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Abstract: In this paper, we studied the stability analysis of the SIR and SIS compartmental epidemiological models in view to finding out the dynamics of the spread of infectious diseases. This work further discusses the general SIR and SIS epidemic model (with vital dynamics); we also obtained the disease free equilibrium and the endemic points. Furthermore, stability analysis of both the disease-free and the endemic equilibrium were obtained accordingly.

Keywords: Disease, dynamics, endemic, epidemic, equilibrium, models

Introduction

The existences of communicable illnesses today, are reality of the modern-day lifestyles all dwelling organisms have come to terms with. As humans, urbanization and different elements which are making our existence less complicated to stay has in a way emerge as the predominant reasons of illnesses which are capable of wiping out a population or causing unborn generations to be infected with it. According to Riedel (2005) infectious diseases are caused by pathogenic microorganisms, such as bacteria, viruses, fungi and parasites and these diseases can spread directly or indirectly from one person to another or from animals/birds to humans. For example, the recent surge of the novel Coronavirus has brought huge economic and societal implications. According to the World Health Organization (WHO) Director General Mandate is public health, but we're working with many partners across all actors to mitigate the social and economic consequences of this pandemic. He also noted that; this is not just a public health crisis. It's a crisis that will touch every sector, therefore every sector and individual must be involved in the fight.

Mathematical modeling has become an invaluable tool to understand the dynamics of infectious disease and to support the development of control strategies. Kermack *et al.* (1927) in their first paper titled contributions to the mathematical theory of epidemics (part I) started with the assumption that all members of the community are initially equally susceptible to the disease, and that a complete immunity is conferred after the infection. The population is divided into three distinct classes: the susceptible (S) (individuals who potentially open to the disease), the infected (I) (those who have the disease and can transmit it) and the removed (R) (individuals who have had the disease and are now immune to the infection) or removed from further propagation of the disease by some other means. Talawar (2008) included a new class of immunized (vaccinated) individuals in SIS endemic model. He obtained that even at smaller values of ϕ (immunization rate) the larger fraction of susceptible population can be protected and number of infected individuals can be reduced to a great extent.

According to the Center for Disease Control and Prevention, epidemics occasionally comes omitted until large quantity of people displays comparable signs pronounced through public health agencies of which may have claimed a lot of lives and probably can't be contained for the reason that it has extensively invaded a massive number of people in the population typically referred to as the infectious class. The novel coronavirus is a classic example, On the 11th March 2020, the WHO Director-General "rang the alarm bell loud and clear" by calling COVID-19 a pandemic. The effective

contact rate can generally impact on the spread of the infectious disease. Olaniyi *et al.* (2014), research work majoring on a deterministic epidemiological model describing the spread of infectious disease characterized by pseudo-recovery due to incomplete treatment, discovered that increasing the value of any of the parameters such as effective contact rate, pseudo-recovery rate increases the basic reproduction number, and the magnitude of the infectious individuals in the community increases accordingly. Conversely, increasing the value of either death rate or recovery rate decreases the basic reproduction number and the magnitude of the infectious individuals in the community decreases accordingly.

In this light, evaluating inoculation (by vaccination and others) or isolation plans will further have significant effect on the mortality rate of a particular epidemic. Farrington (2003) studied the impact of vaccination program on the transmission potential of the infection in large populations. He also obtained the relationship between vaccine efficacy against transmission, reproduction number and vaccine coverage. Stability evaluation of compartmental models in epidemiology has been used to furnish an understanding of the underlying mechanisms that govern the emergence of infectious diseases and in the procedure it suggests management strategies for the eradication of these ailments. This broad principle, constant with observations and quantified by means of epidemiological models, has been persistently used to estimate the usefulness of vaccination policies and the prospect that a disease may additionally be eliminated or eradicated.

Statement of the Problem

The primary hazard to human existence today is diseases. It can successfully wipe out a populace or cause unborn generations to be carrier or contaminated with it. These illnesses typically come as an epidemic. The Center for Disease control and Prevention defines Epidemic as an increase, often sudden in the number of cases of a disease above what is normally expected in that area. Epidemic usually affects enormous number of individuals and can result to complications that include disabilities and even death. Epidemic situations also deteriorate the already overburdened health services, as the scares available resources have to be diverted for controlling and management of epidemics. For instance, during a sudden outbreak of an epidemic, social and political tension arises from the spread thus hindering economic activities of that particular society lacking strong and healthy working population due to the infection. The efficacy and productivity of a society, region or country invaded by epidemics usually have a descending slide resulting from scarcity of clean food and water, hence increasing starvation, death and leaving behind weak and

sickly population which are neither helpful to themselves and the society at large.

However, to counter these setbacks the role of mathematical epidemiology is to model the establishment and spread of the epidemics. A commonly effective method of doing so is to use the notion of selecting the population into compartments under certain assumptions, which represent their health status with respect to the pathogen in the system. Kermack and MC Kendrick in the early 1900s successfully developed an approach to this model. These models are known as compartmental models in epidemiology, and serve as a base mathematical framework for understanding the complex dynamics of these systems.

Objectives of the Study

This research work aims at finding out the stability analysis of the SIR and SIS compartmental models with vital dynamics with the following objectives

- i. To determine the disease free equilibrium and the endemic equilibrium points
- ii. To have a general look at stability analysis of these models majoring on the local stability, at disease-free equilibrium and the endemic equilibrium point.

Materials and Methods

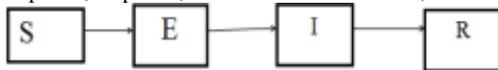
Compartmental models

Compartmental Models are made up of a finite number of homogenous well blended sub systems called compartments which interacts with each other and with the surroundings so that the concentration of materials inside each compartment may additionally be described via a First Order Differential Equation. Compartmental models are divided according to; One compartmental modeling and two or multi-compartmental modeling

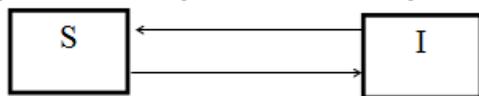
Some examples of epidemiological compartmental models

Many mathematical models with regards to epidemiology are important equipment in analyzing the expansion control of infectious diseases. The model formation technique clarifies assumptions, variables and parameters used in offering conceptual result such as the disease free equilibrium point and endemic equilibrium points thereby understanding the transmission traits and dynamics of infectious disease. The following are some examples of compartmental mode

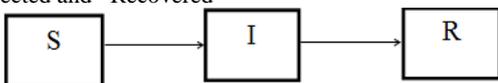
- i. **SEIR Model:** This model basically addresses diseases that allow a window period referred as latent period (period when infected is not infectious). This population is divided into four compartment namely Susceptible, Exposed, Infected and Recovered;



- ii. **SIS Model:** This model studies the disease that doesn't confer lasting immunity to the once infected individual e.g. common cold, STDs, etc. It divides population into Susceptible and infected compartment.



- iii. **SIR Model:** This is used to study disease in which the infected individual may recover i.e. which confers immunity against re-infection. This model divides the population into three compartments; Susceptible Infected and Recovered



These compartments interact homogeneously with each other at a particular time

The SIR model with vital dynamics

The SIR model in epidemiology gives a simple dynamic description of the three interacting population consisting of;

- i. **Susceptible.** Individuals that are not infected yet, but have the tendency or likelihood of been infected provided all condition of being infected is satisfied. it is denoted by **S**
- ii. **Infected.** Individuals infected by the disease (casual organism). Denoted by the symbol **I**.
- iii. **Recovered.** Individuals that have returned to normal state of health after they have been infected. Denoted by **R**

The SIR model discussed here put into consideration the vital dynamics (birth rate and death rate) since over a period of time this two factors determine the stability of the model. In spite of its simplicity, the SIR model exhibits the basic structure generally associated to the spread of a disease in a population. The way these compartments interact is often based on assumptions, and the model is built up from there. These models are usually investigated through Ordinary Differential Equations (which are deterministic).

Variables of the model

- i. **S (t)** denotes the number of individuals who are susceptible to the disease, that is, who are not (yet) infected at time **t**.
- ii. **I (t)** denotes the number of infected individuals, assumed infectious and able to spread the disease by contact with susceptible.
- iii. **R (t)** denotes the number of individuals who have been infected and then removed from the possibility of being infected again or of spreading infection. Removal is carried out either through isolation from the rest of the population, through immunization against infection or through recovery from the disease with full immunity against reinfection or through death caused by the disease.

Assumptions of the SIR model with vital dynamics

This model is appropriate under the following assumptions:

- i. The population is fixed.
- ii. The only way a person can leave the susceptible group is to become infected. Once a person has recovered, the person received immunity.
- iii. Age, sex, social status, and race do not affect the probability of being infected.
- iv. There is no inherited immunity.
- v. The member of the population mix homogeneously (have the same interactions with one another to the same degree).
- vi. The natural birth and death rates are included.
- vii. All births are into the susceptible class.
- viii. The death rate is equal for members of all three classes, and it is assumed that the birth and death rates are equal so that the total population is stationary

Model Formulation

To understand the SIR model the basic notations are given below;

The **SIR model** labels these three compartments

S (t) = number of susceptible at time **t**

I (t) = number of infectious, at time **t**

R (t) =number recovered (immune) at time **t**.

N= total population size

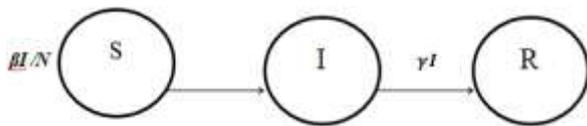


Fig. 1: Flow chart for the SIR model

From the above, we have the following system of differential equations,

$$\frac{dS}{dt} = \mu N - \beta I(t)S(t) - \mu S(t) \dots\dots\dots (1)$$

$$\frac{dI}{dt} = \beta I(t)S(t) - \gamma I(t) - \mu R(t) \dots\dots\dots (2)$$

$$\frac{dR}{dt} = \gamma I(t) - \mu R(t) \dots\dots\dots (3)$$

Where: β = effective contact rates). This refers to the rate of transition from S and I; μ = death and birth rate (which are assumed to be equal); γ = recovery rate of the infected Since birth rate is equal to the death rate the population is constant thus;

$$S(t) + I(t) + R(t) = N$$

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$$

We can consider the prevalence i.e. the proportions by redefining;

$$s = \frac{S}{N} \text{ \{proportion of the susceptible in the entire population\} } \dots\dots (4)$$

$$i = \frac{I}{N} \text{ \{proportion of the infected individuals in the entire population\} } \dots\dots\dots (5)$$

$$r = \frac{R}{N} \text{ \{proportion of the recovered or removed individuals in the entire population\} } \dots\dots\dots (5^*)$$

now substituting the above into equations (1), (2) and (3) for simplification.

Knowing that,

$$\frac{ds}{dt} = \frac{d}{dt} \left[\frac{S}{N} \right] = \frac{1}{N} \left[\frac{dS}{dt} \right] \dots\dots\dots (6)$$

$$\text{Substituting (1) into the above equation (6) we have,}$$

$$= \frac{1}{N} [\mu N - \beta S \frac{I}{N} - \mu S(t)] \dots\dots\dots (7)$$

Expanding the above equation we have;

$$\frac{1}{N} \left[\mu N - \frac{1}{N} \beta S \frac{I}{N} - \frac{1}{N} \mu S \right]$$

Multiply through by N and using equation (4) and (5) we have

$$\left[\mu \frac{N}{N} - \frac{1}{N} \beta \left[\frac{S}{N} \right] \left[\frac{I}{N} \right] - \frac{1}{N} \mu \frac{S}{N} \right]$$

$$\frac{ds}{dt} = \mu - \beta i s - \mu s \dots\dots\dots (8)$$

Similarly we get for the other two compartment.

Knowing that;

$$\frac{dI}{dt} = \frac{d}{dt} \left[\frac{I}{N} \right] = \frac{1}{N} \frac{dI}{dt}$$

Substituting equation 2 into the above equation (9) we have;

$$\frac{1}{N} [\beta I S \frac{I}{N} - \gamma I - \mu R] \dots\dots\dots (8)$$

Expanding the above equation we have;

$$\frac{1}{N} \left[\beta I S \frac{I}{N} - \gamma \frac{I}{N} - \frac{S}{N} \mu \right]$$

Multiply through by N and using equation (4) and (5) we have;

$$\left[\beta I \frac{S}{N} \frac{I}{N} - \gamma \frac{I}{N} - \frac{S}{N} \mu \right]$$

$$\frac{dI}{dt} = \beta i s - \gamma i - \mu i \dots\dots\dots (10)$$

Similarly following the above process once again we have

$$\frac{dR}{dt} = \gamma i - \mu R \dots\dots\dots (11)$$

The resulting system of differential equation is given as follow;

$$\frac{ds}{dt} = \mu - \beta i s - \mu s$$

$$\frac{dI}{dt} = \beta i s - \gamma i - \mu i$$

$$\frac{dR}{dt} = \gamma i - \mu R$$

Considering the entire the population;

$$s(t) + i(t) + r(t) = 1$$

With initial condition as $s(0) \geq i(0) \geq r(0) \geq 0$

The equation has the set positively invariant $\Omega\{s(t), i(t) \in \mathbb{R}^2, s(t), i(t) \leq 1\}$

Equilibrium Points of the SIR Model

Disease free equilibrium point

The Disease Free Equilibrium (DFE) is the state where disease do not exist or has been totally eradicated or wiped out from the population. At the DFE point the infected and recovered class is usually empty.

Given that;

$$s(t) + i(t) + r(t) = 1$$

With initial condition at $t = 0$

$$S(0) = s_0, \quad i(0) = i_0, \quad r(0) = r_0$$

This implies;

$$\text{At } t = 0; \quad s(t) = 1 - i(t) - r(t);$$

$$S_0 = 1 - 0 - 0$$

$$S_0 = 1$$

Similarly;

$$\text{At } t = 0; \quad i(t) = 1 - s(t) - r(t)$$

$$i_0 = 1 - 1 - 0$$

$$i_0 = 0$$

Finally;

$$\text{At } t = 0; \quad r(t) = 1 - s(t) - i(t)$$

$$r_0 = 1 - 1 - 0$$

$$r_0 = 0$$

Therefore the DFE point is $E^0(s_0, i_0, r_0) = (1, 0, 0)$

Endemic equilibrium

The endemic equilibrium state is the state where the disease cannot be totally eradicated but remains in the population. For the disease to persist in the population the compartments or Classes must not be zero at equilibrium state. In other words, if it is the endemic equilibrium state, then $E(S^*, I^*, R^*) \neq (0, 0, 0)$.

From the resulting system of differential equation we have;

$$\mu - \beta i s - \mu s = 0 \dots\dots\dots (12)$$

$$\beta i s - \gamma i - \mu i = 0 \dots\dots\dots (13)$$

$$\gamma i - \mu r = 0 \dots\dots\dots (14)$$

from equation (14)

$$\gamma i - \mu r = 0$$

$$\text{this implies} \quad i = \frac{\mu r}{\gamma} \dots\dots\dots (15)$$

Substituting equation 15 into equation 13

$$\beta s \left[\frac{\mu r}{\gamma} \right] - \gamma \left[\frac{\mu r}{\gamma} \right] - \mu \left[\frac{\mu r}{\gamma} \right] = 0 \quad \text{expanding gives}$$

$$\frac{\beta s \mu r}{\gamma} = \frac{\gamma \mu r}{\gamma} + \frac{\mu^2 r}{\gamma}$$

Dividing both side by $\frac{\beta s \mu r}{\gamma}$ to make s subject of formulae

$$s = \frac{\gamma \mu r + \mu^2 r}{\gamma} \times \frac{\gamma}{\beta \mu r}$$

$$s = \frac{\gamma \mu r + \mu^2 r}{\beta \mu r} \quad \text{factoring } \mu \text{ we have}$$

$$s^* = \frac{\gamma r + \mu r}{\beta r} \dots\dots\dots (16)$$

$$\text{Implies } s^* = \frac{\gamma + \mu}{\beta}$$

now substituting (16) and (15) into (12)

$$\mu - \beta \left[\frac{\mu r}{\gamma} \right] \left[\frac{\gamma r + \mu r}{\beta r} \right] - \mu \left[\frac{\gamma r + \mu r}{\beta r} \right] = 0$$

$$\mu - \left[\frac{\beta \mu r^2 (\gamma + \mu)}{\beta r \gamma} \right] - \mu r \left[\frac{\gamma + \mu}{\beta r} \right] = 0$$

$$\mu - \left[\frac{\mu r (\gamma + \mu)}{\gamma} \right] - \mu \left[\frac{\gamma + \mu}{\beta} \right] = 0$$

Making r subject formulae

$$- \left[\frac{\mu r (\gamma + \mu)}{\gamma} \right] = - \mu + \mu \left[\frac{\gamma + \mu}{\beta} \right]$$

Now dividing both side by - $\frac{\mu(\gamma + \mu)}{\gamma}$

$$r = \left[-\mu + \frac{\mu(\gamma + \mu)}{\beta} \right] \times \frac{\gamma}{\mu(\gamma + \mu)}$$

$$= \frac{-\mu\gamma}{\mu(\gamma + \mu)} + \frac{\gamma\mu(\gamma + \mu)}{\beta\mu(\gamma + \mu)}$$

Resolving to a single fraction we have

$$\frac{-\mu\gamma(\beta\mu(\gamma + \mu)) + \gamma\mu(\gamma + \mu)\mu(\gamma + \mu)}{\mu(\gamma + \mu)\beta\mu(\gamma + \mu)}$$

Now factoring $\mu(\gamma + \mu)$ we have

$$\frac{-\beta\mu\gamma + \mu\gamma(\gamma + \mu)}{\beta\mu(\gamma + \mu)}$$

Again factoring μ we have

$$r^* = \frac{-\beta\gamma + \gamma(\gamma + \mu)}{\beta(\gamma + \mu)}$$

$$r^* = \frac{\gamma(-\beta + \gamma + \mu)}{\beta(\gamma + \mu)} \quad (18)$$

Now since we have obtain r^* in (18) we substitute into (15)

$$I^* = \frac{\mu r^*}{\gamma}$$

this implies

$$i^* = \frac{\gamma\mu(-\beta + \gamma + \mu)}{\beta\gamma(\gamma + \mu)}$$

Therefore

$$i^* = \frac{\mu(-\beta + \gamma + \mu)}{\beta(\gamma + \mu)} \quad (19)$$

hence, endemic equilibrium points are

$$E^*(s^*, i^*, r^*) = \left[\frac{\gamma + \mu}{\beta}, \frac{\mu(-\beta + \gamma + \mu)}{\beta(\gamma + \mu)}, \frac{\gamma(-\beta + \gamma + \mu)}{\beta(\gamma + \mu)} \right] \quad (20)$$

The SIS model with vital dynamics

The SIS model is a model used in studying the dynamics of disease that does not confer lasting immunity (e.g. gonorrhoea) or recovery to the infected individuals in a population. To study the stability of the SIS model it is necessary to consider the inflow of new births into the susceptible class and the death removal from both the susceptible and infected compartment. The death and birth rate are assumed to be equal so the population is constant.

Model Formulation

To formulated model is given by the following set of differential equation

$$\frac{dS}{dt} = -\beta S(t) \frac{I}{N} + \gamma \frac{I}{N} + \mu N - \mu S(t) \quad (1)$$

$$\frac{dI}{dt} = \beta IS(t) \frac{I}{N} - \gamma I(t) - \mu I(t) \quad (2)$$

Given initial condition

$$S(0) = S_0 \quad I(0) = I_0$$

Where: μ – proportional constant called removal rate

$\frac{1}{\mu}$ = Average life expectancy

μN = rate of inflow of new borns into susceptible compartment

The total population density is given thus since the population is constant

$$S(t) + I(t) = N$$

To simplify the above model or system of differential equations

$$s = \frac{S}{N} \quad \left\{ \text{proportion of the susceptible in the entire population} \right\} \dots \dots \dots (4)$$

$$i = \frac{I}{N} \quad \left\{ \text{proportion of the infected individuals in the entire population} \dots \dots \dots (5) \right.$$

from equation (1) and (2)

now dividing through by N it results to

$$\frac{ds}{dt} = -\beta \frac{s}{N} \frac{i}{N} + \gamma \frac{i}{N} + \mu \frac{N}{N} - \mu \frac{s}{N} \dots \dots \dots (6)$$

$$\frac{di}{dt} = \beta \frac{s}{N} \frac{i}{N} - \gamma \frac{i}{N} - \mu \frac{i}{N} \dots \dots \dots (7)$$

By equations (4), (5) and (3) for simplification, we get

$$\frac{ds}{dt} = -\beta si + \gamma i + \mu - \mu s \dots \dots \dots (8)$$

$$\frac{di}{dt} = \beta si - \gamma i - \mu i \dots \dots \dots (9)$$

Equilibrium Points of the SIS Model

Disease free equilibrium point

Since the population is constant

$$S(t) + I(t) = 1$$

$$s(t) = 1 - I(t)$$

At $t=0$

$$S(0) = 1 - I(0)$$

$$S_0 = 1 - 0 = 1$$

Also

$$I(t) = 1 - s(t)$$

At $t=0$

$$I(0) = 1 - S(0)$$

$$I_0 = 1 - 1 = 0$$

The disease equilibrium point is $E^0(s_0, i_0) = (1, 0)$

Endemic equilibrium point

To find the endemic equilibrium point it is resolved as follows;

$$-\beta si + \gamma i + \mu - \mu s = 0 \dots \dots \dots (10)$$

$$\beta si - \gamma i - \mu i = 0 \dots \dots \dots (11)$$

From 11 it result to

$$\beta si - \gamma i - \mu i = 0$$

$$s = \frac{\gamma + \mu}{\beta} \dots \dots \dots (12)$$

for I

from 10

$$-\beta si + \gamma i + \mu - \mu s = 0$$

Making i the subject of formula by dividing both side by $\beta s + \gamma$ it results to

$$i = \frac{-\mu + \mu s}{-\beta s + \gamma} \dots \dots \dots (13)$$

Substituting 12 into 13

$$-\mu + \mu \left[\frac{\gamma + \mu}{\beta} \right] \div \left[-\beta \left[\frac{\gamma + \mu}{\beta} \right] + \gamma \right]$$

$$= \frac{-\mu\beta + \mu\gamma + \mu^2}{\beta} \times \left[\frac{\beta}{-\beta\gamma + \beta\mu + \beta\gamma} \right]$$

Factorizing μ result to

$$I = \frac{(-\beta + \gamma + \mu)}{-\beta}$$

the Endemic equilibrium points are

$$E^*(S^*, i^*) = \left(\frac{\gamma + \mu}{\beta}, \frac{(-\beta + \gamma + \mu)}{-\beta} \right)$$

The equilibriums points will now be use determine the stability of the model under consideration.

Results and Discussion

Stability of the SIR model

Stability at disease free equilibrium point

To determine the stability of the model at the disease free equilibrium point we use the Jacobin matrix;

$$J(s, i, r) = \begin{bmatrix} \frac{\partial S}{\partial s} & \frac{\partial S}{\partial i} & \frac{\partial S}{\partial r} \\ \frac{\partial I}{\partial s} & \frac{\partial I}{\partial i} & \frac{\partial I}{\partial r} \\ \frac{\partial R}{\partial s} & \frac{\partial R}{\partial i} & \frac{\partial R}{\partial r} \end{bmatrix}$$

At DFE equilibrium point $E^0(s_0, i_0, r_0) = (1, 0, 0)$

$$S = \mu - \beta i S - \mu S$$

$$I = \beta i S - \gamma i - \mu i$$

$$R = \gamma i - \mu r$$

Now partially differentiating according to the Jacobin matrix we have

$$J(s, i, r) = \begin{bmatrix} \beta i - \mu & -\beta s & 0 \\ \beta i & \beta s - \gamma - \mu & 0 \\ 0 & \gamma & -\mu \end{bmatrix}$$

The above 3x3 Jacobin matrix can be resolved to obtain the eigenvalues

Using $|A - \lambda I|$

$$\begin{bmatrix} \beta i - \mu & -\beta s & 0 \\ \beta i & \beta s - \gamma - \mu & 0 \\ 0 & \gamma & -\mu \end{bmatrix} - \lambda \begin{bmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix}$$

$$\begin{bmatrix} \beta i - \mu - \lambda & -\beta s & 0 \\ \beta i & \beta s - \gamma - \mu - \lambda & 0 \\ 0 & \gamma & -\mu - \lambda \end{bmatrix}$$

The characteristics polynomial followed by determinant

$$(\beta i - \mu - \lambda)(\beta s - \gamma - \mu - \lambda)(-\mu - \lambda) - 0$$

$$(\beta i - \mu - \lambda)[- \mu\beta s + \mu\gamma + \mu^2 + \mu\lambda - \lambda\beta s + \lambda\gamma + \lambda\mu + \lambda^2]$$

Opening the bracket we have

$$-\lambda[\lambda^2 + 2\mu\lambda + \lambda\gamma - \beta s\lambda + \mu^2 - \mu\beta s + \mu\gamma]$$

$$-\lambda^3 - 2\mu\lambda^2 - \gamma\lambda^2 + \beta s\lambda^2 - \mu^2\lambda + \mu\beta s\lambda - \mu\gamma\lambda$$

$$(1)$$

$$-\mu[\lambda^2 + 2\mu\lambda + \lambda\gamma - \beta s\lambda + \mu^2 - \mu\beta s + \mu\gamma]$$

$$-\mu\lambda^2 - 2\mu^2\lambda - \mu\gamma\lambda + \beta s\mu\lambda - \mu^3 + \mu^2\beta s - \mu^2\gamma$$

$$(2)$$

$$\beta i[\lambda^2 + 2\mu\lambda + \lambda\gamma - \beta s\lambda + \mu^2 - \mu\beta s + \mu\gamma]$$

$$\beta i\lambda^2 + 2\beta i\mu\lambda + \beta i\lambda\gamma - \beta^2 i s\lambda + \beta i\mu^2 - \beta^2 i\mu s + \beta i\mu\gamma$$

$$(3)$$

$$-\beta s [\beta i(-\mu - \lambda)] = \beta^2 i s\mu + \beta^2 i s\lambda$$

(4)

Combining (1), (2), (3) and (4)

$$= -\lambda^3 + [-2\mu - \gamma + \beta s + \beta i - \mu]\lambda^2 + [-\mu^2 + \mu\beta s - \mu\gamma - 2\mu^2 - \mu\gamma + \beta s\mu + 2\beta i\mu + \beta i\gamma - \beta^2 i s + \beta^2 i s]\lambda - \mu^3 + \mu^2\beta s - \mu^2\gamma + \beta i\mu^2 - \beta^2 i\mu s + \beta i\mu\gamma + \beta^2 i s\mu$$

$$= -\lambda^3 + [-2\mu - \gamma + \beta s + \beta i - \mu]\lambda^2 + [-\mu^2 + \mu\beta s - \mu\gamma - 2\mu^2 - \mu\gamma + \beta s\mu + 2\beta i\mu + \beta i\gamma]\lambda - u[\mu^2 - \mu\beta s + \mu\gamma + \beta i\gamma - \beta i\mu^2]$$

Multiplying through by minus we have;

$$\lambda^3 - [-2\mu - \gamma + \beta s + \beta i - \mu]\lambda^2 - [-3\mu^2 + 2\mu\beta s - 2\mu\gamma + 2\beta i\mu + \beta i\gamma]$$

$$+ u[\mu^2 - \mu\beta s + \mu\gamma + \beta i\mu - \beta i\gamma] \quad (5)$$

The above equation is the characteristic polynomial of the model

Hence, at Disease free equilibrium point equation (5) becomes The DFE point are $E(s, i, r) = (1, 0, 0)$

$$P(\lambda) = \lambda^3 - [-2\mu - \gamma + \beta - \mu]\lambda^2 - [-3\mu^2 + 2\mu\beta - 2\mu\gamma] + u[\mu^2 - \mu\beta + \mu\gamma] \quad (6)$$

For simplification we test for a factor of the cubic polynomial and thus reduce it to quadratic polynomial.

$$\text{Let } \lambda = -\mu \quad (7)$$

Substituting (7) into (6) we obtain

$$P(\lambda) = 0$$

Hence we conclude that $(\lambda + \mu)$ is a factor of the polynomial

$$\text{Therefore } \lambda_1 = -\mu$$

equation (6) is reduced to a quadratic equation by polynomial division

$$\lambda^2 - [-4\mu - \gamma + \beta]\lambda + [-\mu^2 + \mu\gamma - \mu\beta]$$

(8)

Now, to determine the stability we find the other two eigenvalues using

$$\lambda = \frac{-b \pm \sqrt{b^2 - 4ac}}{2a} \quad (8^*)$$

$$b = -[-4\mu - \gamma + \beta]$$

$$b^2 = 16\mu^2 + 8\mu\gamma - 8\mu\beta + \gamma^2 - 2\beta\gamma + \beta^2$$

$$4ac = 4[-\mu^2 + \mu\gamma - \mu\beta] = -4\mu^2 + 4\mu\gamma - 4\mu\beta$$

$$b^2 - 4ac = 16\mu^2 + 8\mu\gamma - 8\mu\beta + \gamma^2 - 2\beta\gamma + \beta^2 + 4\mu^2 - 4\mu\gamma + 4\mu\beta$$

$$= 20\mu^2 + 4\mu\gamma - 4\mu\beta + \gamma^2 - 2\beta\gamma + \beta^2$$

Substituting into (8*) we have

$$\lambda_{2,3} = \frac{[-4\mu - \gamma + \beta] \pm \sqrt{20\mu^2 + 4\mu\gamma - 4\mu\beta + \gamma^2 - 2\beta\gamma + \beta^2}}{2}$$

Therefore

$$\lambda = -\mu$$

$\lambda_{2,3}$

$$= \frac{[-4\mu - \gamma + \beta] \pm \sqrt{20\mu^2 + 4\mu\gamma - 4\mu\beta + \gamma^2 - 2\beta\gamma + \beta^2}}{2}$$

Considering $\lambda_1 = -\mu$, according to **theorem 1** we have that $\lambda_1 < 0$ satisfying the condition for stability and $\lambda_{2,3}$ similarly satisfies the condition for the stability if and only if the determinant of the function is less than zero giving rise to complex function. While considering the real $\text{Re}(\lambda_{2,3})$ part we observed that $\text{Re}(\lambda_{2,3}) \leq 0$ which satisfies the condition in **theorem 1**. Hence the model is said to be locally stable at the Disease free equilibrium point.

Stability at the endemic equilibrium point

Similarly, we look for the stability of the model at the Endemic equilibrium point. Hence, using equation (5) above

$$\lambda^3 - [-2\mu - \gamma + \beta s + \beta i - \mu]\lambda^2 - [-3\mu^2 + 2\mu\beta s - 2\mu\gamma + 2\beta i\mu + \beta i\gamma]$$

$$+ u[\mu^2 - \mu\beta s + \mu\gamma + \beta i\mu - \beta i\gamma] \quad (i)$$

Endemic equilibrium points are

$$E^*(s^*, i^*, r^*) = \left[\frac{\gamma + \mu}{\beta}, \frac{\mu(-\beta + \gamma + \mu)}{\beta(\gamma + \mu)}, \frac{\gamma(-\beta + \gamma + \mu)}{\beta(\gamma + \mu)} \right] \quad (ii)$$

Substituting (ii) into (5) above we have

$$\lambda^3 - [-2\mu - \gamma + \beta \left(\frac{\gamma + \mu}{\beta} \right) + \beta \left(\frac{\mu(-\beta + \gamma + \mu)}{\beta(\gamma + \mu)} \right) - \mu]\lambda^2$$

$$- \left[-3\mu^2 + 2\mu\beta \left(\frac{\gamma + \mu}{\beta} \right) - 2\mu\gamma + 2\beta \left(\frac{\mu(-\beta + \gamma + \mu)}{\beta(\gamma + \mu)} \right) \mu + \beta \left(\frac{\mu(-\beta + \gamma + \mu)}{\beta(\gamma + \mu)} \right) \gamma \right]$$

$$+ u \left[\mu^2 - \mu\beta \left(\frac{\gamma + \mu}{\beta} \right) + \mu\gamma + \beta \mu \left(\frac{\mu(-\beta + \gamma + \mu)}{\beta(\gamma + \mu)} \right) - \beta \left(\frac{\mu(-\beta + \gamma + \mu)}{\beta(\gamma + \mu)} \right) \gamma \right]$$

Resolving the above resulted to

$$P(\lambda) = \lambda^3 - \left[-2\mu + \frac{\mu(-\beta + \gamma + \mu)}{(\gamma + \mu)} \right] \lambda^2 - \left[-3\mu^2 + 2\mu(\gamma + \mu) - 2\mu\gamma + \frac{2\mu^2(-\beta + \gamma + \mu)}{(\gamma + \mu)} + \frac{\mu\gamma(-\beta + \gamma + \mu)}{(\gamma + \mu)} \right] \lambda + u \left[\mu^2 - \mu(\gamma + \mu) + \mu\gamma + \frac{\mu^2(-\beta + \gamma + \mu)}{(\gamma + \mu)} - \frac{\mu\gamma(-\beta + \gamma + \mu)}{(\gamma + \mu)} \right] \quad (iii)$$

The above (iii) is the characteristic polynomial at the Endemic equilibrium point

Now, to determine the stability of the model we solve for the eigenvalues as follows for (iii)

To find a linear factor we solve for (iii)

$$\text{Let } \lambda = -\mu$$

$$P(-\mu) = 0$$

This implies that $(\lambda + \mu)$ is linear factor of the polynomial hence first eigenvalue is $\lambda_1 = -\mu$

We look for the other two eigenvalues by reducing the cubic polynomial to quadratic and evaluated the eigenvalues to be $\lambda_{2,3}$

$$= \frac{-\mu\beta \pm \sqrt{4\mu^4 - 4\beta\mu^3 + \beta^2\mu^2 - 8\beta\gamma\mu^2 + 12\gamma^2\mu^2 - 4\beta\gamma^2\mu + 4\gamma^3\mu}}{2\gamma + \mu}$$

Therefore, according to **theorem 1**, we have that $\lambda_1 = -\mu$ which satisfy the criteria for stability since its $\lambda_1 < 0$ and $\lambda_{2,3}$ will satisfy the stability condition if and only if the determinant (values under square root) is less than zero i.e. it is a complex function containing real imaginary part. Thus, we consider the real part $\text{Re}(\lambda_{2,3})$ which is negative ($\lambda_{2,3} \leq 0$) satisfying the condition for stability. Hence we say the model is local stable at the Endemic equilibrium point

Stability of the SIS Model

Stability at disease free equilibrium point

To determine the stability of the model at DFE point we use the Jacobin matrix.

$$J(s,I) = \begin{bmatrix} \frac{\partial S}{\partial s} & \frac{\partial S}{\partial i} \\ \frac{\partial I}{\partial s} & \frac{\partial I}{\partial i} \end{bmatrix}$$

$$S = -\beta si + \gamma i + \mu - \mu s$$

$$I = \beta si - \gamma i - \mu i$$

The disease equilibrium point is $E^0(s_0, i_0) = (1, 0)$

$$J(s_0, I_0) = \begin{bmatrix} -\beta i - \mu & -\beta s + \mu \\ \beta i & \beta s - \gamma - \mu \end{bmatrix}$$

To determine the stability of the model we look at the nature of the eigenvalues of the matrix above by finding the characteristic polynomial using

$$|A - \lambda I|$$

$$J(s_0, I_0) = \begin{bmatrix} -\beta i - \mu - \lambda & -\beta s + \mu \\ \beta i & \beta s - \gamma - \mu - \lambda \end{bmatrix}$$

Now we find the determinant **Det (j)**

$$\text{Det (j)} = [(-\beta i - \mu - \lambda)(\beta s - \gamma - \mu - \lambda)] - [\beta i(-\beta s + \mu)]$$

Now applying the values of the variables at equilibrium point it results to

$$= [(-\mu - \lambda)(\beta s - \gamma - \mu - \lambda)] - [0(-\beta s + \mu)]$$

$$[(-\mu - \lambda)(\beta s - \gamma - \mu - \lambda)]$$

$$-\beta\mu + \mu\gamma + \mu^2 + \mu\lambda - \beta\lambda + \gamma\lambda + \mu\lambda + \lambda^2 = 0$$

$$\lambda^2 + \mu\lambda + \gamma\lambda - \beta\lambda + \mu\lambda - \beta\mu + \mu\gamma + \mu^2 = 0$$

Re organizing we have that

$$\lambda^2 + (\mu + \gamma - \beta + \mu)\lambda - \beta\mu + \mu\gamma + \mu^2 = 0$$

The above gives us the characteristic polynomial. Now to obtain the eigenvalues of the characteristic polynomial we use the quadratic formula

$$\lambda = \frac{-b \pm \sqrt{b^2 - 4ac}}{2a}$$

$$b = (\mu + \gamma - \beta + \mu)$$

$$b^2 = (\mu + \gamma - \beta + \mu) (\mu + \gamma - \beta + \mu)$$

$$= 4\mu^2 + 4\mu\gamma - 4\beta\mu + \gamma^2 - 2\beta\gamma + \beta^2$$

$$4ac = 4(-\beta\mu + \mu\gamma + \mu^2) = -4\beta\mu + 4\mu\gamma + 4\mu^2$$

$$b^2 - 4ac = 4\mu^2 + 4\mu\gamma - 4\beta\mu + \gamma^2 - 2\beta\gamma + \beta^2 + 4\beta\mu - 4\mu\gamma - 4\mu^2$$

$$\sqrt{b^2 - 4ac} = \sqrt{\gamma^2 - 2\beta\gamma + \beta^2} = (\gamma - \beta)$$

Therefore,

$$\lambda_1 = \frac{-\mu + \gamma - \beta + \mu + (\gamma - \beta)}{2} = \frac{-2\mu}{2}$$

$$\lambda_1 = -\mu$$

$$\lambda_2 = \frac{-\mu + \gamma - \beta + \mu - (\gamma - \beta)}{2}$$

$$\lambda_2 = \frac{2(-\mu - \gamma)}{2}$$

$$\lambda_2 = -\mu - \gamma$$

Thus from theorem 1, we notice that the eigenvalues $\lambda_1 < 0$ $\lambda_2 < 0$ signifying it is less than zero and hence proves the system at DFE point to be locally stable

Stability at the endemic equilibrium point

To determine the stability of the model at the endemic equilibrium we also use the Jacobin matrix.

The endemic equilibrium point is $E^*(S, I^*) = \left[\frac{\gamma + \mu}{\beta}, \frac{-(-\beta + \gamma + \mu)}{\beta} \right]$

$$J(s^*, i^*) = \begin{bmatrix} -\beta i - \mu - \lambda & -\beta s + \mu \\ \beta i & \beta s - \gamma - \mu - \lambda \end{bmatrix}$$

To obtain the characteristic polynomial we first find the determinant

$$\text{Det(j)} = \left[(-\beta \frac{(-\beta + \gamma + \mu)}{\beta}) - \mu - \lambda \right] (\beta \frac{(\gamma + \mu)}{\beta} - \gamma - \mu - \lambda) - \left[\beta \frac{\gamma + \mu}{\beta} (-\beta \frac{(\gamma + \mu)}{\beta} + \mu) \right]$$

Expanding the above results

$$[(-\beta + \gamma + \mu - \mu - \lambda)(\gamma + \mu - \gamma - \mu - \lambda)] - [(\gamma + \mu - \mu)(-\gamma - \mu + \mu)]$$

$$\lambda^2 + \lambda (\beta - \gamma) - \gamma\beta + \gamma\mu + \gamma^2 = 0$$

The above is the characteristic polynomial. We investigate the polynomial for stability of the of the model by finding out the eigenvalues

$$\text{Using } \lambda = \frac{-b \pm \sqrt{b^2 - 4ac}}{2a}$$

$$b^2 = (\beta - \gamma) (\beta - \gamma)$$

$$= \beta^2 - \gamma\beta - \gamma\beta + \gamma^2$$

$$4ac = 4(-\gamma\beta + \gamma\mu + \gamma^2)$$

$$\lambda = \frac{-(\beta - \gamma) \pm \sqrt{\beta^2 + 2\gamma\beta - 3\gamma^2 - 4\gamma\mu}}{2}$$

Considering the equation (discriminant) under the square root sign, if the value of $b^2 < 4ac$ then the eigenvalues are complex with the real part $-(\beta - \gamma)$ (the real part is negative), then according to theorem 1, we conclude that the endemic equilibrium is stable since the real parts of both eigenvalues are negative. This shows that system is locally stable at the endemic equilibrium point

Discussion

Effective contact rate β and recovery rate γ are two main parameters that impact the spread of diseases. Consequently it determines the number of people in a particular compartment in a population. These two usually works to counter act or nullifies the other, while one reduces, the other increases vice versa. We observed that an increase in effective contact rate (signifying spread of the disease) while recovery rate is less, increased the number of the infectious compartment in the population over the given period of time. On the contrary, an increase in recovery rate (signifying decline of the disease) while effective contact rate is less, increased the number of the susceptible population over a given period of time.

However, effective contact rate could be frequency dependent or mass action dependent, while the recovery rate depends on vaccination and isolation strategies. Effective contact rate is said to be dependent on frequency mode of transmission if transmission of the disease depends on the number of time the infectious compartment come in contact with susceptible individuals in the population that leads to successful transmission of the disease. The mass action mode of transmission depends on the population density. These two parameters are always in consideration when eradicating epidemics.

Conclusion and Recommendation

The study of the evaluation of SIR and SIS compartmental epidemiological models suggests that there are two equilibrium points which are the disease-free equilibrium point and the endemic equilibrium point. We further obtained the local stability of these models at these points to confirm stability. The disease equilibrium point indicates that the disease will be wiped out or it dies out implying that the population can be said to be disease free because, the infected compartment is zero (empty) and so there is no recuperation. On the contrary moving on to the endemic equilibrium point these adjustments as the contaminated category maintain some value. The endemic equilibrium state is the point where the disorder can't be completely eradicated but stays in the population. For the ailment to persist in the population the compartments ought to no longer be zero at equilibrium point.

In different words, if it is the endemic equilibrium state, then $E(S^*, I^*, R^*) \neq (0, 0, 0)$.

These points have been further used to obtain the stability of the models in accordance to theorem 1, proposing that if the eigenvalues are negative, then the system is stated to be stable. However, the practical use of epidemic models must rely heavily on the reality put into the models. This doesn't mean that a reasonable model can include all possible effects but rather includes the mechanisms in the simplest possible way so as to maintain major components that influence disease epidemics.

The following recommendations were made in the study;

- i. That the population must work in earnest to avoid continual spread of the disease by affording themselves the public enlightenment by professional bodies or agencies
- ii. In the case where endemic equilibrium exist the government and other major stake holders in public health sector should understand how effective inoculation (vaccination), isolation plan helps to increase the number of individuals the susceptible class by reducing the effective contact rate.
- iii. Government and other agencies should be proactive about winning the war against epidemics by making free or affordable preventive tools or instrument to the populace and also consider sponsoring more researches related to mathematical modeling of diseases
- iv. Great care should be taken before epidemic models are used for prediction of real world phenomena because even simple models pose important questions about the underlying mechanisms of infection spread and possible means of control of the disease or epidemic

Conflict of Interest

The authors declare that there is no conflict of interest related to this study.

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