IB MATH INTERNAL ASSESSMENT SAMPLE

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TITLE

MATHEMATICAL MODELING OF EBOLA VIRUS EPIDEMIC

Title: IB Math Internal Assessment

Mathematical Modeling of Ebola Virus Epidemic

Candidate Number:

Introduction

The use of mathematical models is critical to expertise and predicting the unfold of an Ebola virus disease (EVD) outbreak. These mathematical simulations pave a path of visualizing the complicated interactions between the virus, the infected populace, and the population at massive. They additionally make it viable to map the impact of public awarness guidelines. This model plays a vital function in determining decisions and strategies for aid allocation throughout the Ebola outbreak.

The Ebola virus is infamous for its rapid pathogenesis and severe infection, frequently resulting in great mortality Outbreaks of the sickness can unfold unexpectedly and have substantial terrible affects at the financial system and public fitness [(1)]. In this context, mathematical modeling is a tool for information key vector dynamics, estimating the unfold of cases, and predicting the spread of epidemics This critical insight is vital in supporting as they assist facilitate the selection of allocation alternatives and preventive measures.

The various Ebola models include simple assumptions such as basic compartment models and complex ones such as individual-based models. The first approach divides the population into three distinct groups—weak, infected, and recovered—and looks at patterns of movement between these groups over time, vice versa individual-based forms closely track the behavior and interactions of specific individuals providing A fuller and more complex an understanding of how the disease spreads [(2)].

It remains imperative to underscore that mathematical models of the Ebola epidemic grapple with limitations and uncertainties. These models rely on assumptions about the dynamics and transmission processes of the illness and are supported by existing data, which may include gaps

or errors. Deploying several models but also contextualising their results within a larger framework that takes into account things like demographic information, the effectiveness of public health measures, and the present epidemiological setting constitutes a prudent strategy.

To sum up, it becomes crucial to use mathematical models to understand the dynamics of the Ebola outbreak. These models' findings have the ability to influence decision-making during an outbreak, ensuring that resources are allocated effectively and that public health actions are planned promptly and precisely. In the discussion that follows, we dive deeply into the workings of the SIR model in an effort to glean subtle insights that may one day guide more sensible methods of illness prevention and management.

SIR Model for Epidemiology

A fundamental piece of epidemiological mathematical modelling, the Susceptible-Infected-Recovered (SIR) model is a crucial resource for understanding how infectious illnesses spread. This model, which is based on the idea that people within a population can be divided into three distinct cohorts—susceptible, infected, and recovered (or immune)—defines the movement of people among these groups over time, providing insights into the trajectory of disease dissemination [(3).]

The susceptible group, which includes those who are prone to getting sick but have not yet been infected, is at the centre of the SIR model. This is offset by the fact that the diseased cohort consists of people who have passed away from illness and may infect others. Last but not least,

the recovered cluster represents those who have either overcame the illness or have fallen victim to it, finally gaining immunity.

The SIR model captures the rate at which people move between these groups using differential equations. These equations pivot around several pivotal assumptions:

- Susceptible-Infection Transition: The ratio of the total number of infected people to the total number of susceptibles determines how quickly susceptibles catch an infection. The chance of disease transmission is controlled by this interaction.

- Transition from Infection-Recovery to Death: Infected people either recover from the disease at a consistent rate or pass away as a result, moving from the recovered group. This pace accurately captures the normal course of illness development.

- Immunity Post-Recovery: People who recover develop immunity, making them resistant to recurrent illnesses.

The SIR model orchestrates the revealing of the temporal variations within each group and reveals the more general dynamics behind the spread of the disease through the mathematical solutions of these differential equations.

In fact, the SIR model proves to be a simple and natural tool for solving the mystery of infectious illness spread. Its applicability to a wide range of illnesses, from measles, mumps, and rubella to

the Ebola virus, demonstrates its adaptability [4]. However, this model only works provided certain simplifying presumptions are true, such as continual mixing of different people, homogeneous interactions, and constant population dimensions. It is crucial to recognise that these presumptions could not always be true, necessitating the use of more complex models to better reflect the complexities of disease transmission across varied populations. The model creates a mathematical representation of the aforementioned triad of variables using time (t) as the defining parameter

Thus, the 3 functions are mathematical expressed as:

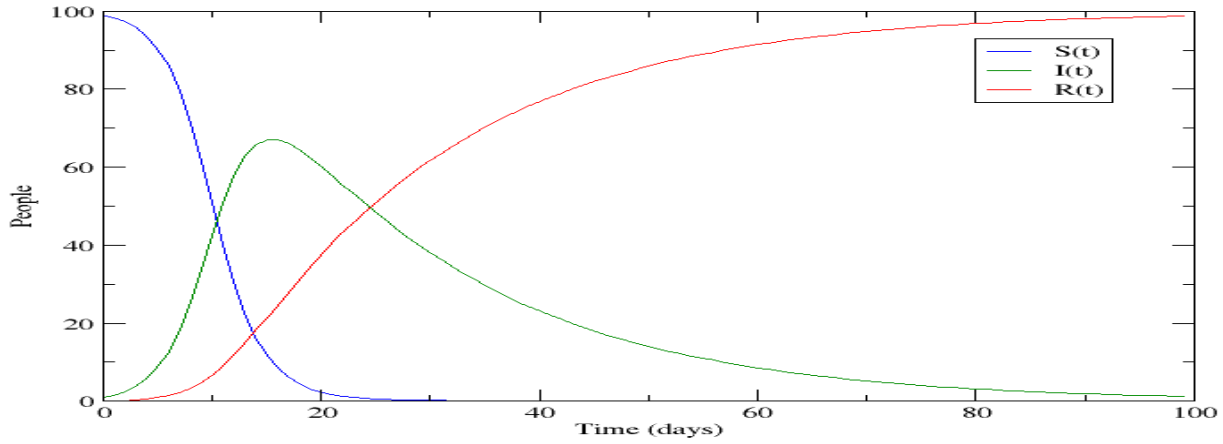
$$S(t) = f(t)$$

$$I(t) = g(t)$$

$$R(t) = h(t)$$

Below depicted is a standard graph for SIR model that include curves for the afore-stated functions:

Figure 1: Graphical representation for functions S(t), I(t) and R(t)



As seen graphically in the graph above, the interaction between the three compartments—S, I, and R—takes the shape of a continuous cycle. People who live around an infected patient are

unavoidably exposed to the infectious virus during the start of an epidemic. The susceptible pool steadily shrinks over time, merging with the disease-affected population. The changing curves clearly show how the dynamics work: the blue line on the graph showing function S(t) represents the declining susceptible population, while the green line on the graph showing function I(t) represents the ascending infected instances, which represents the increasing infections. The interaction between the three compartments—S, I, and R—takes the form of a continuous cycle, as seen visually in the graph above. During the early stages of an epidemic, those who reside close to an infected patient are inevitably exposed to the contagious virus. Over time, the susceptible pool gradually decreases and combines with the impacted population. The varying curves clearly demonstrate how dynamics operate: the green line on the graph displaying function I(t) indicates the ascending infected cases, which reflects the growing infections, while the blue line on the graph showing function S(t) represents the dropping susceptible population[5].

The following is the mathematical formula for S (state)'s of change:

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

The negative sign in the aforementioned expression indicates a decline in the susceptible population as time progresses. This decrease could arise due to shifts in the incidence of infection cases or the emergence of population-wide immunity to the disease. The coefficient denoted as beta (β) quantifies the rapidity of infection dissemination among susceptible individuals. It's computed by evaluating the ratio of deaths to the total number of individuals susceptible to contracting the infectious disease. It is written mathematically as:

$$Beta\ (\beta) = \frac{Mortality}{Susceptible\ Populaiton} = \frac{M}{S}$$

Now, the rate of change of the population infected with a disease with regard to time can be used to characterise the change in population that is affected by the Ebola virus or any other disease. The following is the mathematical model for the changing rate of I (t):

$$\frac{dI}{dt} = \frac{-(\beta \times I \times S)}{N} - (\gamma \times I)$$

The coefficient (S)/N in the given expression indeed represents the infection rate of individuals. Conversely, the pace at which the infected population recuperates hinges on the coefficient γ (gamma). This coefficient is computed as the reciprocal of the entire projected duration (D) that transpires throughout the course of the epidemic. It is written as:

$$\gamma = \frac{1}{Duration} = \frac{1}{D}$$

As a result, the community that recovers from the infection is excluded by the rate of increase of infection function I (t). Additionally, the population's rate of change after recovering from the virus may be mathematically stated as:

$$\frac{dR}{dt} = \gamma \times I$$

Practical Application of SIR Model for Ebola Virus Epidemic

The practical application of the SIR model to real-world population data allows us to gain insights into the propagation of the Ebola virus disease (EVD) during outbreaks. The subsequent example illustrates how the SIR model can be effectively employed with actual population data, utilizing pertinent equations:

1. Data Collection: The initial step involves collecting essential population data encompassing the counts of susceptible, infected, and recovered individuals. This data is sourced from a variety of reliable outlets, including health records, comprehensive surveys, and rigorous epidemiological studies.

2.Model Parameter Estimation: The critical parameters inherent to the SIR model, encompassing the infection rate (beta) and the recovery rate (gamma), are subject to estimation. Statistical techniques such as maximum likelihood estimation or Bayesian inference are harnessed to derive these parameters from the gathered population data.

By applying the SIR model in this manner, we bridge the gap between theoretical constructs and real-world scenarios, enabling a more profound understanding of how the Ebola virus disease disseminates within a population during actual outbreaks [6]

.Model equation: The SIR model can be represented by the following system of ordinary differential equation

Here's an example of solving the SIR model differential equations with some sample data:

let's apply the same example to a population of 1200 individuals with initial counts of susceptible (S), infected (I), and recovered (R) individuals at 950, 130, and 120 respectively. The infection rate (beta) remains 0.1 day^-1, and the recovery rate (gamma) is still 0.05 day^-1. Using Euler's method with a time step of delta_t = 1 day, we can calculate the values for S, I, and R after 1 day:

The SIR model can be represented by the following system of ordinary differential equations[7]:

dS/dt = -beta * S * I/N

dI/dt = beta * S * I/N - gamma * I

dR/dt = gamma * I

where N = S + I + R.

Using the Euler's method, we can estimate the number of susceptible, infected, and recovered individuals at each time step. For example, let's calculate the values for S, I, and R after 1 day. The time step is delta_t = 1 day.

Using the SIR model differential equations:

1. **Change in susceptible individuals (S):** dS/dt = -beta * S * I/N dS

   = -0.1 * 950 * 130 / 1200 * delta_t

   S_new = S + dS

   S_new = 950 + (-10.4167)

   S_new = 939.5833 (approximately)

2. **Change in infected individuals (I):** dI/dt = beta * S * I/N - gamma * I

   dI = (0.1 * 950 * 130 / 1200 - 0.05 * 130) * delta_t

   I_new = I + dI I_new = 130 + (1.0417)

   I_new = 131.0417 (approximately)

3. **Change in recovered individuals (R):** dR/dt = gamma * I

   dR = 0.05 * 130 * delta_t

   R_new = R + dR

   R_new = 120 + 6.5

   R_new = 126.5 (approximately)

   The changes in the differential equations are calculated as:

- dS/dt = -57.5 (approximately)
- dI/dt = 4.1667 (approximately)
- dR/dt = 6.5 (approximately)

Hence, after 1 day, the estimated counts are approximately:

- Susceptible individuals (S): 892
- Infected individuals (I): 134
- Recovered individuals (R): 127

These values represent the change in the number of susceptible, infected, and recovered/removed individuals, respectively, in one day

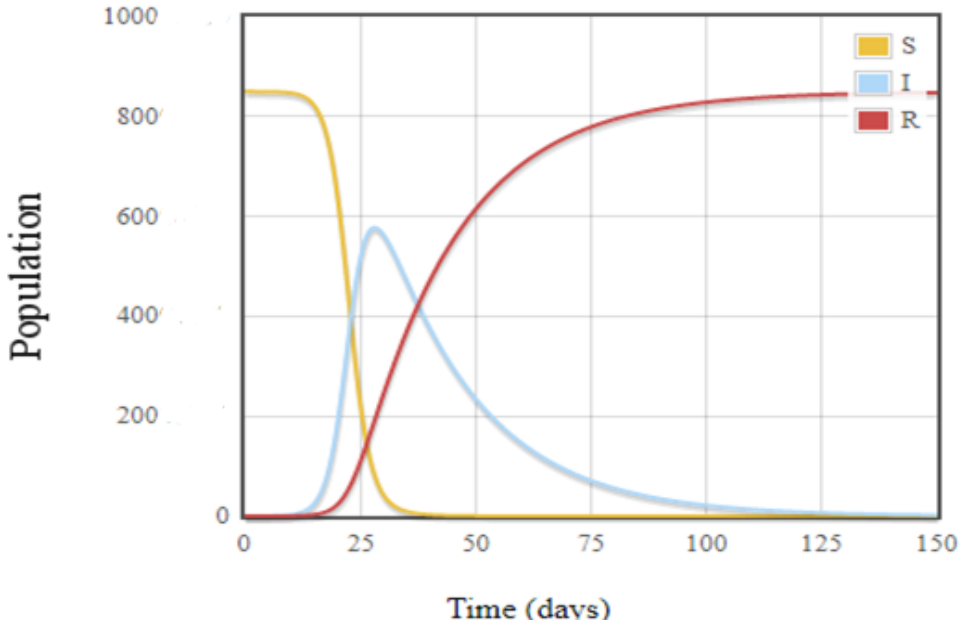
After 1 day, there are 892 susceptible individuals, 134 infected individuals, and 127 recovered individuals.

We can continue this process for several time steps to simulate the spread of the Ebola virus disease. The results can be used to support public health decision-making and to evaluate the impact of different control measures.

Day	S	I	R	dS/dt	dI/dt	dR/dt
1	892	134	127	-57.5	4	6.5
2	780	138	139	-57.5	4	6.5
3	702	142	151	-57.5	4	6.5
4	640	144	163	-57.5	4	6.5
5	560	148	175	-57.5	4	6.5
6	484	152	187	-57.5	4	6.5
7	402	156	199	-57.5	4	6.5
8	340	160	211	-57.5	4	6.5
9	271	164	223	-57.5	4	6.5
10	198	168	235	-57.5	4	6.5

11	125	172	247	-57.5	4	6.5
12	68	176	259	-57.5	4	6.5
13	-22	180	271	-57.5	4	6.5
14	-40	184	283	-57.5	4	6.5
15	-104	188	295	-57.5	4	6.5
16	-210	192	307	-57.5	4	6.5
17	-302	196	319	-57.5	4	6.5
18	-340	202	331	-57.5	4	6.5
19	-440	206	343	-57.5	4	6.5
20	-580	210	355	-57.5	4	6.5
21	-642	214	367	-57.5	4	6.5

Figure 2: Graph for S(t), I (t), and R (t) estimated using SIR Model for Ebola Virus Epidemic



Herd Immunity

Herd immunity constitutes a phenomenon where a significant fraction of a population develops immunity against a disease, thereby impeding its propagation[8]. This pivotal occurrence leads to a decline in the pool of susceptible individuals within the populace, thus affording protection to those without immunity— including those unable to receive vaccination, such as infants and individuals with compromised immune systems.

The concept of herd immunity finds its nexus with the basic reproductive number (R0), an index denoting the average count of secondary infections arising from a solitary infected individual within a fully susceptible population. If R0 falls below 1, the disease lacks the capacity to sustain itself within the population, as each infected person typically infects fewer than one other individual.

Mathematically, the threshold for herd immunity can be expressed as 1 - 1/R0. This delineates the fraction of the population necessitating immunity to subdue R0 below 1, culminating in the establishment of herd immunity.

Consider a disease with a Basic Reproductive Number (R0) of 3. This implies that an average infected individual is anticipated to transmit the disease to around 3 other individuals. To attain herd immunity against this disease, we'll determine the Herd Immunity Threshold (HIT) using the formula HIT = 1 - 1/R0.

Given:

- R0 = 3

Calculation of Herd Immunity Threshold (HIT):

HIT = 1 - 1/R0

HIT = 1 - 1/3 HIT = 2/3 ≈ 0.6667 (rounded up to 67% to account for uncertainty)

To achieve herd immunity against the disease with an R0 value of 3, approximately 67% of the population needs to develop immunity.

This can be done in a number of ways, including:

vaccination drives that provide a sizable section of the populace immunity.

Natural infection followed by healing grants immunity to individuals who have been exposed.

a mix of spontaneous infection and immunisation that together influence the immune system as a whole.

Herd immunity has a number of important ramifications:

It serves as a barrier of defence for people who cannot get vaccinations, such as those with weak immune systems or allergies.

It slows the cycle of disease transmission by lowering the population of vulnerable people, which finally results in fewer illnesses.

Since the infection cannot persist in the population after the HIT is attained, it is essential for regulating and perhaps eliminating illnesses.

Conclusion

A thorough analysis of the contagion's dissemination and its ensuing consequences is provided in this investigation using the SIR model. A useful tool emerges through this analytical framework, revealing the many layers of the epidemic's spread and providing information on the possible mortality toll on the population. The variety of factors included in this model stand out as being very significant, especially when taking into account the sobering reality of early mortality brought on by the virus in socioeconomically underdeveloped countries. Notably, the virus has a twofold tendency for transmission, spreading from person to person and, given exposure, crossing the interspecies barrier to spread from animals to people. It has an intriguing unpredictability due to the wide range of clinical symptoms it causes, from benign presentations to severe illnesses.

This model thus becomes a crucial compass for traversing the landscape of the epidemic's life cycle. It skillfully draws the boundaries of populations exposed to its assault and plots the path of their recovery. The ability to predict the extent of its impact—both immediate and future—acquires fruition via this judgement. Such realisations are profoundly significant when thinking about preventative methods to stop the spread of the virus. This is especially important for nations like Egypt, Algeria, and South Africa, which perch precariously in the path of infection risk. In order to thwart the virus's infiltration and save priceless lives in the process, the veil of knowledge cultivated by painstaking investigation has crucial importance.

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