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On the Existence, Uniqueness, Stability of Solution and Numerical Simulations of a Mathematical Model for Measles Disease

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ABSTRACT.In the study, we model a measles disease using deterministic Susceptible-Exposed-Infectives-Recovered (SEIR) epidemiological model to study the prevalence and control of the measles disease in Senegal. By using measles data pertinent to Senegal , we carried out the stability of the model, established the existence and uniqueness of the solution to the model. Runge-Kutta fourth order method is used to solve the model numerically. This is used to do a simulation of the model by using MATLAB programming language to determine the best strategies to adopt in controlling the measles disease. The model realized that the exposed individuals at latent period play a significant role in controlling the disease. It is established that if more people at latent period goes for treatment and therapy during this state, before they become infectives, the disease will be eradicated more speedily with time.

1. Introduction

Measles disease is a highly contagious childhood disease with a person-to-person transmission mode through the air by infectious droplets [4]; which has over 90% attack rates among susceptible persons [1]. Measles disease is caused by measles virus, an infectious agent belonging to the virus family Paramyxoviridae that causes an infection of the respiratory system with a common symptom of maculopapular (red) rash on the infected person's skin [2]. Infectious diseases has been a serious concern for both human and animals populations globally. Control

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and prevention measures are therefore important tasks both from a human and economic point of views. For effective intervention measures, complete understanding of disease transmission and how burden they can be in a population is therefore necessary.

Life-threatening measles cases are more likely to occur among young children who are malnourished, most especially children with vitamin A deficiency and children whose immune systems has been lessened due to HIV/AIDS or other severe diseases. Most deaths that come as a results of measles are as a results of complications from the consequences of the disease. Complications are more common in children who are under the age five, or adults over the age twenty [4]. It is estimated that close to 10% of measles cases recorded result to death in populations which has high levels of malnutrition (food deficiency related diseases) and lack of adequate heath care facilities and supports. Measles disease occurrences is still very common in many developing African countries [4]. More than 20 million stood at risk of been affected by measles every year. Overwhelmingly, majority (greater than 95%) of measles death globally occurs in countries with low per capital incomes and poor health facilities and supports which are found mostly in Africa [3].

In Senegal, with the targeted regular vaccination coverage, there are still risk for illness and death from measles which is still in existence as proven by the measles epidemic episode in 2009-2010 [5]. Evidently, measles disease is still one of the most infectious diseases in Senegal as well as some developing countries in Africa.

Epidemiological mathematical models are useful tools in proposing and testing theories, and in comparing, planning, implementing and evaluating various detection, prevention, therapy and control intervention programs for infectious diseases. [6] opined that beginning from the 20^{th} century, researchers has been using epidemiological models to modeled infectious diseases. Examples of such models can be seen in [7], [8] and [9]. Only few of these models fully represent some essential aspects of childhood epidemics like measles disease. [4] reported measles disease as one of the top five causes of death in children less than five of age in many African countries. [9], [12], [13], [14] and [15] discovered from their works that vaccination protects susceptibles individuals against infectious diseases like measles by producing herd immunity. Finding a threshold conditions that determines whether an infectious disease will continue to spread or will die out in a given population remains one of the fundamental questions of epidemiological modeling. So, there exists a fundamental epidemiological quantity R_0 , called the basic reproduction number. [10] carried out a study on Predicting and Preventing of measles disease epidemics in New Zealand. In their work, they used a deterministic SIR model to model the dynamics of measles disease under varying immunization strategies in a population with size and age structure. Although, this model achieved a tremendous success in predicting an epidemic of measles disease in 1997 and was instrumental in the decision for the health authority to carry out an intensive MMR (Measles, Mumps and Rubella) immunization campaign in that year in New Zealand, the authors did not take into account the latent period effect of measles disease of the exposed individuals over the entire populations in their work.[11], formulated a mathematical epidemiological model for control of measles epidemiology. They used SEIR model for varying population size which best describe a population dynamics of developing countries like Africa to determine the impact of exposed individuals at delayed period using assumed parameters and hypothetical population size through the stability analysis of the model and numerical simulation. The authors did not explicitly state the numerical method employed in solving their model to carry out their simulation.

In this work, a Deterministic Susceptible-Exposed-Infective-Recovered epidemic model is given to model a measles disease with its corresponding mathematical analysis and numerical simulations by solving the model with Explicit Fourth Order Runge-Kutta ($RK4$) method .

The general objective of this work is for us to model the measles disease transmission in Senegal and to come up with the control measures to enable the reduction (possibly elimination) of the transmission of the disease in the country. The specific objectives of this work are enumerated below:

(1) Formulating of SEIR epidemiological model for measles transmission in Senegal using compartmental model approach which will help in deriving a system of differential equations.

(2) To present the existence of solution, uniqueness of solution, stability of the model at the disease free equilibrium state and numerical simulations of the model using data pertinent to Senegal.

(3) To discuss the implications of the model and propose recommendations for the control and possibly elimination of the disease.

2. Materials and Methods

SEIR model is a deterministic model because it can be applied to a large population. An SEIR model is a compartmental model where the total population is divided into four classes . The population under our consideration are homogeneously mixing and reflect the demography of a typical developing countries [16], so the model fit well as it experiments an exponentially increasing dynamics of the measles disease.

2.1: Model Assumptions

To represent an infectious disease with a mathematical model, some assumptions are needed to be considered based on the characteristics of the disease being considered, in this case; measles. The following are the assumptions for the SEIR model for measles disease as it is used in this work:

1. The recruitment rate (Newborns and Migrants) are assume to be susceptible
2. It is assumed that every person in our population is susceptible to the measles disease.
3. Individuals are equally likely to be infected by the infectious individuals in a case of contact except for those who are immune against measles.
4. Infectious individuals are detected early and isolated for immediate treatment and education
5. The population is homogeneously mixed. By homogeneously mixed we mean a population that interacts among themselves in such a uniformly manner.
6. The population is a varying population where recruitment rate and leaving rate are differ within a given time steps.
7. There is no treatment failure, a patient will either recover or die.

8. Recovered individuals are permanently immune against the disease.

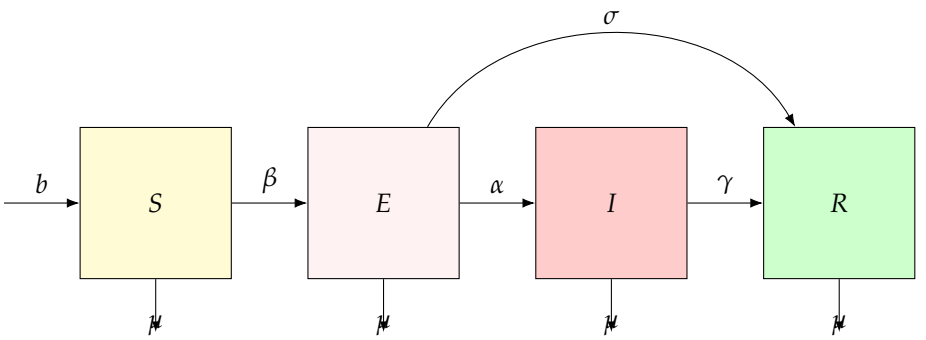

Figure 1: The transmission dynamics of measles disease in a population

Let N be the total population, we now divide the population into four (4) classes to explains the transmission dynamics of the measles disease in a given human population viz.:

- S (Susceptible),
- E (Exposed),
- I (Infective) and
- R (Recovered)

Figure 1 shows the transmission dynamics of measle disease in population as you move from one compartment to the other. The variables and their meaning are given in table 1 below:

Table 1: Variables used and their meanings.

Variable	**Meaning**
$S(t)$	The number of susceptible individuals at a given time, t
$E(t)$	The number of exposed individuals at a given time, t
$I(t)$	The number of infective individuals at a given time, t
$R(t)$	The number of recovered individuals at a given time, t

What follows below is the details of transmissions phases among these four compartments.

The susceptible class; S; is increasing by recruitment (birth and/or immigration rate) which we denoted by b. It is decreasing by infection if there is a contact with infected individuals at a rate of β, and is diminishing by leaving (normal death and emigration) rate which we denoted by μ.

For the second class,Exposed; E, the individuals here is formed by direct contact with infected individuals at a rate of β.

Table 2: Model Parameters used and their meanings.

Parameter Used	**It's Meaning**
b	Recruitment rate (Birth and Immigrants rate)
μ	Leaving rate (Death and Emigrants rate)
β	The contact rate
γ	The rate at which an infective individuals recovered per unit time
σ	The rate of exposed individuals who have undergone testing and therapy
α	The rate at which an exposed become infective

The class E is decreasing by individuals who have undergone testing and measles therapy at a rate of σ, the individuals who progresses into infected class at a rate of α and also this class is diminishing by leaving rate of μ.

The third class; I, of infective individuals is formed by individuals who progresses from exposed class to this class at a rate α. This class is decreasing by individuals who are recovering from infection at a rate of γ and is diminishing by leaving rate of μ.

From the SEIR model; it is assumes that both Exposed-Recovered individuals and Infected-Recovered individuals become immune to the measles disease permanently, i.e. you can only be infected once with the disease. We use this assumption to generates the fourth class; R; of individuals who have complete protection against the disease. This class R; of recovered individuals is diminishing by leaving rate of μ. We define parameters and their meaning in Table 2 below.

This SEIR model is now represented by the following system of first order differential equations and shows the transitions between the four compartments of the model:

$$\frac{dS}{dt} = b - \beta SI - \mu S, \tag{2.1}$$

$$\frac{dE}{dt} = \beta SI - (\mu + \alpha + \sigma)E \tag{2.2}$$

$$\frac{dI}{dt} = \alpha E - (\mu + \gamma)I \tag{2.3}$$

$$\frac{dR}{dt} = \gamma I + \sigma E - \mu R \tag{2.4}$$

where $N(t) = S(t) + E(t) + I(t) + R(t)$; since N represents the whole population.

2.2: Properties of the Model

The basic properties of the SEIR model are properties of (i) "feasible solution" and (ii) "positivity of the solution". The feasible solution of the model equations shows the region in which the solution of the equations are

β is defined to be the average number of effective contacts with other (susceptible) individuals per infective individuals per unit time.

biologically meaningful and the positivity of the solutions tells the non-negativity of the solutions of the model equations.

2.2.1: Feasible Solution.

The SEIR model is used here to model infectious disease in a human population, it is reasonable to assumed that the parameters used and variables in all classes are non negative; that is $t \geq 0$. We will provide proof that all variables of the model are non-negative for all given non-negatives initial conditions.

The feasible solution set which is positively invariant set of the model is given by:

$$\omega = \left\{ (S, E, I, R) \in \mathbf{R}_+^4 | N(t) = S(t) + E(t) + I(t) + R(t) \to \frac{b}{\mu} \right\} \tag{2.5}$$

The following lemma established this assertion.

Lemma 1. *The set ω is positively invariant and attracts all solution in $\mathbf{R}_+^4$.*

Proof. Since $N(t) = S(t) + E(t) + I(t) + R(t)$. We Adds equations (1) to (4) together to gives us the rate of change of the total population; i.e.

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

That is;

$$\frac{dN}{dt} = b - \mu S - \mu E - \mu I - \mu R$$
$$\frac{dN}{dt} = b - \mu(S + E + I + R).$$

Which gives;

$$\frac{dN}{dt} = b - \mu N \qquad (since\ N\ = S + E + I + R).$$

This is a first order linear differential equation.

A first order linear differential equation of the form:

$$\frac{dN}{dt} + \mu N = b \qquad (After\ \ re - arranging) \tag{2.6}$$

Can be solved by introducing integrating factor. Here, μ and b are both constants;

So, the integrating factor $I.F$ is given as:

$$I.F = \exp\left(\int \mu dt \right)$$

We now multiply both sides of equation (2.6) with $\exp(\int \mu dt)$. That is;

$$\exp\left(\int \mu dt \right)\left(\frac{dN}{dt} + \mu N \right) = b \exp\left(\int \mu dt \right) \tag{2.7}$$

The L.H.S. of equation (2.7) is;

$$\frac{d}{dt}\left[N(t).\exp\left(\int \mu dt \right) \right]$$

Therefore;

$$\frac{d}{dt}\left[N(t).\exp\left(\int \mu dt \right) \right] = b.\exp\left(\int \mu dt \right)$$

Integrating both sides, we have that:

$$N(t).\exp(\mu dt) = \frac{b}{\mu}\exp(\mu dt) + K$$

where K is a constant.

So that;

$$N(t) = \frac{b}{\mu} + K\exp(-ut).$$

When $t = 0$, we have that;

$$N(0) = \frac{b}{\mu} + K$$

Therefore;

$$K = N(0) - \frac{b}{\mu}$$

Substituting the value of K, the solution (with simplification) of this linear differential equation will becomes:

$$N(t) = N(0)\exp^{-\mu t} + \frac{b}{\mu}(1 - \exp(-\mu t)).$$

Taking the limit as $t \to \infty$, we have;

$$N(t) \leq \frac{b}{\mu}$$

Therefore, we have established that ω is positively invariant and attracts all solution in $\mathbf{R}_+^4$. □

2.2.2. Positivity of Results

What follows now is to prove that all variables in the SEIR model equations of (2.1) to (2.4) are non-negative.

Lemma 2. *Let the initial data set be $(S, E, I, R)(0) \geq 0 \in \omega$, then the solution set $(S, E, I, R)(t)$ of the equations (2.1) to (2.4) is positive for all $t > 0$.*

Proof. From equation (2.1) if we assumed that:

$$\frac{dS}{dt} = b - \beta SI - \mu S \geq -(\beta SI + \mu S).$$

That is;

$$\frac{dS}{dt} \geq -(\beta I + \mu)S.$$

OR;

$$\frac{dS}{S} \geq -(\beta I + \mu)dt \qquad (by\ separation\ of\ variables,\ since\ S \neq 0).$$

Integrating both sides of the inequalities; we have;

$$In\big(S(t)\big) \geq -(\beta I + \mu)t + K$$

So that;

$$S(t) \geq K \exp\Big(- (\beta I + \mu)t \Big)$$

At $t = 0$; this becomes:

$$S(t) \geq S(0) \exp\Big(- (\beta I + \mu)0 \Big) \geq 0 \qquad (since\ (\beta I + \mu) > 0)$$

That is;

$$S(t) > 0$$

Similarly from equation (2.2)

$$\frac{dE}{dt} = \beta SI - (\mu + \alpha + \sigma)E \geq -(\mu + \alpha + \sigma)E$$

i.e;

$$\frac{dE}{dt} \geq -(\mu + \alpha + \sigma)E$$

OR;

$$\frac{dE}{E} \geq -(\mu + \alpha + \sigma)dt \qquad (by\ separation\ of\ variables,\ since\ E \neq 0).$$

Integrating both sides of the inequalities we have;

$$In\big(E(t)\big) \geq -(\mu + \alpha + \sigma)t + K$$

$$E(t) \geq K \exp\Big(- (\mu + \alpha + \sigma)t \Big)$$

At $t = 0$, we have that;

$$E(t) \geq E(0) \exp\Big(- (\mu + \alpha + \sigma \Big) 0 \geq 0, \qquad (since\ (\mu + \alpha + \sigma) > 0$$

That is;

$$E(t) > 0$$

Also from equation (2.3);

$$\frac{dI}{dt} = \alpha E - (\mu + \gamma)I \geq -(\mu + \gamma)I$$

That is;

$$\frac{dI}{dt} \geq -(\mu + \gamma)I$$

Then we have that:

$$\frac{dI}{I} \geq -(\mu + \gamma)dt$$

On integrating we obtained;

$$In\big(I(t)\big) \geq -(\mu + \gamma)t + K$$

That is;

$$I(t) \geq K \exp\Big(-(\mu+\gamma)t \Big)$$

At $t = 0$;

$$I(t) \geq I(t) \exp\Big(-(\mu+\gamma)0 \Big)$$

So that;

$$I(t) > 0$$

Finally from equation (2.4):

$$\frac{dR}{dt} = \gamma I + \sigma E - \mu R > \sigma E - \mu R$$

That is;

$$\frac{dR}{dt} > \sigma E - \mu R$$

Which has an integrating factor:

$$I.F = \exp(-\mu t)$$

Then we have:

$$\frac{dR}{dt}.\exp(-\mu t) > \sigma E.\exp(-\mu t) - \mu R.\exp(-\mu t)$$

Integrating at constant K, we have;

$$R(t) > \frac{\sigma E}{\mu} + K \exp(-\mu t)$$

When $t = 0$ we obtained that;

$$R(0) > \frac{\sigma E}{\mu} + K$$

The solution then becomes;

$$R(t) > R(0) \exp(-\mu t) + \frac{\sigma E}{\mu}(1 - \exp(-\mu t))$$

That is;

$$R(t) > 0.$$

Hence, we have proved that all variables are positive $\forall\ t > 0$. □

2.3. Existence and Uniqueness of Solution for the SEIR Model

The general first-order ODE is in the form:

$$x' = f(t, x), \qquad x(t_0) = x_0 \tag{2.8}$$

One will be interested in asking the following questions:

(i) Under what conditions can we say solution to equation (2.8) exists?

(ii) Under what conditions can we say there is a unique solution to equation (2.8)?

To answers these:

Let:

$$f_1 = b - \beta SI - \mu S,$$

$$f_2 = \beta SI - (\mu + \alpha + \sigma)E,$$

$$f_3 = \alpha E - (\mu + \gamma)I,$$

and

$$f_4 = \gamma I + \sigma E - \mu R$$

We use the following theorem to established the existence and uniqueness of solution for our SEIR model.

Theorem 1 (Uniqueness of Solution)

Let use D to denotes the domain:

$$|t - t_0| \leq a, ||x - x_0|| \leq b, x = (x_1, x_2, ..., x_n), x_0 = (x_{10}, x_{20}, ..., x_{n0}) \tag{2.9}$$

and suppose that $f(t, x)$ satisfies the Lipschitz condition:

$$||f(t, x_1) - f(t, x_2)|| \leq k||x_1 - x_2||, \tag{2.10}$$

and whenever the pairs (t, x_1) and (t, x_2) belong to the domain D , where k is used to represent a positive constant.

Then, there exist a constant $\delta > 0$ such that there exists a unique (exactly one) continuous vector solution $x(t)$ of the system (2.8) in the interval $|t - t_0| \leq \delta$. It is important to note that condition (2.10) is satisfied by requirement that:

$$\left\{ \frac{\partial f_i}{\partial x_j}, \quad {\scriptstyle i,j=1,2,...,n} \right.$$

be continuous and bounded in the domain D.

Lemma 3: *If $f(t, x)$ has continuous partial derivative $\frac{\partial f_i}{\partial x_j}$ on a bounded closed convex domain $\mathcal{R}$ (i.e, convex set of real numbers), where $\mathcal{R}$ is used to denotes real numbers , then it satisfies a Lipschitz condition in $\mathcal{R}$. Our interest is in the domain:*

$$1 \leq \epsilon \leq \mathcal{R}. \tag{2.11}$$

So, we look for a bounded solution of the form

$$0 < \mathcal{R} < \infty.$$

We now prove the following existence theorem.

Theorem 2: (Existence of Solution)

Let D denote the domain defined in (2.9) such that (2.10) and (2.11) hold. Then there exist a solution of model system of equations (2.1)-(2.4) which is bounded in the domain D.

Proof. Let:

$$f_1 = b - \beta SI - \mu S, \tag{2.12}$$

$$f_2 = \beta SI - (\mu + \alpha + \sigma)E, \tag{2.13}$$

$$f_3 = \alpha E - (\mu + \gamma)I, \tag{2.14}$$

and

$$f_4 = \gamma I + \sigma E - \mu R \tag{2.15}$$

We shows that:

$\frac{\partial f_i}{\partial x_j}$, $i, j = 1, 2, 3, 4$ are continuous and bounded. That is, the partial derivatives are continuous and bounded. We explored the following partial derivatives for all the model equations:

From equation (2.12);

$$\frac{\partial f_1}{\partial S} = -\beta I - \mu, \left|\frac{\partial f_1}{\partial S}\right| = \left| -\beta I - \mu\right| < \infty,$$

$$\frac{\partial f_1}{\partial E} = 0, \left|\frac{\partial f_1}{\partial E}\right| = |0| < \infty,$$

$$\frac{\partial f_1}{\partial I} = -\beta S, \left|\frac{\partial f_1}{\partial I}\right| = \left| -\beta S\right| < \infty,$$

$$\frac{\partial f_1}{\partial R} = 0, \left|\frac{\partial f_1}{\partial R}\right| = |0| < \infty.$$

Similarly, from equation (2.13) we also have that:

$$\frac{\partial f_2}{\partial S} = \beta I, \left|\frac{\partial f_2}{\partial S}\right| = \left|\beta I\right| < \infty,$$

$$\frac{\partial f_2}{\partial E} = -(\mu + \alpha + \sigma), \left|\frac{\partial f_2}{\partial E}\right| = \left| -(\mu + \alpha + \sigma)\right| < \infty,$$

$$\frac{\partial f_2}{\partial I} = \beta S, \left|\frac{\partial f_2}{\partial I}\right| = \left|\beta S\right| < \infty,$$

$$\frac{\partial f_2}{\partial R} = 0, \left|\frac{\partial f_2}{\partial R}\right| = |0| < \infty.$$

Also from equation (2.14);

$$\frac{\partial f_3}{\partial S} = 0, \left|\frac{\partial f_3}{\partial S}\right| = |0| < \infty,$$

$$\frac{\partial f_3}{\partial E} = \alpha, \left|\frac{\partial f_3}{\partial S}\right| = |\alpha| < \infty,$$

$$\frac{\partial f_3}{\partial I} = -(\mu+\gamma), \left|\frac{\partial f_3}{\partial I}\right| = |-(\mu+\gamma)| < \infty,$$

$$\frac{\partial f_3}{\partial R} = 0, \left|\frac{\partial f_3}{\partial R}\right| = |0| < \infty.$$

Finally we have from equation (2.15) that:

$$\frac{\partial f_4}{\partial S} = 0, \left|\frac{\partial f_4}{\partial S}\right| = |0| < \infty,$$

$$\frac{\partial f_4}{\partial E} = \alpha, \left|\frac{\partial f_4}{\partial E}\right| = |\alpha| < \infty,$$

$$\frac{\partial f_4}{\partial I} = \gamma, \left|\frac{\partial f_4}{\partial I}\right| = |\gamma| < \infty,$$

$$\frac{\partial f_4}{\partial R} = -\mu, \left|\frac{\partial f_4}{\partial R}\right| = |-\mu| < \infty.$$

We have clearly established that all these partial derivatives are continuous and bounded, hence, by **Theorem (1)**, we can say that there exist a unique solution of (2.1)-(2.4) in the region D. □

2.4. Existence of Steady States of the System

Under this section we will find the equilibrium points and established asymptotic stability of the SEIR model.

In order to obtained the equilibrium points of the system, we equate the system of equations to zeros; i.e.;

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0 \tag{2.16}$$

2.5. Stability of the SEIR Model (Asymptotic Stability)

For us to find the stability of the model, we first needs to find the equilibrium points (disease free equilibrium) of the system (2.1) to (2.4).

By equating the system to zeros; we have:

$$b - \beta SI - \mu S = 0 \tag{2.17}$$

$$\beta SI - (\mu + \alpha + \sigma)E = 0 \tag{2.18}$$

$$\alpha E - (\mu + \gamma)I = 0 \tag{2.19}$$

$$\gamma I + \sigma E - \mu R = 0 \tag{2.20}$$

From equation (2.17) above;

$$b = \beta SI + \mu S$$

$$b = (\beta I + \mu)S$$

So that;

$$S = \frac{b}{\beta I + \mu}$$

But at the disease free state, $\beta = 0$.

Implying;

$$S = \frac{b}{\mu}$$

Also from equation (2.18);

$$\beta SI = (\mu + \alpha + \sigma)E$$

So that;

$$E = \frac{\beta SI}{(\mu + \alpha + \sigma)}$$

Implying that;

$$E = 0 \qquad (since\ \beta = 0)$$

Similarly from equation (2.19);

$$\alpha E = (\mu + \gamma)I$$

So we have that;

$$I = \frac{\alpha E}{(\mu + \gamma)}$$

We now have that;

$$I = 0 \qquad (since\ E = 0)$$

Finally from equation (2.20);

$$\gamma I + \sigma E = \mu R$$

So that;

$$R = \frac{\gamma I + \sigma E}{\mu}$$

Implying that;

$$R = 0 \qquad (since\ I\ and\ E = 0)$$

Thus we have that the disease free equilibrium for this SEIR model is given by:

$$\mathcal{P}_0 = \left(\frac{b}{\mu}, 0, 0, 0\right)$$

Note: We assumed $b \neq \mu$ for this model.

The next thing in our analysis is to shows that an endemic equilibrium:

$$\mathcal{P}^* = (S^*, E^*, I^*, R^*) > 0.$$

From our equilibrium equations of (2.17) to (2.20) above; considering equations (2.18) and (2.19); we have that:

$$\beta SI = (\mu + \alpha + \sigma)E$$

$$(\mu + \gamma)I = \alpha E$$

Which when we divide the first by the second yields;

$$S = S^* = \frac{(\mu + \alpha + \sigma)(\mu + \gamma)}{\alpha}$$

That is;

$$S^* = \frac{(\mu + \alpha + \sigma)(\mu + \gamma)}{\alpha}$$

Which is clearly greater than zero.

Similarly, adding equations (2.17) and (2.18), we have that:

$$b - \beta SI - \mu S + \beta SI - E(\mu + \alpha + \sigma) = 0$$

$$b - \mu S - E(\mu + \alpha + \sigma) = 0$$

$$\mu S = b - E(\mu + \alpha + \sigma)$$

Implying;

$$S = \frac{b - E(\mu + \alpha + \sigma)}{\mu}$$

So that;

$$S = \frac{-(\mu + \alpha + \sigma)E + b}{\mu} \tag{2.21}$$

Now, from equation (2.19),

$$\alpha E = (\mu + \gamma)I$$

Which gives us:

$$I = \frac{\alpha E}{(\mu + \gamma)} \tag{2.22}$$

From equation (2.17) we have that:

$$\beta SI = (\mu + \alpha + \sigma)E \tag{2.23}$$

But from equations (2.21) and (2.22) above; substituting the values of S and I into equation (2.23) we now have that:

$$(\mu+\alpha+\sigma)E = \frac{\beta\Big[(-\mu-\alpha-\sigma)E+b\Big]*(\alpha E)}{\mu(\mu+\gamma)}$$

So that;

$$(\mu+\alpha+\sigma)E = \frac{\beta\Big[(-\mu-\alpha-\sigma)\alpha E^2+\alpha Eb\Big]}{\mu(\mu+\gamma)}$$

$$\frac{-\beta(\mu+\alpha+\sigma)\alpha E^2+(\beta\alpha Eb)}{\mu(\mu+\gamma)} - (\mu+\alpha+\sigma)E = 0$$

$$E\Big[\frac{-\beta(\mu+\alpha+\sigma)\alpha E}{\mu(\mu+\gamma)} + \frac{\beta\alpha b}{\mu(\mu+\gamma)} - (\mu+\alpha+\sigma)\Big] = 0$$

Either;

$$E = 0$$

Or;

$$\frac{-\beta\alpha(\mu+\alpha+\sigma)E}{\mu(\mu+\gamma)} + \frac{\beta\alpha b}{\mu(\mu+\gamma)} - (\mu+\alpha+\sigma) = 0$$

Now,

$$\frac{-\beta\alpha(\mu+\alpha+\sigma)E}{\mu(\mu+\gamma)} = (\mu+\alpha+\sigma) - \frac{\beta\alpha b}{\mu(\mu+\gamma)}$$

On Dividing both sides by $\beta\alpha(\mu+\alpha\ +\sigma)$ and multiplying both sides by $\mu(\mu+\gamma)$ we obtained that:

$$E = \frac{b}{\mu+\alpha+\sigma} - \frac{\mu(\mu+\gamma)}{\beta\alpha}$$

Therefore,

$$E^* = \frac{b}{\mu+\alpha+\sigma} - \frac{\mu(\mu+\gamma)}{\beta\alpha}$$

$$E^* = \frac{b}{\mu+\alpha+\sigma}\Big[1 - \frac{\mu(\mu+\alpha+\sigma)(\mu+\gamma)}{\beta\alpha b}\Big]$$

Let:

$$R_0 = \frac{\beta\alpha b}{\mu(\mu+\alpha+\sigma)(\mu+\gamma)}$$

We now have that:

$$E^* = \frac{b}{(\mu+\alpha+\sigma)}\Big[1 - \frac{1}{R_0}\Big]. \tag{2.24}$$

Now, considering I from equation (2.23);

That is;

$$I = \frac{\alpha E}{(\mu+\gamma)}$$

Substituting E^* for E, we obtained that:

$$I = \frac{\alpha}{(\mu+\gamma)}\left(\frac{b}{(\mu+\alpha+\sigma)}\left[1-\frac{\mu(\mu+\alpha+\sigma)(\mu+\gamma)}{\beta\alpha b}\right]\right)$$

So that;

$$I^* = \frac{\alpha b}{(\mu+\gamma)(\mu+\alpha+\sigma)}\left[1-\frac{1}{R_0}\right] \tag{2.25}$$

Finally from equation (2.20),

$$\gamma I + \alpha E = \mu R$$

So that;

$$R = \frac{\gamma I + \alpha E}{\mu}$$

On substituting the values of I^* and E^* for I and E in above, we obtained that:

$$R = \frac{\gamma\alpha b}{\mu(\mu+\gamma)(\mu+\alpha\ +\sigma)}\left[1-\frac{1}{R_0}\right] + \frac{\sigma b}{\mu(\mu+\alpha+\sigma)}\left[1-\frac{1}{R_0}\right]$$

$$R = \frac{b}{\mu(\mu+\alpha+\sigma)}\left(\frac{\alpha\gamma}{\mu+\gamma}+\sigma\right)\left[1-\frac{1}{R_0}\right]$$

On multiplying the R.H.S. by $\frac{\mu(\mu+\alpha+\sigma)}{b}$ we have that:

$$R^* = \left(\frac{\alpha\gamma}{\mu+\gamma}+\sigma\right)\left[1-\frac{1}{R_0}\right] \tag{2.26}$$

We have shown that S^*, E^*, I^* and R^* are all positives .That is; $P^* = (S^*, E^*, I^*, R^*) > 0$. ■

P^* represent an endemic steady state with constant number of people in the population being infected with the measles disease.

This is biologically reasonable when $S^* < N$, that is when:

$$R_0 = \frac{\beta\alpha b}{\mu(\mu+\alpha+\sigma)(\mu+\gamma)} > 1.$$

In another words, the necessary and sufficient condition for a unique P^* to exist in the feasible region ω is that:

$$0 < S^* \leq \frac{b}{\mu}.$$

Or equivalently;

$$\frac{b}{\mu} \geq 1.$$

We can also established the local stability of the disease-free equilibrium using Jacobian matrix of equations (2.1) to (2.4) and evaluate at $\mathcal{P}_0$. To achieved this we will evaluate the Jacobian matrix of equations (2.1) to (2.4) at $\mathcal{P}_0 = (\frac{b}{\mu}, 0, 0, 0)$ The local stability of the model will now be determined from the eigenvalues of the Jacobian matrix of the model equations at $\mathcal{P}_0$.

Now, the Jacobian matrix of equations (2.1) to (2.4) is given by:

$$J(f_i, i = 1, ..., 4) = \begin{pmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial R} \end{pmatrix}, \tag{2.27}$$

So that;

$$J(f_i, i = 1, ..., 4) = \begin{pmatrix} -\beta I - \mu & 0 & -\beta S & 0 \\ 0 & -(\mu + \alpha + \sigma) & \beta S & 0 \\ 0 & \alpha & -(\mu + \gamma) & 0 \\ 0 & \sigma & \gamma & -\mu \end{pmatrix} \tag{2.28}$$

But at the disease free equilibrium, $S = \frac{b}{\mu}$, and $I = 0$. Substituting these values into the equation (2.28) above, we get:

$$J(\mathcal{P}_0) = \begin{pmatrix} -\mu & 0 & -\frac{b\beta}{\mu} & 0 \\ 0 & -(\mu + \alpha + \sigma) & \frac{b\beta}{\mu} & 0 \\ 0 & \alpha & -(\mu + \gamma) & 0 \\ 0 & \sigma & \gamma & -\mu \end{pmatrix}. \tag{2.29}$$

The disease free equilibrium; $\mathcal{P}_0$, will be locally asymptotically stable if all the eigenvalues of $J(\mathcal{P}_0) < 0$. We will established this by finding the eigenvalues. We find the eigenvalues as follows:
Using:

$$|J(\mathcal{P}_0) - \lambda I| = 0. \tag{2.30}$$

Now,

$$|J(\mathcal{P}_0) - \lambda I| = \begin{vmatrix} -\mu - \lambda & 0 & -\frac{b\beta}{\mu} & 0 \\ 0 & -(\mu + \alpha + \sigma) - \lambda & \frac{b\beta}{\mu} & 0 \\ 0 & \alpha & -(\mu + \gamma) - \lambda & 0 \\ 0 & \sigma & \gamma & -\mu - \lambda \end{vmatrix} = 0 \tag{2.31}$$

To solve for the eigenvalues from equation (2.31), we find the characteristics equations as follows: We used minors and cofactors method and by inspection we observed that the matrix contains zeros elements, we choose the first rows to evaluate the determinant:

Recall:

Note: I in this case is a 4×4 identity matrix.

$$\begin{vmatrix} + & - & + & - \\ - & + & - & + \\ + & - & + & - \\ - & + & - & + \end{vmatrix}$$

are the signs used when evaluating 4×4 matrix where the $+$ or $-$ signs indicates the sign the corresponding element in the same position will have.

Now;

$$|J(\mathcal{P}_0) - \lambda I| = 0,$$

Becomes,

$$-(\mu+\lambda)\begin{vmatrix} -(\mu+\alpha+\sigma+\lambda) & \frac{b\beta}{\mu} & 0 \\ \alpha & -(\mu+\gamma+\lambda) & 0 \\ \sigma & \gamma & -(\mu+\lambda) \end{vmatrix} = 0$$

$$(\mu+\lambda)^2\left[(\mu+\alpha+\sigma+\lambda)(\mu+\gamma+\lambda+\frac{b\beta\alpha}{\mu})\right] = 0$$

Either;

$$(\mu+\lambda)^2 = 0,$$

Or,

$$\left[(\mu+\alpha+\sigma+\lambda)(\mu+\gamma+\lambda+\frac{b\beta\alpha}{\mu})\right] = 0$$

Solving we obtained that:

$$\lambda_1 = -\mu, \;\; \lambda_2 = -\mu, \;\; \lambda_3 = -(\mu+\sigma+\alpha), \;\; \lambda_4 = -\left(\frac{b\beta\alpha}{\mu}+\mu+\gamma\right).$$

Since all the parameters used in the model are non- negative, we have shown that all the eigenvalues for $J(\mathcal{P}_0)$ are negatives, implying that the disease free equilibrium is locally asymptotically stable. ■

Table 3 below gives the model parameters, its values and sources

Numerical Method Solution for the Model

We will consider the explicit $RK4$ method for solving numerically the non-linear first order ordinary differential equations (ODEs) of our SEIR model of equations (2.1) to (2.4) with given initial conditions. Explicit $RK4$ method is the most commonly used of the Runge-Kutta methods. It is highly recommended in solving numerically systems of ODEs that are not analytically solvable . This is due to the fact that the method is very accurate in comparison to

Reference: Klamka. J. et all, (2004); Google Scholar

Table 3: Model parameter values Used and Sources

Model Parameter symbol	Model Parameter value	Parameter Source
b	0.0784	[17] and [20]
μ	0.0545	[18] and [20]
β	0.09091 per day	Immunization Action Coalition
γ	0.125 per day	[21]
σ	0.00, 0.30, 0.60 & 0.90	Assumed Values
α	0.14286 per day	[21]

other methods of solving ODEs numerically. It has an advantage of the possibility of using a changeable integrate step. However, there are short-comings of the method. The Runge-Kutta methods generally take comparatively long time for computations and to evaluate errors is not an easy task. $RK4$ method is widely used because of its consistent (stability) nature. Higher orders may not be consistent.

Model and Algorithm:

Suppose we have a system of ordinary differential equations of the form:

$$\begin{aligned}
\dot{y}_1 &= f_1(t, y_1, y_2, ..., y_n),\\
\dot{y}_2 &= f_2(t, y_1, y_2, ..., y_n),\\
\dot{y}_3 &= f_3(t, y_1, y_2, ..., y_n),\\
&\vdots\\
\dot{y}_n &= f_n(t, y_1, y_2, ..., y_n).
\end{aligned}$$

with a given initial conditions:

$$y_i(0) = y_0^i, \quad i = 0, 1, 2, ..., n.$$

We can put them together in a vector form to obtained:

$$\dot{Y}_n = F_n(t, y), \quad Y(t_0) = y_0$$

Where:

$$Y(t) = \begin{pmatrix} y_1 \\ y_2 \\ \vdots \\ y_n \end{pmatrix}; \quad F(t, y) = \begin{pmatrix} f_1 \\ f_2 \\ \vdots \\ f_n \end{pmatrix}.$$

The standard formula for explicit fourth-order Runge-Kutta method is given as:

$$y_{i+1} = y_i + \frac{h}{6}(K_1 + 2K_2 + 2K_3 + K_4).$$

Where:

$$K_1 = f(t_i, y_i),$$
$$K_2 = f(t_i + \frac{h}{2}, y_i + h\frac{K_1}{2}),$$
$$K_3 = f(t_i + \frac{h}{2}, y_i + h\frac{K_2}{2}),$$

and:

$$K_4 = f(t_i + \frac{h}{2}, y_i + hK_3).$$

Now, for our SEIR model equations (2.1) to (2.4); the explicit fourth-order Runge-Kutta method becomes:

$$S_{i+1} = S_i + \frac{h}{6}(K_{1S} + 2K_{2S} + 2K_{3S} + K_{4S}),$$
$$E_{i+1} = E_i + \frac{h}{6}(K_{1E} + 2K_{2E} + 2K_{3E} + K_{4E}),$$
$$I_{i+1} = I_i + \frac{h}{6}(K_{1I} + 2K_{2I} + 2K_{3I} + K_{4I}),$$

and:

$$R_{i+1} = R_i + \frac{h}{6}(K_{1R} + 2K_{2R} + 2K_{3R} + K_{4R}).$$

Where:

$$K_{1S} = f_1\Big(t_i, S_i, E_i, I_i, R_i\Big),$$
$$K_{1E} = f_2\Big(t_i, S_i, E_i, I_i, R_i\Big),$$
$$K_{1I} = f_3\Big(t_i, S_i, E_i, I_i, R_i\Big),$$
$$K_{1R} = f_4\Big(t_i, S_i, E_i, I_i, R_i\Big),$$

$$\vdots$$

$$K_{4S} = f_1\Big(t_i + h, S_i + hK_{3S}, E_i + hK_{3E}, I_i + hK_{3I}, R_i + hK_{3R}\Big),$$
$$K_{4E} = f_2\Big(t_i + h, S_i + hK_{3S}, E_i + hK_{3E}, I_i + hK_{3I}, R_i + hK_{3R}\Big),$$
$$K_{4I} = f_3\Big(t_i + h, S_i + hK_{3S}, E_i + hK_{3E}, I_i + hK_{3I}, R_i + hK_{3R}\Big),$$
$$K_{4R} = f_4\Big(t_i + h, S_i + hK_{3S}, E_i + hK_{3E}, I_i + hK_{3I}, R_i + hK_{3R}\Big).$$

Let the matrix Q represents f_1, f_2, f_3 and f_4 and the initial condition $y(0)$ for S_i, E_i, I_i and R_i , the time t and the step size h. The algorithm to compute the vector $Y_{(t+1)}$ in a given time t from the formula above is given below:

1. $P \leftarrow y(0)$;
2. for i = 1,..., $\frac{t}{h}$; $t_{i+1} = t_i + h$; do:
   - $K_1 \leftarrow QP$
   - $K_2 \leftarrow Q(P + h\frac{K_1}{2})$
   - $K_3 \leftarrow Q(P + h\frac{K_2}{2})$
   - $K_4 \leftarrow Q(P + hK_3)$
   - $y(0) + \frac{h}{6}(K_1 + 2K_2 + 2K_3 + K_4)$;
3. $Y_{(t+1)} \leftarrow P$

   end.

3. Results and Discussion

Numerical solution to the model equations (2.1) to (2.4) were given by employing MatLab programming language using explicit Runge-Kutta fourth order method to give an approximate solutions since all the model equations (S,E,I,R) can not be solved analytically. The solution of SEIR model is then used to do numerical simulations using the parameter values pertinent to Senegal which are given in Table 3 below. These parameters were obtained from different sources in these medical field literatures: (Index Mundi (I.M.) [[17] & [18]], Immunization Action Coalition (2004)[19], Migration au Senegal: Profil National (2009) [20] and Trottier and Philippe (2003) [21]).

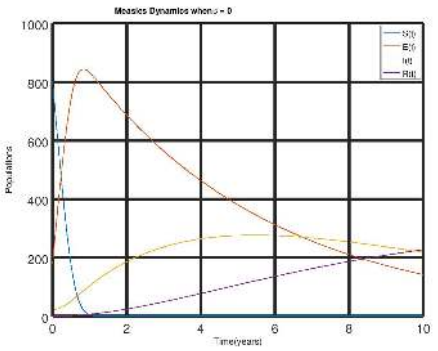

Figure 2: Measles Dynamics when $\sigma = 0.00$

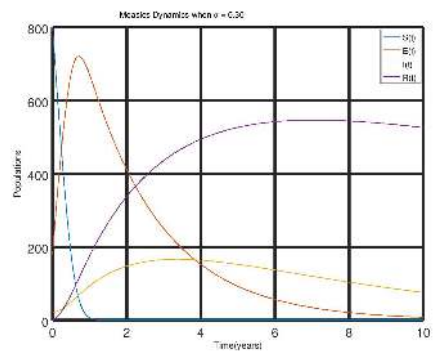

Figure 3: Measles Dynamics when $\sigma = 0.30$

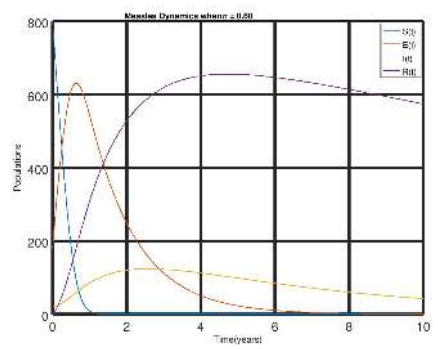

Figure 4: Measles Dynamics when $\sigma = 0.60$

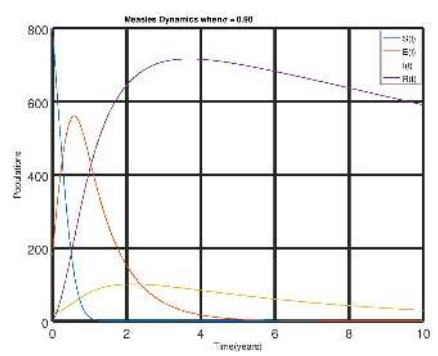

Figure 5: Measles Dynamics when $\sigma = 0.90$

Interpretations

Figure 2 above shows the dynamics of measles disease when $\sigma = 0.00$. We see in the Figure that, if none of the exposed individuals at latent period are diagnosed and treated, it will take much longer time for exposed individuals to decrease. Also, it takes significant longer period before noticing any significant improvement for individuals to Recovered from the disease. Similarly, The infectives individuals will significantly increases before noticing a decline in the number of infectives individuals.

In Figure 3, we do the simulation of the model when $\sigma = 0.30$. Looking at the Figure, we discovered that if only 30% of exposed individuals at latent period are diagnosed and treated, it will take lesser time for exposed individuals to decrease significantly. Also, it takes significant lesser period before noticing any significant improvement for individuals to Recovered from the disease. Similarly, The infectives individuals will significantly decline over time.

Figure 4 above shows the dynamics of measles disease when $\sigma = 0.60$. After thorough study of the Figure, we discovered that if only 60% of exposed individuals at latent period are diagnosed and treated, there is a sharp decline of exposed individuals with time which show a significantly improved result compared with the above two figures. Also, it takes significant much lesser period before noticing a huge improvement for individuals to Recovered from the disease. Similarly, The infectives individuals will significantly take lesser period to decline over time.

In Figure 5, we do the simulation of the model when $\sigma = 0.90$. Looking at the Figure closely, we discovered that if 90% of exposed individuals at latent period are diagnosed and treated, we have a better result in comparison to the previous three results we had stated above.

3.1: Effect of σ on Recovered and Infectives Populations

We carried out an experiment to see how σ affects recovered and infectives populations by varying the value of σ. We use the values 0.0, 0.1, 0.2, 0.5, 0.7 and 1.0 while the values of other parameters are the same as in table 3.

It is discovered from figures 6 and 7 that σ has a significant effects on both the Recovered and Infectives populations over time. As σ increases, the number of recovered also increases given a linear relationship between Recovered and σ. On the other hand,as σ increases, the number of infectives individuals decreases given us an inverse relationship between Infectives and σ. This gives us an intuition that σ plays a vital role in controlling the disease.

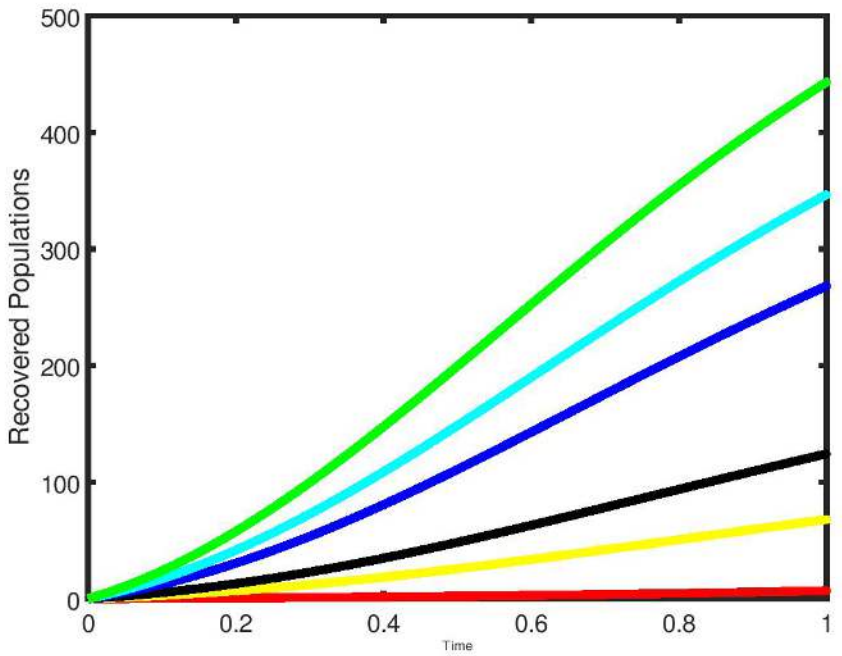

Figure 6: Effects of σ on Recovered Populations over Time

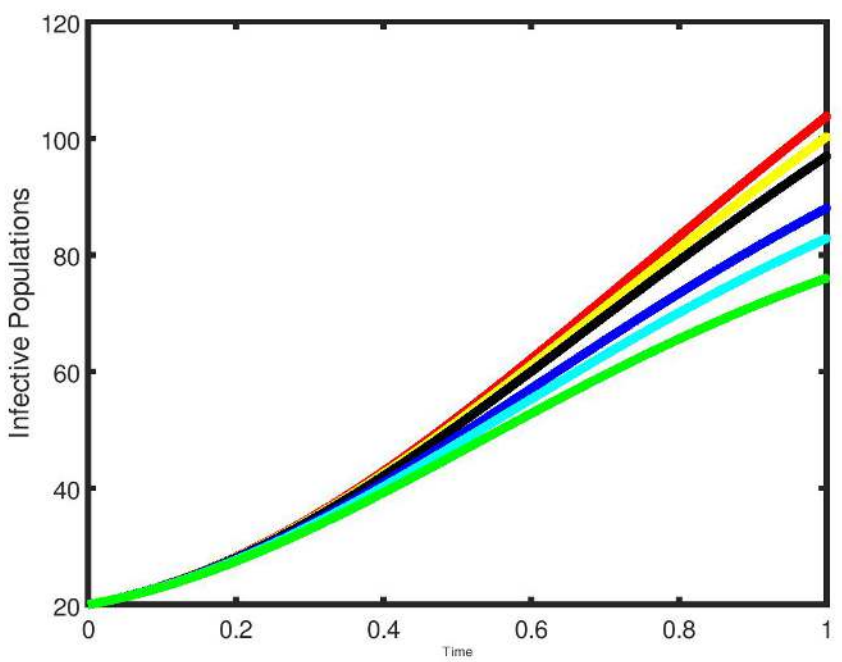

Figure 7: Effects of σ on Infectives Populations over Time

Data: *Red: $\sigma = 0.0$ *Yellow: $\sigma = 0.1$ *Black: $\sigma = 0.2$ *Blue: $\sigma = 0.5$ *Cyan: $\sigma = 0.7$ *Green: $\sigma = 1.0$

3.2: Effect of β on Infectives Populations

We investigated the effects β will have on the infectives populations by varying the value of β by using the values 0.0, 0.1, 0.2, 0.5, 0.7 and 1.0. See figure 8. We take $\sigma = 0.50$. The values of other parameters are the same as in table 3. It is clearly shown that as the values of β increases, the number of infectives populations increases. This gives us a linear relationship between infective populations and β. Thus, the infection is by contact rate. The higher the contact rate, the higher the infection will be.

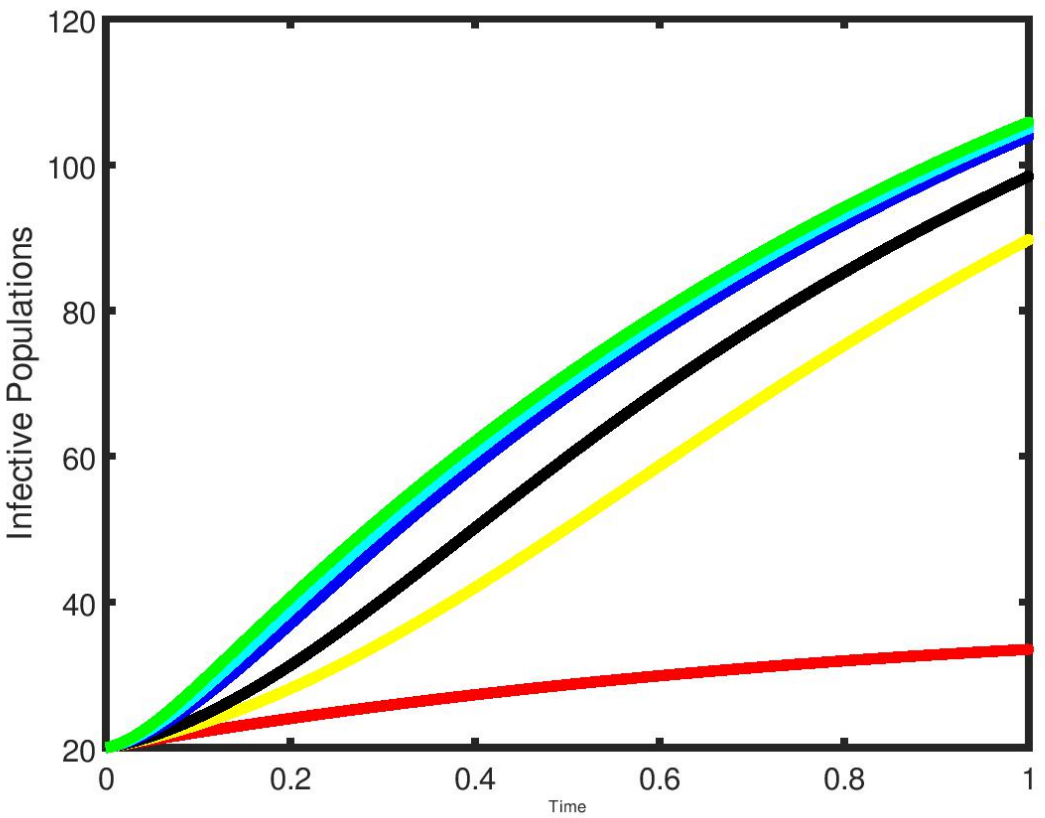

Figure 8: Effects of β on Infectives Populations over Time

Data: Infectives Populations when:
*Red: $\beta = 0.0$ *Yellow: $\beta = 0.1$ *Black: $\beta = 0.2$ *Blue: $\beta = 0.5$ *Cyan: $\beta = 0.7$ *Green: $\beta = 1.0$

4. Conclusion

With the assumed values for our state variables, we modeled a measles disease in Senegal using Compartmental SEIR model to investigate the impact the exposed individuals at latent period can have over the entire

population dynamics in controlling and eliminating the disease. In addition, the existence of solution, uniqueness of solution, stability and numerical simulations of the model with $RK4$ method was presented and adequately discussed.

5. Future Work

The initial goal is to get measles Data of Senegal from Institut de Pasteur, Dakar, but is not currently available. We now used, as a consequences of this, assumed value for our state variables: $S(0)$, $E(0)$, $I(0)$ and $R(0)$. That is, initial values for all the compartments of the SEIR model are assumed in order to do the simulation. Though the simulation results show great success in measles control (reduction and possibly elimination), we are planning, in our future work, to use more detailed and authentic data when we have access to measles data which will be employed in our SEIR model. In practical sense, it is easy to quickly determine the number of Susceptibles, Infectives and Recovered Compartments from a given epidemiological data of measles disease, but how to measure or determined the number of exposed individuals may not be trivial. To overcome this shortcoming in fully integrating this model with real life measles data, more researches are needed to be done in order to devise techniques of accurately determined the number of exposed individuals at latent period before they started showing symptoms of measles disease at a point of collecting and recording data wherever there is an outbreak of the disease.

Data Availability

The data used in our numerical simulations (as provided in table 3 above) are from some already published articles and online sources. They have been duly cited in this work and appropriate links provided to access them.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this work.

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