

In this section we are going to compare the 100 and 1000 people model for networks and the adjusted ones from the dynamical system.

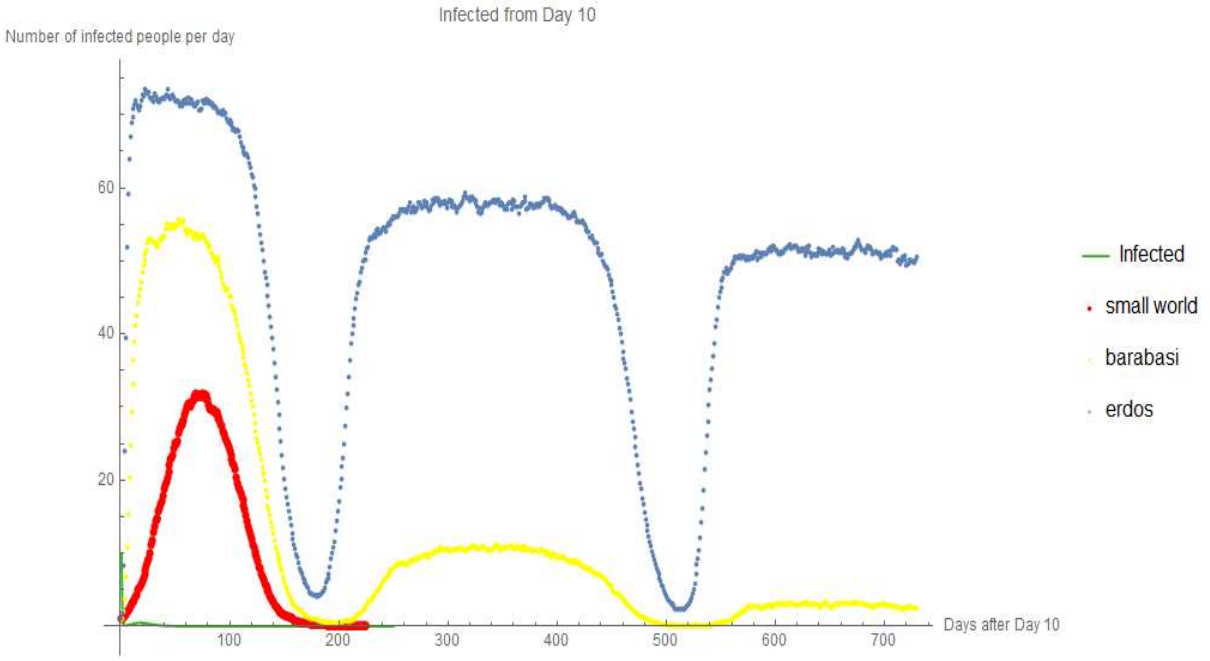

Figure 12: Comparison for population of 100 people

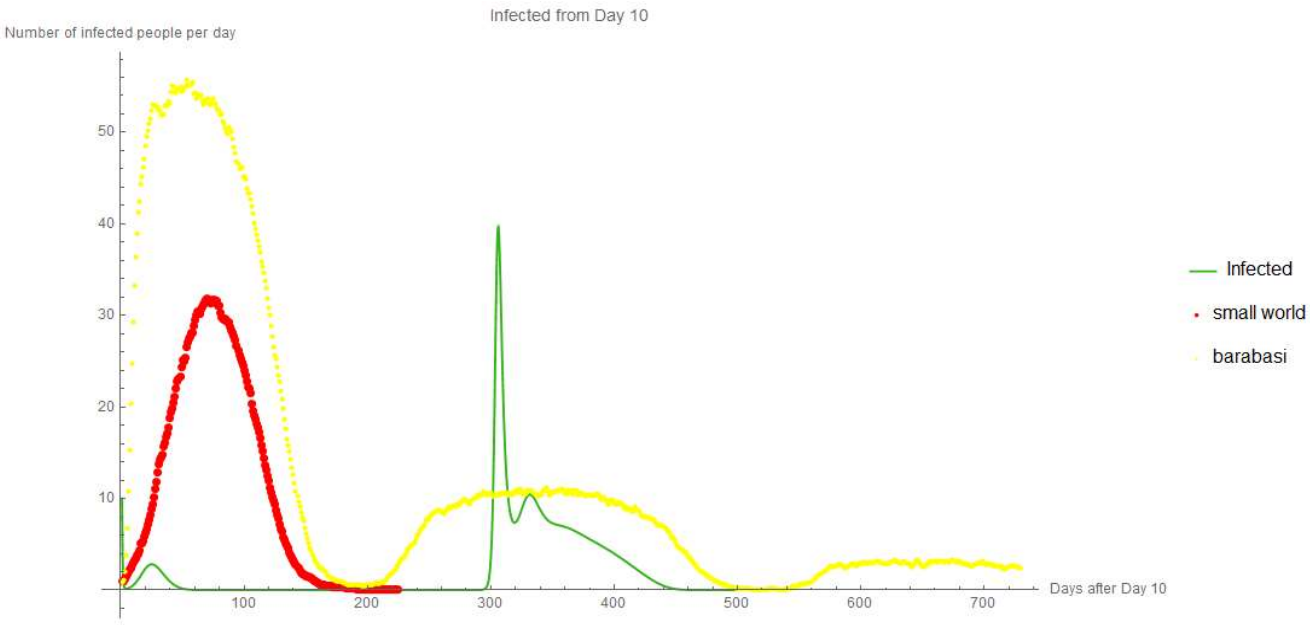

Figure 11: Comparison for population of 1000 people

As we can see the results differ a lot. For both cases the Watts-Strogatz model fails to predict a second wave at all. The Barabasi-Albert model for 1000 people comes closer to the predicted behaviour during the second wave but is still quite off.

The reason the results between networks and dynamical system might be a result of the following reasons:

1. The population plays a pivotal role in determining the behaviour of the epidemic. A severe lock down or social isolation measures.
2. The social distancing acts in a way that disturbs the interhuman relationships, meaning that a network with static topology (non-interchangeable vertices) is not well equipped to simulate the dynamical model.
3. Maybe the number of realizations is small, since they are uneven in size and there might be the need to adjust our sampling methods.

5.3. Application of Entropy

As was mentioned in part 5.1, there is a way to study the evolution of an epidemic and whether or not it reaches an end, or the virus remains active in the population. This is done usually by studying the system's stability.

5.3.1. System Stability and Epidemiology

The stability of a system as we have seen, is a useful tool that allows us to predict whether a dynamic process reaches a condition where its components remain stable[33]. For an epidemic a desired outcome is a stable point [34]where all groups except the recovered (or susceptibles in case of reintroduction) are equal to zero. A saddle point is worrisome, since any introduction of carriers would lead to a new epidemic cycle.

For the initial modeling (Simple SEIR model, we did study the system's stability. The results were not pleasing. We found 2 points in the 4-dimensional space. The first was the case that the infected person was healed during the first step, and the number of infected started to decline so fast ($i<1$) , so that almost all of population was unaffected. This version was a recuring result in the network simulations. The second solution is a perpetual cycle of epidemic, where an unstable point is defined as by the existence of infected and carriers. Such a grim result could mean that the new COVID cannot be removed from the human population.

Then we tried to solve the SEIDR-S system with static parameters. In a similar manner, the first two solutions mirrored the previous results (with unstable solution for the case of everyone returning to susceptibles), while a new third solution included negative population sizes, something that has no meaning.

5.3.2. Entropy comparison between networks and dynamical systems

We have decided to use a concept from statistical physics to try to predict whether the system reaches an eventual equilibrium point where the epidemic no longer persists, at least without invading carriers.

As we have seen, there are many types of entropy. We will use the classic Boltzmann- Gibbs entropy, however other types of entropy could have equally interesting results.

For this case we tried to make the following ansatz, each group of people is a different state where their total is a ground state. The entropy of each state is equal to $P_k = \log(\frac{p_k}{N})$, where p_k is the group we study. The percentage of people in each state shows that there is a dynamic process of diffusion. The system will reach its equilibrium when all people belong to one state, or the entropy stabilizes to one non-zero value. Those two states have the following properties:

- All people are in the same state means a system entropy equal to zero. This would mean that the system cannot evolve further from that condition, as all the people are in a final condition. The only group for which this is possible, is if everyone is in the susceptible group, since leaving this group is density based.
- The entropy is a stable non-zero point if all groups have at least some people in them. This would mean a permanent of the epidemic going through cycles of high and low spreading rates.

The way the system is modeled, we have only one possible equilibrium point which is covid-free for this system, when everyone is back at the susceptible group. This cannot tell us about whether that point is stable or unstable, however it can tell us if the epidemic cycle ends. If we have a system without mass transference (ie new viral content entering the system through tourists or immigration) then we may have new cycles begin after the entropy reaches 0. Every time the total entropy of the system reaches the stable point (population entropy $P_{tot} = 0$) a cycle ends.

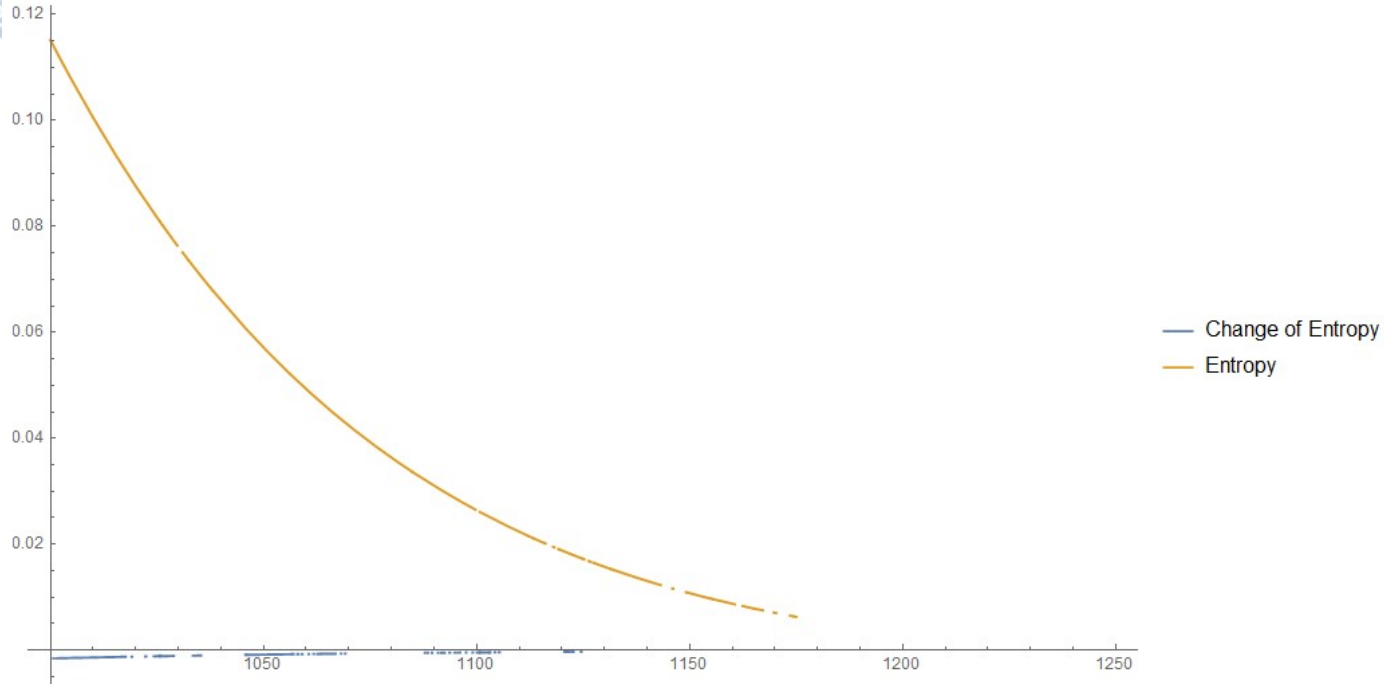

Figure 14: Entropy and Change of Entropy for the SEIDR-S model per day of the epidemic

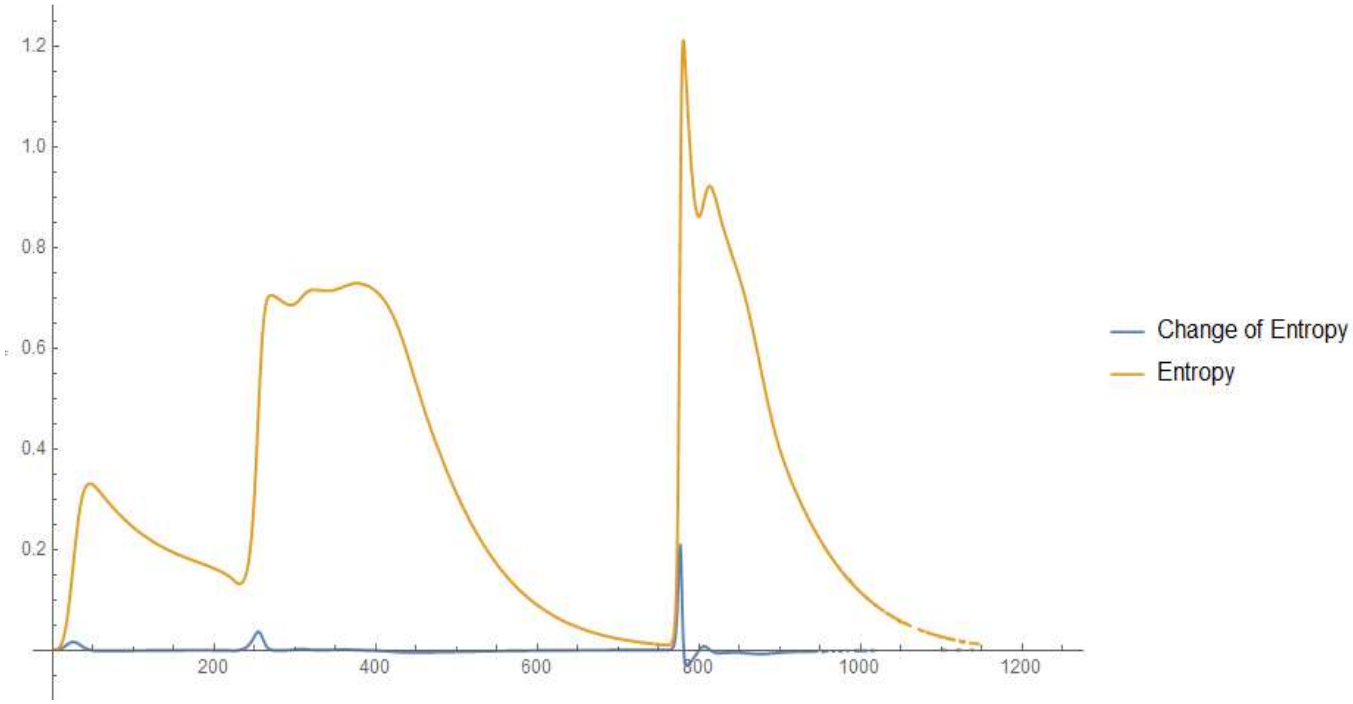

Figure 13: A closer look toward the cycle's end

As we see in the above figures, the entropy of the epidemic reaches zero for the first time around day 790 (approximately 2 years after the epidemic started, and reaches its first zero, close to day 1150. This would mean that based on our current epidemiological model, the COVID-19 epidemic (not taking vaccination under consideration) has a lifetime of ~3 and a half years.

After day 1150, we see that the systems entropy is hard to define. This is because the population of carriers and infected is only some decimals below 0.1, therefore a small but not strong enough to start the epidemic again. In a discreet system, this numerical problem would not be present.

Conclusions

Overall our model works as a flexible tool which if localized properly for the country-area of interest can yield incredible results. The model can be adjusted as to:

- How quickly the virus mutates.
- The effect that seasons and internal migration have to the spreading rate.
- The results severity of social distancing, civil unrest and prevention measures have.
- The size of the population and yearly fluctuations of it.
- Tourism and immigration effects, when carriers cross the borders.

We could expand the model with varying degrees of restriction as people enter the main population, add vaccinated group either as a group or as a dampening factor.

When it comes to the use of networks, the problem requires refinement of the algorithm used in order to reduce calculation time. Parallel programming might offer a little help, however the problem tends to be a NP problem, therefore any position cannot be supported until new versions of the algorithm are used. This means that models using networks in this scale are inconclusive.

For smaller size networks the results between networks and dynamical systems differ greatly. This is due to the fact that the distancing parameter was not destructive to the vertices and rewiring was not allowed (for Erdos-Renyi and Barabasi-Albert type of networks). The small world network did not adjust well to the social distancing, since although it rewired often, the number of links did not adjust to low social "mobility". It appears that we need to make more dynamic the vertices for a long-term simulation, however this would add to the complexity of the problem. We do not have data for smaller groups of people, therefore we cannot know whether the dynamic system or the networks provide more realistic data, however we make guesses about ways to improve the networks algorithms for future use.

Finally entropy appears to be a concept which could if properly prepared and studied across many models, provide an alternative to study stability in dynamical systems with time dependent parameters.

As a closing note, I would like to mention that all ideas about improving the model and the network equivalent, as well as the use of entropy as a tool for dynamical system diagnostics will be worked further by our team and any assistance would be welcome.

Appendices

Appendix A: Mathematica Model

The models used in the Wolfram Mathematica environment for simulating the epidemic. Comments will be highlighted in Green. Any piece of code can be copied and pasted into a Mathematica notebook (.nb , .m) and reproduce our results. In case of errors or results that make no sense, either check version sensibility or contact us at vvachtse@physics.auth.gr .

Appendix A.1. : Mathematica Model #1

```
(*Greece Pops *)
t0 = 14;
N0 = 10816286; (*total population (2011)*)
P = ((86440/tmax) + 79.554)/
  N0; (*Births (2018) and legal immigration (2011)*)
m = 120297/(N0*tmax); (*deaths (2018) *)

(*COVID-19 characteristics (world statistic\[Rule] we expect normal
\distribution due to the central limitation theorem *)

tsic = 30;(*coronavirus is present in Greece for 30 days as of today
\28/3/202*)
(*for our first estimate we will consider that the attack rate for
\all 3 types of infectants is the same *)
a = (0.01); (*mortality : https://coronavirus.jhu.edu/map.html \
28-03-2020)*)

(*At 28-03-2020 we had 966 confirmed cases, we suppose that there \
were at least 2000 sick people, either infected or asymptomatic*)

sig = 2000/60000;(*carriers who fall sick*)
```

```
    b1 = (2000/tsic);(* carrier spreading rate*)(*cases for the first
month*)

    b2 = b1;(* infected spreading rate*)

    k2 = 1 - a;(*cured infected *)

    k1 = (1/3); (* cured carriers*) (* From sars *)

    T=0.01;

    (* Initial Conditions*)

    i0 = 1;

    e0 = 0;

    s0 = N0 - i0 - e0;

    tmax = 365;



    ivs = {deq1, deq2, deq3, deq4, s[1] == s0, e[1] == e0, i[1] == i0,

       r[1] == 0};


    sol = NDSolve[{s'[t] == P - m*s[t] - T[t]*(b1*e[t] +
b2*i[t])*s[t]/(s[t] + i[t] + e[t] + r[t]),

        e'[t] == -(m + sig + k1)*e[t] + T[t]*(b1*e[t] +
b2*i[t])*s[t]/(s[t] + i[t] + e[t] + r[t]),

        i'[t] == sig*e[t] - (m + a)*i[t] - k2*i[t],

        r'[t] == k1*e[t] + k2*i[t] - m*r[t], s[1] == s0, e[1] == e0,

        i[1] == i0, r[1] == 0}, {s[t], e[t], i[t], r[t]}, {t, 1, tmax}];


    R0:=(T[t]*(b1+b2)/(2*m+k1+a+k2));

    Print["R0 ="]

    R0//N;

    Print["Number of Deaths:"]

    Integrate[a*isol[t],{t,1,tmax}]//N
```

Appendix A.2. : Mathematica Model #2

The following model describes a reverse bell distribution for the T parameter, and the parameters based on the previous model.

```
(*Greece Pops *)
t0 = 14;
N0 = 10816286; (*total population (2011)*)
P = ((86440/tmax) + 79.554)/
  N0; (*Births (2018) and legal immigration (2011)*)
m = 120297/(N0*tmax); (*deaths (2018) *)

(*COVID-19 characteristics (world statistic\[Rule] we expect normal
\distribution due to the central limitation theorem *)

tsic = 30;(*coronavirus is present in Greece for 30 days as of today
\28/3/202*)
(*for our first estimate we will consider that the attack rate for
\all 3 types of infectants is the same *)

(*We we will use the upper limit proposed by WHO, 2.5 R0  *)

a = (0.01); (*mortality : https://coronavirus.jhu.edu/map.html \
28-03-2020)*)

(*At 28-03-2020 we had 966 confirmed cases, we suppose that there \
were at least 2000 sick people, either infected or asyptomatic*)

sig = 2000/60000;(*carriers who fall sick*)
```

```
    b1 = (2000/tsic);(* carrier spreading rate*)(*cases for the first
month*)

    b2 = b1;(* infected spreading rate*)

    k2 = 1 - a;(*cured infected *)

    k1 = (1/3); (* cured carriers*) (* From sars *)


    (* Our Estimate for the Greek Quarantine \[Rule] 1/4 of cases*)

    tquar = 90;

    solpdf = Solve[(220/(Pi*Sqrt[14 t*(tquar - t)])) - 0.39 == 1, t];

    t1 = t /. solpdf[[1]];

    t2 = t /. solpdf[[2]];

    T[t_] := If[t2 > t > t1, (220/(Pi*Sqrt[14 (t)*(tquar - t)])) -
0.39,1] ;


    (* Initial Conditions*)

    i0 = 1;

    e0 = 0;

    s0 = N0 - i0 - e0;

    tmax = 365;



    ivs = {deq1, deq2, deq3, deq4, s[1] == s0, e[1] == e0, i[1] == i0,
       r[1] == 0};


    sol = NDSolve[{s'[t] == P - m*s[t] - T[t]*(b1*e[t] +
b2*i[t])*s[t]/(s[t] + i[t] + e[t] + r[t]),
        e'[t] == -(m + sig + k1)*e[t] + T[t]*(b1*e[t] +
b2*i[t])*s[t]/(s[t] + i[t] + e[t] + r[t]),
        i'[t] == sig*e[t] - (m + a)*i[t] - k2*i[t],
        r'[t] == k1*e[t] + k2*i[t] - m*r[t], s[1] == s0, e[1] == e0,
        i[1] == i0, r[1] == 0}, {s[t], e[t], i[t], r[t]}, {t, 1, tmax}];
```

Appendix A.3. : Mathematica Model #3

```
(*Greece Pops *)
t0 = 14;
N0 = 10816286; (*total population (2011)*)
P = ((86440/tmax) + 79.554)/
  N0; (*Births (2018) and legal immigration (2011)*)
m = 120297/(N0*tmax); (*deaths (2018) *)

(*COVID-19 characteristics (world statistic\[Rule] we expect normal
\
distribution due to the central limitation theorem *)

tsic = 30;

a = (0.01);(*mortality 28-03-2020)*)

(*At 28-03-2020 we had 966 confirmed cases, we suppose that there \
were at least 2000 sick people, either infected or asyptomatic*)

sig = 1/28;(*carriers who fall sick*) (**)

b1 = 966/tsic;(* carrier spreading rate*)(*cases for the first
month*)
b2 = b1;(* infected spreading rate*)
k2 = 1 - a;(*cured infected *)
k1 = (1/3);(* cured carriers*) (* From sars *)
tquar = 60;(*quarantine/isolation duration*)
rec = 1/t0;
qu = 9/10; (*quarantined patients*)
```

```
ttq = 1/tquar;
dis = 9/10;(*social distancing*)

(* Our Estimate for the Greek Quarantine \[Rule] 1/4 of cases*)
T = 0.025;

i0 = 1;
e0 = 0;
s0 = 0;
s0 = N0 - i0 - e0;

tmax = 365;


sol = NDSolve[{s'[t] ==
     P - m*s[t] -
      T*(b1*e[t] + b2*i[t])*s[t]/(s[t] + i[t] + e[t] + r[t] + d[t])
-
      dis*s[t]*i[t]/(s[t] + i[t] + e[t] + r[t] + d[t]) + ttq*d[t],
    e'[t] == -(m + sig + k1)*e[t] +
      T*(b1*e[t] + b2*i[t])*s[t]/(s[t] + i[t] + e[t] + r[t] + d[t])
-
      dis*e[t]*i[t]/(s[t] + i[t] + e[t] + r[t] + d[t]),
    i'[t] == sig*e[t] - (m + a)*i[t] - k2*i[t] - qu*i[t],
    r'[t] == k1*e[t] + k2*i[t] - m*r[t],
    d'[t] ==
     dis*(e[t] + s[t])*i[t]/(s[t] + i[t] + e[t] + r[t] + d[t]) +
      qu*i[t] - ttq*d[t],
     s[1] == s0, e[1] == e0, i[1] == i0, r[1] == 0, d[1] == 0},
{s[t],
     e[t], i[t], r[t], d[t]}, {t, 1, tmax}];
```

```
R0 = (T*(b1 + b2)/(2 m + sig + k1 + dis + 1 + qu));
Print["R0 ="]
R0 // N
Print["Number of Deaths:"]
Integrate[a*isol[t], {t, 1, tmax}] // N
```

Appendix A.4. : Mathematica Model #4

```
(*Greece Pops *)

t0 = 14; (*Life cyrcle of COVID19*)
N0 = 10816286; (*total population (2011)*)
P = ((86440/tmax) + 79.554)/
  N0; (*Births (2018) and legal immigration (2011)*)
m = 120297/(N0*tmax); (*deaths (2018) *)

(*COVID-19 characteristics (world statistic\[Rule] we expect normal
\distribution due to the central limitation theorem *)

tsic = 30;(*coronavitus is present in Greece for 30 days as of today
\28/3/202*)
(*for our first estimate we will consider that the attack rate for
\all 3 types of infectants is the same *)
(*We we will use the upper limit proposed by WHO, 2.5 R0  *)

a = (0.01);(*mortality : https://coronavirus.jhu.edu/map.html \
28-03-2020)*)

(*At 28-03-2020 we had 966 confirmed cases, we suppose that there \
were at least 2000 sick people, either infected or asyptomatic*)
```

```
sig = 1/t0;(*carriers who fall sick*) (*one per day for a 14 day \
cycle*)

b1 = 2.415;(* carrier spreading rate*)(*for the period that the
daily cases where rising, the linear approximation had the inclination
a=2.415*)

b2 = 2.415/5;(* infected spreading rate*)(* infected spreading rate
is the 1/5 of the carriers, due to people avoiding them ,on the basis
of their symptoms*)

k2 = 1 - a;(*cured infected *)

k1 = 1/3; (* based on SARS models*)

tquar = 60;(*quarantine/isolation/social distancing duration*)

rec = 1/t0; (*each individual is considered susceptible after one
life cycle of COVID*)

qu = 9.5/10; (*quarantined patients*)

ttq = 1/tquar;(* one person per tquar days returns to the
susceptibles team*)


dis = 1000;(*social distancing [Rule] 1000 people per percentage of
sick people in the whole populace*)


(* Our estimate of the safety parameter due to prevention from the
use of masks, gloves etc *)


T = 0.30;


(* initial conditions*)

i0 = 10;

e0 = 0;

s0 = 0;

s0 = N0 - i0 - e0;
```

```
(*one year*)

tmax = 365;


sol = NDSolve[{s'[t] ==

     P - m*s[t] -

      T*(b1*e[t] + b2*i[t])*s[t]/(s[t] + i[t] + e[t] + r[t] + d[t])
- dis*s[t]*i[t]/(s[t] + i[t] + e[t] + r[t] + d[t]) + rec*r[t] +  ttq*d[t],

    e'[t] == -(m + k1 + sig)*e[t] + T*(b1*e[t] + b2*i[t])*s[t]/(s[t]
+ i[t] + e[t] + r[t] + d[t]) -   dis*e[t]*i[t]/(s[t] + i[t] + e[t] +
r[t] + d[t]),

    i'[t] == sig*e[t] - (m + a)*i[t] - k2*i[t] - qu*i[t],

    r'[t] == k1*e[t] + k2*i[t] - m*r[t] - rec*r[t],

    d'[t] == dis*(e[t] + s[t])*i[t]/(s[t] + i[t] + e[t] + r[t] +
d[t]) +  qu*i[t] - ttq*d[t],

     s[1] == s0, e[1] == e0, i[1] == i0, r[1] == 0, d[1] == 0},
{s[t],

     e[t], i[t], r[t], d[t]}, {t, 1, 500}, MaxStepSize -> 1];


(*spreading rate without prevention*)

R0 = ((b1 + b2)/(2 m + sig + 1 + k1));

Print["R0 ="]

R0 // N


(*spreading rate with prevention*)

RT = (T (b1 + b2)/(2 m + sig + 1 + k1));

Print["RT ="]

RT // N


Print["Number of Deaths (Yearly):"]

Integrate[a*isol[t], {t, 1, tmax}] // N
```

Appendix A.5. : Mathematica Model #5

```
(*Greece Pops *)
t0 = 14; (*Life cyrcle of COVID19*)
N0 = 10816286; (*total population (2011)*)
P = ((86440/tmax) + 79.554)/
   N0; (*Births (2018) and legal immigration (2011)*)
m = 120297/(N0*tmax); (*deaths (2018) *)
(*COVID-19 characteristics (world statistic\[Rule] we expect normal
dis[t]tribution due to the central limitation theorem *)

tsic = 30
a = (0.01);

sig[t_] :=
  1/t0 *(1 + t/120);(*carriers who fall sick*) (*one per day for a
14 day cycle*)

b1 = 2.415;
b2 = 2.415/5   5;
k2 = 1 - a;(*cured infected *)
k1 = 1/3; (* based on SARS models*)

tquar = 60;(*quarantine/isolation/social dis[t]tancing duration*)
rec = 1/t0; (*each individual is considered susceptible after on
life cycle od COVID*)
qu = 9.5/10; (*quarantined patients*)
ttq = 1/tquar;(* one person per tquar days returns to the
susceptibles team*)
```

```
    dis[t_] :=  1000 (Abs[(175 - t)/175] Abs[(1 - (t/300))]);(*social
dis[t]tancing \[Rule] 1000 people per percentage of sick people in the
whole populace*)

    T = 0.30;


    (* Tourist influx parameter*)

    tt[t_] := Abs[Sin[Pi*(t/365)]];


    (*Seasonality*)

    tp[t_] := (1 + (1/2)*Abs[Sin[Pi*(t/175)]]);


    sol = NDSolve[{s'[t] == P + 1000*tt[t] - m*s[t] -
T*(b1*tp[t]*e[t] + b2*tp[t]*i[t])*s[t]/(s[t] + i[t] + e[t] + r[t] + d[t])
- dis[t]*s[t]*i[t]/(s[t] + i[t] + e[t] + r[t] + d[t]) + rec*r[t] +
ttq*d[t],e'[t] == 50*tt[t] - (m + k1 + sig[t])*e[t] +
T*(b1*tp[t]*e[t] + b2*tp[t]*i[t])*s[t]/(s[t] + i[t] + e[t] + r[t] + d[t])
- dis[t]*e[t]*i[t]/(s[t] + i[t] + e[t] + r[t] + d[t]),i'[t] ==
sig[t]*e[t] - (m + a)*i[t] - k2*i[t] - qu*i[t],r'[t] == k1*e[t] + k2*i[t]
- m*r[t] - rec*r[t],  d'[t] == dis[t]*(e[t] + s[t])*i[t]/(s[t] + i[t] +
e[t] + r[t] + d[t]) + qu*i[t] - ttq*d[t],s[1] == s0, e[1] == e0, i[1]
== i0, r[1] == 0, d[1] == 0}, {s[t], e[t], i[t], r[t], d[t]}, {t, 1,
500}, MaxStepSize -> 1];

    (*spreading rate without prevention*)

    R0 = ((b1 + b2)/(2 m + sig[t] + 1 + k1));

    Print["R0 ="]

    R0 // N

    (*spreading rate with prevention*)


    RT = (T (b1 + b2)/(2 m + sig[t] + 1 + k1));

    Print["RT ="]

    RT // N


    Print["Number of Deaths (Yearly):"]

    Integrate[a*isol[t], {t, 1, 365}] // N
```

Appendix A.6. : Mathematica Model #6

```
(*Greece Pops *)

t0 = 14; (*Life cyrcle of COVID19*)

N0 = 10816286; (*total population (2011)*)

P = ((86440/tmax) + 79.554)/N0; (*Births (2018) and legal immigration (2011)*)

m = 120297/(N0*tmax); (*deaths (2018) *)

tsic = 30;


atr[t_] := 2*((1 + (t/1460))/100);(*mortality : https://coronavirus.jhu.edu/map.html \28-03-2020)*)

sig[t_] := 1/t0 *(1 + t/120);(*carriers who fall sick*) (*one per day for a 14 day \cycle*)


b1 = 2.415

b2 = 2.415/ 5


k2 = 1 - atr[t];(*cured infected *)

k1 = 1/3; (* based on SARS models*)


tquar = 60;(*quarantine/isolation/social dis[t]tancing duration*)

rec = 1/t0; (*each individual is considered susceptible after on life \cycle of COVID*)

qu = 9.5/10; (*quarantined patients*)

ttq = 1/tquar;(* one person per tquar days returns to the susceptibles *)


dis[t_] := 1000 (Abs[(175 - t)/175] Abs[(1 - (t/300))]);

(*social dis[t]tancing \[Rule] 1000 people per percentage of sick people in the whole populace*)
```

```
T = 0.30;

(* Tourist influx parameter*)
tt[t_] := Abs[Sin[Pi*(t/365)]];

(*Seasonality*)
tp[t_] := (1 + (2/5)*Sin[Pi*((t - 14)/(160))])*(1 +
     Cos[Pi*((t + 35)/(222))]);

(* initial conditions*)
i0 = 10;
e0 = 0;
s0 = 0;
s0 = N0 - i0 - e0;

(*one year*)
tmax = 365;

sol = NDSolve[{s'[t] ==
     P + 1000*tt[t] - m*s[t] -
      T*(b1*tp[t]*e[t] + b2*tp[t]*i[t])*
       s[t]/(s[t] + i[t] + e[t] + r[t] + d[t]) -
      dis[t]*s[t]*i[t]/(s[t] + i[t] + e[t] + r[t] + d[t]) + rec*r[t]
+  ttq*d[t],
    e'[t] ==
     50*tt[t] - (m + k1 + sig[t])*e[t] +
      T*(b1*tp[t]*e[t] + b2*tp[t]*i[t])*
       s[t]/(s[t] + i[t] + e[t] + r[t] + d[t]) -
      dis[t]*e[t]*i[t]/(s[t] + i[t] + e[t] + r[t] + d[t]),
    i'[t] == sig[t]*e[t] - m*i[t] - atr[t]*i[t] - k2*i[t] - qu*i[t],
```

```
        r'[t] == k1*e[t] + k2*i[t] - m*r[t] - rec*r[t],
       d'[t] ==
        dis[t]*(e[t] + s[t])*i[t]/(s[t] + i[t] + e[t] + r[t] + d[t]) +
         qu*i[t] - ttq*d[t],
        s[1] == s0, e[1] == e0, i[1] == i0, r[1] == 0, d[1] == 0},
{s[t],
        e[t], i[t], r[t], d[t]}, {t, 1, 5000}, MaxSteps -> Infinity];

    (*spreading rate without prevention*)
    R0 = ((b1 + b2)/(2 m + sig[t] + 1 + k1));
    Print["R0 ="]
    R0 // N
    (*spreading rate with prevention*)

    RT = (T (b1 + b2)/(2 m + sig[t] + 1 + k1));
    Print["RT ="]
    RT // N

    Print["Number of Deaths (Yearly):"]
    Integrate[atr[t]*isol[t], {t, 1, 365}] // N
```

Appendix A.7. : Mathematica Model #7

```
(*Greece Pops *)
t0 = 7; (*Life cyrcle of COVID19*)
N0 = 10816286;
P = ((86440/tmax) + 79.554)/ N0;
m = 120297/(N0*tmax);
tsic = 30;

atr[t_] := (0.215) ((1 + (t/1825))/ 10);

sig[t_] :=1/t0 *(1 +  t/120);

b1 = 2.415;
b2 = 2.415/5;

k2 = 1 - atr[t];
k1 = 1/3;
tquar = 60;
rec = 1/t0;
qu = 9.5/10;
ttq[t_] := (1/tquar) + ((Exp[(t - 210)/60]))/(10^9) ;

dis[t_] := 1000 (Abs[ Cos[(t/900)*2* Pi]]*(1/(Exp[(t - 275)/30] +
1)));
T = 0.30;

(* Tourist influx parameter*)
tt[t_] := Abs[Sin[Pi*(t/365)]]*(1/(Exp[(t - 450)/15] + 1));
```

```
(*Seasonality*)
mt[t_] := Exp[t/2500]; (* Mutation*)
tp[t_] := mt[t]*(1 + Cos[2*Pi*((t + 106)/(455))]);

i0 = 1;
e0 = 0;
s0 = 0;
s0 = N0 - i0 - e0;
tmax = 365;

sol = NDSolve[{s'[t] ==
     P + 1000*tt[t] - m*s[t] -
      T*(b1*tp[t]*e[t] + b2*tp[t]*i[t])*
       s[t]/(s[t] + i[t] + e[t] + r[t] + d[t]) -
      dis[t]*s[t]*i[t]/(s[t] + i[t] + e[t] + r[t] + d[t]) + rec*r[t]
+
       ttq[t]*d[t],
     e'[t] ==
      50*tt[t] - (m + k1 + sig[t])*e[t] +
      T*(b1*tp[t]*e[t] + b2*tp[t]*i[t])*
       s[t]/(s[t] + i[t] + e[t] + r[t] + d[t]) -
      dis[t]*e[t]*i[t]/(s[t] + i[t] + e[t] + r[t] + d[t]),
     i'[t] == sig[t]*e[t] - m*i[t] - atr[t]*i[t] - k2*i[t] - qu*i[t],
     r'[t] == k1*e[t] + k2*i[t] - m*r[t] - rec*r[t],
     d'[t] ==
      dis[t]*(e[t] + s[t])*i[t]/(s[t] + i[t] + e[t] + r[t] + d[t]) +
       qu*i[t] - ttq[t]*d[t],
      s[1] == s0, e[1] == e0, i[1] == i0, r[1] == 0, d[1] == 0},
{s[t],
      e[t], i[t], r[t], d[t]}, {t, 1, 1000}, MaxSteps -> Infinity];
```

Appendix B: R Code/ Model

```
library(stats)
library(igraph)
#network characteristics
netsize=1000 #network size
iin=1 #infected cases at the start
tsteps=730 #2 years approximately
runs=5 # number of simulations
#We can use barabasi.game or erdos.renyi.game for different network, but for
#this instance we consider that the small world approach works better for a real
#society. We will not take into consideration the dynamics of social interaction
# where new people meet or people stop interacting
#We set new attributes that beter describe the Infected of the people and later,
#their isolation
#dead V(netg) will be removed, based on the attribute "alive"
#1. Create the network based on the chosen specs.
#Here we use a barabasi-albert
netg <- barabasi.game(netsize, m = 5, directed=FALSE)
#2.Create the attributes required, Infected, Alive and Isolation
set_vertex_attr(netg, "Infected", index = V(netg), value = TRUE)
set_vertex_attr(netg, "Carrier", index = V(netg), value = TRUE)
set_vertex_attr(netg, "Alive", index = V(netg), value = TRUE)
set_vertex_attr(netg, "Isolation", index = V(netg), value = TRUE)

#3. Initialise the attributes accordingly
V(netg)$Infected=FALSE
```

```
V(netg)$Carrier=FALSE
V(netg)$Alive=TRUE
V(netg)$Isolation=FALSE
#The next step is to create functions that will make the dynamic process
#of simulating the spread of the virus
netmulation<-function(netg,netsize,iin,runs,tsteps){
  linf<- c()
  lcaf<- c()
  lisf<- c()
  for (runt in 1:runs) {
    V(netg)$Infected=FALSE
    V(netg)$Carrier=FALSE
    V(netg)$Alive=TRUE
    V(netg)$Isolation=FALSE
  #Initialization
    n1<-sample(1:netsize,iin)
    V(netg)$Infected[n1]<-TRUE
    V(netg)$Carrier[n1]<-TRUE
    lin<-c()
    lca<-c()
    lis<-c()
    for (t in 1:tsteps) {
    #Probabilities
      p1<-(1 + (2/5)*sinpi(((t - 14)/134)))*(1 + cospi((t + 14)/168))/20 #Infection rate
      p11<-(1 + (2/5)*sinpi(((t - 14)/134)))*(1 + cospi((t + 14)/168))/4 #Infection rate for carriers

      p2<-1/14 #Healing factor
      p22<-1/14 #Healing factor for carriers
```

```
  p3<-0.0013*((1 + (t/1460))/100) #Mortality

  p4<-1/3 #Isolation
  p41<-1/4#Getting out of the isolation

  p5<-1/3 #Carrier falling sick

  lister<-c()
  lister2<-c()
  lister3<-c()

  for (i in 1:netsize) {
    if(V(netg)$Alive[i]==TRUE){
    #List of infected people
      if(V(netg)$Carrier[i]==TRUE){
        lister2<-append(lister2,i)
        if(V(netg)$Infected[i]==TRUE)
          lister<-append(lister,i)
    }
  }
}

#Isolated People

  for (iii in 1:netsize) {
    if(V(netg)$Isolation[iii]==TRUE)
      lister3<-append(lister3,iii)
}
```

```
  eg1<-ego(netg,order=1,lister)  #First  neigbhours  for  each
infected person
  eg2<-ego(netg,order=1,lister2)  #First  neigbhours  for  each
carrier person

  l1<- length(eg1) #Number of Infected per step
  l11<- length(eg2) #Number of Carriers per step

  l2<- length(lister)
  l22<- length(lister2)

  l3<-length(lister3) #Number of Isolated people



  if(l22<1){
    break
}

#Death Process
  for (n in 1:l1){
    if(runif(1,0,1)<p3)
      V(netg)$Alive[i]==FALSE }
#Healing Process

  for (k in 1:l22) {
    if(V(netg)$Infected[lister2[k]]==TRUE){
      if(runif(1,0,1)<p2){
        V(netg)$Carrier[lister2[k]]=FALSE
```

```
            V(netg)$Infected[lister2[k]]=FALSE
          }}
          else
            if(runif(1,0,1)<p22){
              V(netg)$Carrier[lister2[k]]=FALSE
          }
        }

        #Carrier fallin sick

          for (jj in 1:l22) {#Check if person is sick or not
            counter2<-lister2[jj]
            if(V(netg)$Infected[counter2]==FALSE){# If not isolated the
spread of the disease goes on as normal
              if(V(netg)$Carrier[counter2]==TRUE){
                if(runif(1,0,1)<p5){
                  V(netg)$Infected[counter2]=TRUE
              }}}}


        #Infection Spreading

          for (j in 1:l22) {#Check if person is isolated or not
            counter1<-lister2[j]
            if(V(netg)$Isolation[counter1]==FALSE){#  If  not  isolated
the spread of the disease goes on as normal
              if(V(netg)$Carrier[counter2]==TRUE){
                if(V(netg)$Infected[counter1]==TRUE){#If  a  person  is
infected
                  egon_net<-eg2[[j]]
                  le<-length(egon_net)
```

```
            for (j1 in 1:le) {
              if(runif(1,0,1)<p1){
                V(netg)$Carrier[egon_net[j1]]=TRUE
            }}}
          else { #From Carriers
            egon_net<-eg2[[j]]
            le<-length(egon_net)
            for (j1 in 1:le) {
              if(runif(1,0,1)<p11){
                V(netg)$Carrier[egon_net[j1]]=TRUE
            }}}
      }}}


#Isolation Process

  for (ii in 1:netsize) {
    if(runif(1,0,1)<p4)
      V(netg)$Isolation[ii]=TRUE
}

  for (jk in 1:l3) {
    if(runif(1,0,1)<p41)
      V(netg)$Isolation[lister3[jk]]=FALSE
}

  lin<-append(lin,l1)
  lin2<-list(lin)
```

```
            lca<-append(lca,l11)
            lca2<-list(lca)

            lis<-append(lis,l3)
            lis2<-list(lis)

          }

          linf<- append(linf,lin2)
          lcaf<- append(lcaf,lca2)
          lisf<- append(lisf,lis2)


        }

        Results <- list("Infected" = linf, "Carriers" = lcaf,"Isolated"=lisf)

        return(Results)

      }
      #Execution

      tt1<-netmulation(netg,netsize,iin,runs,tsteps)

      Ison<-as.data.frame.vector(tt1$Infected)
      Rson<-as.data.frame.vector(tt1$Infected)
      Isson<-as.data.frame.vector(tt1$Isolated)

      sink("Results_Infected07.csv",append = F)
```

```
Ison
sink()

sink("Results_Carriers07.csv",append = F)
Rson
sink()

sink("Results_Isolated07.csv",append = F)
Isson
sink()
```

References

[1] Klaus Dietz, J.A.P. Heesterbeek, Daniel Bernoulli's epidemiological model revisited, Mathematical Biosciences, Volume 180, Issues 1–2, 2002, Pages 1-21, ISSN 0025-5564, https://doi.org/10.1016/S0025-5564(02)00122-0

[2] A Contribution to the Mathematical Theory of Epidemics.By W. 0. Kermack and A. G. McKendrick.(Communicated by Sir Gilbert Walker, F.R.S.—Received May 13, 1927.) (From the Laboratory of the Royal College of Physicians, Edinburgh.)

[3] The cholera near Golden-Square, and at Deptford, John Snow, The Medical Times and Gazette. 2nd series. 9: 321–322.; see p. 322, 1854

[4] Epidemiology : Principles and Practical Guidelines, J Van den Broeck , J R Brestoff, , Springer

[5] Concepts of Epidemiology, Raj Bhorpal, Oxford

[6] Calculation of Infection Rates, Utah Department of Health. 2017.

[7] Introduction to non-linear dynamical systems, G. Voyatzis, E.Meletlidou , ISBN: 978-960-603-103-8

[8] Non linear ordinary differential equations, A. Bountis

[9] Dynamical Systems and Chaos, A. Bountis

[10] Contninuous and Discrete Dynamical Systems, A. Bountis

[11] Lecture notes from "Network Theory and Dynamical Systems " from the interdisciplinary Master's Program, Networks and Complexity 2019

[12] A Network-Based Stochastic Epidemic Simulator:Controlling COVID-19 with Region-Specific Policies, A. Kuzdeuov et al, https://doi.org/10.1101/2020.05.02.20089136

[13] General properties of entropy, A. Wehrl

[14] The Generalized Boltzmann Distribution is the Only Distribution in Which the Gibbs-Shannon Entropy Equals the Thermodynamic Entropy, X. Gao et al,

[15] Entropy production theorems and some consequences, A.Saha et al, PACS numbers: 05.40-a, 05.70.Ln, 05.20.-y

[16] Early dynamics of transmission and control of COVID-19: a mathematical modelling study, A.J. Kucharski, T. W. Russell et al , https://doi.org/10.1016/S1473-3099(20)30144-4

[17] Novel coronavirus 2019-nCoV: early estimation of epidemiological parameters and epidemic predictions , J.M.Read, J.R.E Bridgen et al , doi: https://doi.org/10.1101/2020.01.23.20018549

[18] Effectiveness of control strategies for Coronavirus Disease 2019: a SEIR dynamic modeling study, J.Pan, Y.Yao , Z.Liu et al , doi: https://doi.org/10.1101/2020.02.19.20025387

[19] Bifurcations and complex dynamics of an SIR model with the impact of the number of hospital beds, C.Shan, H.Zhu, J. Differential Equations 257 (2014) 1662-1688

[20] Bifurcations analysis and tracking control of an epidemic model with nonlinear incidence rate, Na Yi, P. Liu, Q. Zhang, Applied Mathematical Modelling 36 (2012)

[21] John Hopkins Hospital COVID19 watch https : // coronavirus.jhu.edu/map.html

[22] Official WHO estimations for mortality rate and spreading rate , as of 28/03/2020 https : // www.worldometers.info/coronavirus/coronavirus - cases/#total - cases

[23] Hellenic Statistical Authority https : // www.statistics.gr/el/statistics/pop

[24] A Novel Dynamic Model Describing the Spread of the MERS-CoV and the Expression of Dipeptidyl Peptidase 4, S.Tang et al , https://doi.org/10.1155/2017/5285810

[25] Middle East respiratory syndrome coronavirus: quantifi cation of the extent of the epidemic, surveillance biases, and transmissibility,S. Cauchemez et al, November 13, 2013, http://dx.doi.org/10.1016/S1473-3099(13)70304-9

[26] Middle East Respiratory Syndrome Coronavirus Outbreak in the Republic of Korea, 2015, Korea Centers for Disease Control and Prevention

[27] Modeling the impact of quarantine during an outbreak of Ebola virus disease, A. Denes , A.B. Gummel, Infectious Disease Modeling 4 (2019)12-27

[28] A Discrete Epidemic Model for SARS Transmission and Control in China, Y. Zhou, Z. Ma, Mathematical and Computer Modelling 40 (2004) 1491-1506

[29] Dynamics of an epidemic model with quadratic treatment, J.C. Eckalbar, W.L. Eckalbar, Nonlinear Analysis: Real World Applications 12 (2011), 320-332

[30] Investigation of nonlinear epidemiological models for analyzing and controlling the MERS outbreak in Korea, I. Ahn et al , Journal of Theoretical Biology 437 (2018) 17-28

[31] A dynamic compartmental model for the Middle East respiratory syndrome outbreak in the Republic of Korea: A retrospective analysis, J. Lee et al, Journal of Theoretical Biology 408 (2016) 118–126
on control interventions and superspreading events

[32] Middle East respiratory syndrome coronavirus (MERS-CoV): Prevention in travelers, A. Pavli, S. Tsiodras, H.C. Maltezou, Travel Medicine and infectious Disease (2014) 12 , 602-608

[33] Stability analysis in a class of discrete SIRS epidemic models, Z.Hu, et al, Nonlinear Analysis: Real World Applications 13 (2012) 2017-2033

[34] Global stability of an SI epidemic model with feedback controls,L.Chen , J. Sun , Applied Mathematics Letters 28 (2014), 53-55