A Differential Equations Analysis of Pandemic Disease Spread in an Apocalyptic State

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Abstract

The biological threat of a pandemic disease eruption has become all too real in the current state of world affairs. Although adorned by Hollywood tycoons, the threatening danger of biochemical disease warfare is a reality. If a weaponized virulent warhead containing a highly infectious contagion were to detonate on American soil, what are the chances of controlling the disease? Depending on the infectious range used of diseased individuals, as well as the radius and population density of the initial disease outbreak, we can model different scenarios that adjust these density parameters using differential equations. This will enable us to more accurately create a plan in which survival and suppression is most effective. In population-dense areas such as the District of Columbia, can a highly transmissive virus even be contained? If the contagion is designed to create a highly aggressive host, survival may not be possible. At this point if the contagion has no known suppressor or cure, in order to save the most lives, the only possibility may be to isolate and eliminate everything within the infected radius.

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‡Special thanks to Dr. Stephen D. Casey - an excellent mentor and professor whose teaching and care for his students shall always remain unmatched.

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Part I

Introduction

Ever since the human population was brought to its knees at the hands of the Black Plague in the Dark Ages, man has developed a vested interested in how disease is spread. Man once stood face to face with the reaper of death, just barely escaping complete extinction. How is it possible that such a small pathogen spread so rapidly? Before anyone knew how to react, the disease had crippled the very fabrics of society.

The purpose of this research is to analyze the way in which a disease system changes as a function of time. The parameters of the disease model will then be altered to model the rates of change for a rapid pandemic disease spread. A hypothetical disease will then be created based on observable characteristics of known biological parasites. Based on the model created, both the equilibrium points of disease eradication and human eradication will be qualitatively interpreted to determine the feasibility of both scenarios.

Part II

Differential Equations and Disease Modeling

Differential Equations are used in order to model a changing system that involves the derivatives of a function coupled with the function itself. This type of system modeling has been found useful in areas of biology, physics, and engineering. The rates at which each system changes with respect to the time is calculated by taking the derivative of the system. A derivative is simply the rate of change on the system[7].

Ordinary Differential Equations are given in the form

$$F(x, y, y', \ldots, y^{(n)}) = 0$$

where y is given as a function of x such that we have the first derivative $y' = \frac{dy}{dx}$ in terms of x. This also gives the n^{th}derivative as $y^{(n)} = d^n y/dx^n$.[7,8]

In terms of disease modeling, the simplest system was created by Kermack and McKendrick. This model was created to explain the rapid rise and fall of the number of infected individuals in epidemics such as the plague. The number of people infected with the illness was modeled over a closed period of time and assumes a fixed population size such that there are no births, death due to disease, or non-disease-related deaths[6,7].

Part III

The SIR Model

The Kermick-McKendrick model, more commonly referred to as the SIR model, models the number of people in a closed population infected during the outbreak of a contagion over time. Kermick and McKendrick created three coupled nonlinear ordinary differential equations to model the system. The first group consisted of the population of susceptible individuals and was labeled as $S(t)$. Those who were in the susceptible group were capable of being infected. The second group was the infected members of the population, labeled as $I(t)$. Those who were infected were capable of spreading the infection to members of the susceptible population. The final group was the recovered individuals, labeled as $R(t)$. Those in the recovered group were assumed to be immune to the contagion[5,6].

All three groups are then subdivided into fractions of the whole population yielding:[1]

$$(1) \quad s(t) = \frac{S(t)}{N}$$

$$(2) \quad i(t) = \frac{I(t)}{N}$$

$$(3) \quad r(t) = \frac{R(t)}{N}$$

Since this model uses a closed population, at all time t, we have $s(t) + i(t) + r(t) = 1$. Moreover, there will never be an addition to the susceptible group since birth rate and immigration are ignored. Therefore, only those who are infected will leave the susceptible group. The rate at which the members of $s(t)$ change will be dependent on the total of infected individuals present. Now we assume that there will be β contacts per unit of time that will be sufficient enough to spread the disease. This will mean that there will be $\beta s(t)$ new infected individuals generated per unit of time[5].

The only way in which an infected can change populations is by recovering and developing an immunity to the disease. We assume that there will be a fixed number of individuals in $i(t)$ recovering by parameter α and entering $r(t)$. This model can be shown by figure 1.

Based on these assumptions, we have the systems changing in the following ways:

$$(4) \quad \frac{ds}{dt} = -\beta s(t) i(t)$$

$$(5) \quad \frac{di}{dt} = \beta s(t) i(t) - \alpha i(t)$$

$$(6) \quad \frac{dr}{dt} = \alpha i(t)$$

[1] N is the total population of the model

These three nonlinear ordinary differential equations lead to the following system[5]:

$$(7) \quad \frac{ds}{dt} + \frac{di}{dt} + \frac{dr}{dt} = 0$$

Figure 1:

SIR Model - Graphic Representation

Part IV

Pathogens and Parasites

What would happen if a contagion infected a human so that they lose control of their functioning and become helpless at the hands of the governing parasite? Organisms have been infected in such ways in various parts of the animal kingdom. The infecting organism changes the behavioral patterns of the host in order to allow the parasite to proliferate, sometimes even at the cost of the host's life.

One parasitic worm, *Euphalorchis californiensis,* infects the gills of killifish. This parasitic worm surrounds the brain of the fish and cause the fish to swim to the surface of the water. Once the organism reaches the surface, they turn onto their sides and flash the reflective gills underneath their fins. This alerts predators of the locations of the killifish and allows them to be eaten. Once the birds have consumed the fish, the parasitic worm is then spread to

other fish in the water via the fecal matter of the birds. These killifish, who rarely breach the surface of the water, are shown to sacrifice their own health in order to allow the *Euphalorchis californiensis* to spread[9]. Not only is the behavior of the killifish changed, but it sacrifices its own life.

Another interesting organism is the *Spinochordodes tellinii*, a nematomorph parasitic hairworm. Grasshoppers accidentally ingest the microscopic larvae of the parasite which can be found deposited throughout the wild. Once the larvae are ingested, they produce a protein that affects the brain and the nervous system of the host grasshopper. The parasite stays within the grasshopper until the time comes in which the worm must transform into an aquatic adult. The proteins that the worm produces directly and indirectly affect the central nervous system of the grasshopper and changes the behavior of the grasshopper so that it will seek out water to plunge into. The worm will then emerge from the dying or dead host in order to seek out a mate[1].

The final organism that will be introduced is the *Leucochloridium paradoxum,* an endoparasitic worm that infects *Succinea* snails in the wild. The flat worm enters the digestive tracts of the snail where the larvae grow and develop sporocysts. The snail is used as an intermediate host as the parasite makes its way into a terminal host. The terminal host for the *Leucochloridium paradoxum* is a bird. The bird is used as a means of spreading the eggs of the parasite throughout the animal kingdom in its excrements. In order to be introduced into the bird, the snail that the worm infects must first be consumed by the bird. The sporocysts inside the snail grow into the tentacles of the host and causes elongation and pulsation[3]. The movement and color attract birds who rip off the tentacles and ingest the parasite. It is interesting that the *Succinea* snails are consumed by the birds since they are normally photophobic. However, the flat worm inside the infected snails somehow causes the organism to become photophylic and climb to the tops of tall trees and grasses[2,3]. All of the behavioral properties of the snail are amazingly changed by this infecting parasite.

Part V

The Human Infection

There are several examples in nature of parasites that infect organisms and change the way in which they behave in an extreme manner. Oftentimes, the host organism will risk their life for the proliferation of the infecting agent. Now suppose a similar parasite was to develop in humans that rapidly develops and reproduces. The human body would act as the host environment for the organism to develop within. Once the parasite is mature and the host environment has reached the saturation level for growth and development, the organism could cause the infected person to seek other environments for the parasite to live within. If the parasite lives within the blood of the host organism, the only logical means of transmission into newer hosts would be via saliva and blood contact.

As we saw in other organisms, the parasites are capable of releasing proteins that change the way in which the host acts. In the case of a human infection with the goal of blood and saliva transmission in mind, chemical signatures could be released into the blood that affect the Limbic System. This is the system that deals with the primitive functions of our brain, which include emotions such as anger, aggression, and hunger. If this area were affected, the host human could show abnormally increased aggression towards others. Moreover, the proteins within the blood could cause pheromone release. This would cause the organism to seek only those who were not already infected. Eventually, the host body would not be capable of supporting both the parasite and itself. As a result, the infected would die.

Part VI

The Disease Model

With the given background on the human parasite discussed, the model that we use to analyze the infection spread would need to change. This would call for a lethal pandemic model in which the infected would be capable of dying. Philip Munz, Ioan Hudea, Joe Imad, and Robert J. Smith used a model in their paper which can be applied to this situation, however, they created a parameter that would move the deceased individuals back into the infected environment[4]. Assuming that once an individual expires from the disease that they cannot actively infect[2] the susceptible, we can alter this model so that it will fit our disease model.

Just as in the SIR model, there will be three different groups that the population will be divided into. The first will be the susceptible, $S(t)$. These are the individuals who are healthy and capable of being infected. The next group is the infected, $Z(t)$. These individuals have been infected by the parasite and are capable of spreading the disease. The final group is the removed individuals, $R(t)$. These individuals have died either of natural causes or via infection[4].

The susceptible are capable of dying and entering the removed group by natural causes via parameter δ, or they can enter the infected group via parameter β. Since the infected are abnormally aggressive towards non-infected individuals, it can be assumed that the susceptible would take defensive moves against the actions of the infected. Via parameter α, those who are infected can be killed by a susceptible and enter the removed group[4]. This will result in the model shown in Figure 2.

Since this model has two mass-action transmissions, there will be more than one nonlinear term in the model, making it more difficult. This mass-action incidence will specify that an

[2]actively infecting can be assumed as infecting via aggression that leads to blood or saliva contact with that of a susceptible individual.

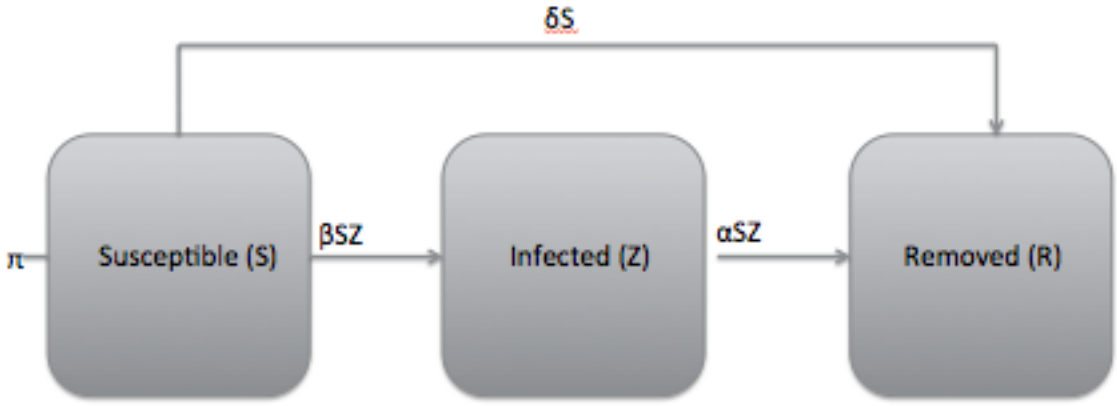

Figure 2:

SZR Model - Graphic Representation

average population member would need to make sufficient contact so that it could spread the infection with βN other individuals in one unit of time. The probability of a susceptible making random contact with a non-infected is S/N. This yields the new total of new infected being $(\beta N)(S/N)Z = \beta SZ$.

Since the susceptible also have the ability to defend themselves, we assume then that αN infected will be killed based on the probability of S/N contacts such that the total infected removed will be $(\alpha N)(Z/N)S = \alpha SZ$[4].

Based on these parameters, these three groups would change as follows:

$$S' = \pi - \beta SZ - \delta S \tag{8}$$

$$Z' = \beta SZ - \alpha SZ \tag{9}$$

$$R' = \delta S + \alpha SZ \tag{10}$$

Equations (8), (9), and (10) can be combined to satisfy

$$(11) \quad S' + Z' + R' = \pi$$

which leads to

$$(12) \quad S + Z + R = \infty$$

as times t approaches ∞ if $\pi \neq 0$.

If done on a short time scale, we can allow the birth rate to be zero. This would yield the following

$$(13) \quad S' = \beta SZ – \delta S = 0$$

$$(14) \quad Z' = \beta SZ – \alpha SZ = 0$$

$$(15) \quad R' = \delta S + \alpha SZ = 0$$

There are only two logically possible scenarios from this point - either $S = 0$ or $Z = 0$.

If $S = 0$, then there are only infected remaining, and we have an apocalypse.

$$(16) \quad (\bar{S}, \bar{Z}, \bar{R}) = (0, \bar{Z}, 0)$$

If $Z = 0$, then only the susceptible remain, and the infection is defeated.

$$(17) \quad (\bar{S}, \bar{Z}, \bar{R}) = (N, 0, 0)$$

Using the Jacobian, we are able to calculate the rate of change of the multivariable system as follows[4]:

$$(18) \quad J = \begin{bmatrix} -\beta Z - \delta & -\beta S & 1 \\ \beta Z - \alpha Z & \beta S - \alpha S & 1 \\ \delta + \alpha Z & \alpha S & 1 \end{bmatrix}$$

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For the disease-free equilibrium we have

$$(19) \quad J(N,0,0) = \begin{bmatrix} 0 & -\beta S & 1 \\ 0 & \beta S - \alpha S & 1 \\ 0 & \alpha S & 1 \end{bmatrix}$$

This will yield eigenvalues such that $det(J - \lambda I) = -\lambda[\lambda^2 + [1 - (\beta N - \alpha N)]\lambda - \beta N]$. Since the values of these eigenvalues are always positive, the system will be unstable.

For the infection-free equilibrium we have

$$(20) \quad J(0,\bar{Z},0) = \begin{bmatrix} -\beta Z - \delta & 0 & 1 \\ \beta Z - \alpha Z & 0 & 1 \\ \delta + \alpha Z & 0 & 1 \end{bmatrix}$$

This will yield eigenvalues such that $det(J - \lambda I) = -\lambda([\beta Z - \delta] - \lambda)(1 - \lambda)$. Since these values are always negative, the system will be asymptotically stable.

In qualitatively analyzing the Jacobian we have learned a great deal about the two equilibrium points. The equilibrium point of the disease-free Jacobian was unstable, and the equilibrium point of the human-free Jacobian was asymptotically stable. Thus, it can be derived that human survival would be impossible under the conditions that we have created.

[3]The third column is a series of 0's, but we will use 1's as a stabilizing factor in order to compute the Jacobian.

Part VII

Discussion

If the fate of the human population was based on the results of the disease model that we created, then our final days would be upon us. These results created, however, are not completely decisive. There are several different parameters that can be included into the system of equations that may possibly slow the rate at which the susceptible are infected with the contagion.

In order to more accurate depict the results of an epidemic disease such as the one described, several other factors must be included. First, this disease that we analyzed was assumed to have a negligible latent infection period, immediately moving the susceptible into the infected group. Should a significant latent period of infection be present in the actual disease, this would lower the rate at which the human population is affected. Next, it can be assumed with reason that if the infection occurred over a period of time in which birth rate becomes significant, then the rate at which the infection overcomes the population would be lower, although not by much.

The most important factors that must be considered in a logical epidemic outbreak are government and military interventions. It can be assumed that significant efforts would be taken in order to ensure that anyone who has become infected or interacted with the infected would be secluded into a quarantine zone. Moreover, if those who were infected displayed notable signs of aggression that caused harm to other individuals, the most likely governmental response would be military based. If the military was used to fight back masses of infected, then it is possible that human infection rate would substantially lower. The final scenario that must be included into the discussion of lethal pandemic outbreak is immediate destruction of the infected area. This would ensure than anyone infected would be removed provided action is taken immediately.

Part VIII

Conclusion

Although it is impossible to perfectly analyze how a population will react to an epidemic disease spread, differential equations can be a useful tool to approixmate how the systems will change. The Kermack-McKendrick model, although useful for certain models, is ineffective when lethal diseases are introduced into a system. When death is introduced, the model used by Munz, et al is most effective. However, these models must be adjusted so that they can more accurately represent the population that is effected by including quarintines, latent periods of infection, and other parameters that are not represented in the SZR model of disease spread. Most importantly, there are several parameters that may not be able to be accounted for in these models such as determination and love. These system that are created only account for how humans are supposed to act. However, it can be reasonably assumed that humans do not always act the way the are supposed to - especially when love is introduced.

Without a means of representing these parameters, we are left with our best approximations. If a disease were to develop with the properties that we created then based on this model, human survival would be impossible. The only humans that would remain on earth would be those that were infected. In the end, even these individuals would die from complications of the parasitic contagion and only the corpses would remain. The end of human existence would begin with a tiny spore, but it will be the brainwashed infected working under the puppeteering hands of the parasite that causes man to end his own species with mindless agressive attacks against his own kind. In a sense, it is the walking dead who end the world.

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