

**MATHEMATICAL MODEL FOR CONTROL
OF MEASLES EPIDEMIOLOGY**

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Abstract: An SEIR epidemic model is investigated to ascertain the impact of exposed individuals at latent period (individuals who are infected but not yet infectious) on the transmission dynamics of measles. Mathematical analysis is carried out that completely determine the dynamics of the model. The impact of exposed individuals at latent period are discussed through the stability analysis and numerical simulation.

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1. Introduction

The measles virus is a paramyxovirus, genus morbillivirus. Measles is an infectious disease which is highly contagious through person-to-person transmission mode, with over 90% attack rates among susceptible persons. It is the first and worst eruptive fever occurring during childhood. It produces also a characteristic red rash and can lead to serious and fatal complications including pneumonia, diarrhea and encephalitis. Many infected children subsequently suffer blindness, deafness or impaired vision. Measles confer life long immunity from further attacks [7]. Infectious diseases pose a great challenge to both humans and animals world-wide. Control and prevention are therefore important tasks both from a human and economic point of views. Efficient intervention hinges on complete understanding of disease transmission and persistence. Measles is a viral respiratory infection that attacks the immune system and is so contagious that any person not immunized will suffer from the disease when exposed. Children under five years are most at risk. Measles infects about 30 to 40 million children each year and causing mortality of over one million often from complications related to pneumonia, diarrhea and malnutrition [9]. It has been reported as a major cause of childhood morbidity and mortality in Nigeria; 212,183 and 168,107 cases were recorded in 2000 and 2001 respectively [10]. For instance in 2005, Adamawa State, Nigeria experienced 3,974 cases and 238 measles-deaths [3]. [11] reported measles as one of the top five causes of death in children less than five of age in many African countries.

Epidemiological mathematical models are useful in proposing and testing theories, and in comparing, planning, implementing and evaluating various detection, prevention, therapy and control programs. An astounding amount of epidemiological models have been elaborated and applied to infectious diseases [1], [4] and [8]. However, few of the models are able to reproduce some essential aspects of childhood epidemics. In this paper, an SEIR epidemic model is investigated to ascertain the impact of exposed individuals at latent period on the transmission dynamics of measles.

2. Model Equations

Following the classical assumptions of [5], [6] and [4], we formulate a deterministic, compartmental, mathematical model to describe the transmission dynamics of measles. The population is homogeneously mixing and reflect the demography of a typical developing country, as it experiments an exponentially

increasing dynamics. In order to describe the model equations, the total population (N) is divided into four classes: Susceptible (S), Exposed (E), infected (I) and Recovered (R). Here we shall detail the transitions among these four classes as depicted in Figure 1.

The class S of susceptible is increased by birth or immigration at a rate B . It is decreased by infection following contact with infected individuals at a rate β , and diminished by natural death at a rate μ . The class E of exposed individuals is generated through contact with infected individuals at rate β . The class E is decreased by testing and measles therapy at a rate σ , breakthrough into infected class at a rate α and diminished by natural death at a rate μ . The class I of infected individuals is generated by breakthrough of exposed individuals at a rate α . The class is decreased by recovery from infection at a rate γ and diminished by natural death at a rate μ . The model assumes that both recovered exposed individuals and recovered infected individuals become permanently immune to the disease. This generates a class R of individuals who have complete protection against the disease. The class R of recovered individuals diminished by natural death at a rate μ .

Thus, the diagram for the deterministic model is as follows:

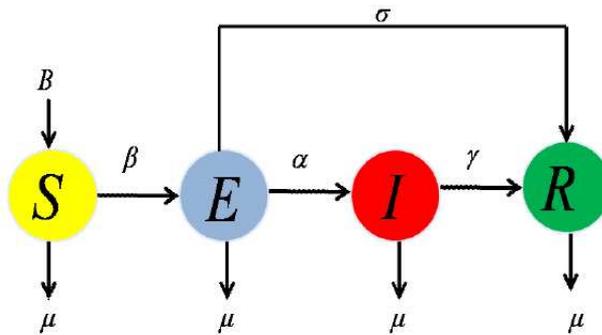


Figure 1: A diagram showing susceptible, exposed, infectious and recovered group

The transitions between model classes can now be expressed by the following system of first order differential equations:

$$\frac{dS}{dt} = B - \beta SI - \mu S, \tag{1}$$

$$\frac{dE}{dt} = \beta SI - (\mu + \alpha + \sigma)E, \tag{2}$$

$$\frac{dI}{dt} = \alpha E - (\mu + \gamma)I, \quad (3)$$

$$\frac{dR}{dt} = \gamma I + \sigma E - \mu R. \quad (4)$$

3. Basic Properties of the Model

Since the model monitors human population, all the associated parameters and state variables are non negative ie $t \geq 0$. It is easy to show that the state variables of the model remain non-negative for all non-negative initial conditions. Consider the biological feasible region

$$\Omega = \left\{ (S, E, I, R) \in R_+^4 : N \rightarrow \frac{B}{\mu} \right\}.$$

Lemma 1. *The closed Ω is positively invariant and attracting.*

Proof. Adding (1)-(4) gives the rate of change of the total population.

$$\frac{dN}{dt} = B - \mu N.$$

Thus, the total human population (N) is bounded by $\frac{B}{\mu}$, so that $\frac{dN}{dt} = 0$ whenever $N(t) = \frac{B}{\mu}$. It can be shown that $N(t) = \frac{B}{\mu} + \left(N_0 - \frac{B}{\mu}\right) e^{-\mu t}$. In particular $N(t) = \frac{B}{\mu}$, if $N(0) = \frac{B}{\mu}$,

Hence, the region Ω is positively invariant and attracts all solution in R_+^4 . \square

3.1. Existence and Uniqueness of Solution for the Model

For the mathematical model to predict the future of the system from its current state at time t_0 , the initial value problem (IVP)

$$x' = f(t, x), \quad x(t_0) = x_0 \quad (5)$$

must have a solution that exist and also unique.

In this subsection, we shall establish conditions for the existence and uniqueness of solution for the model/system of equations. Let

$$f_1(t, x) = B - \beta si - \mu s, \quad (6)$$

$$f_2(t, x) = \beta si - \mu e - \mu e - \mu e, \tag{7}$$

$$f_3(t, x) = \alpha e - \mu i - \gamma i, \tag{8}$$

$$f_4(t, x) = \gamma i + \sigma e - \mu r. \tag{9}$$

So that

$$x' = f(t, x) = f(x). \tag{10}$$

Theorem 1. (see [2]) *Let D' denotes the region*

$$|t - t_0| \leq a, \|x - x_0\| \leq b, x = (x_1, x_2, \dots, x_n), x_0 = (x_{10}, x_{20}, \dots, x_{n0}) \tag{11}$$

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

$$\|f(t, x_1) - f(t, x_2)\| \leq k \|x_1 - x_2\|, \tag{12}$$

whenever the pairs (t, x) and (t, x_2) belong to D' , where k is a positive constant. Then, there exist a constant $\delta > 0$ such that there exist a unique continuous vector solution $\bar{x}(t)$ of the system (5) in the interval $|t - t_0| \leq \delta$.

It is important to note that condition (12) is satisfied by requirement that $\frac{\partial f_i}{\partial x_j}, i, j = 1, 2, \dots, n$ be continuous and bounded in D' .

Lemma 2. *If $f(t, x)$ has continuous partial derivative $\frac{\partial f_i}{\partial x_j}$ on a bounded closed convex domain R , then it satisfies a Lipchitz condition in R .*

We are interested in the region

$$1 \leq \varepsilon \leq R. \tag{13}$$

We look for a bounded solution of the form

$$0 < R < \infty. \tag{14}$$

We shall prove the following existence theorem.

Theorem 2. *Let D' denote the region defined in (12) such that (13) and (14) hold. Then there exist a solution of model system (6)-(10) which is bounded in the region D' .*

Proof. Let

$$f_1 = B - \beta si - \mu S,$$

$$f_2 = \beta si - \mu e - \alpha e - \sigma e,$$

$$f_3 = \alpha e - \mu i - \gamma i,$$

$$f_4 = \gamma i + \sigma e - \mu r.$$

It suffices to show that $\frac{\partial f_i}{\partial x_j}$, $i, j = 1, 2, 3, 4$ are continuous.
Consider the partial derivatives

$$\frac{\partial f_1}{\partial s} = -\beta i^* - \mu, \left| \frac{\partial f_1}{\partial s} \right| = |-\beta i^* - \mu| < \infty,$$

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

$$\frac{\partial f_1}{\partial i} = -\beta s^*, \left| \frac{\partial f_1}{\partial i} \right| = |-\beta s^*| < \infty,$$

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

Similarly

$$\frac{\partial f_2}{\partial s} = \beta i^*, \left| \frac{\partial f_2}{\partial s} \right| = |\beta i^*| < \infty,$$

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

$$\frac{\partial f_2}{\partial i} = \beta s^*, \left| \frac{\partial f_2}{\partial i} \right| = |\beta s^*| < \infty,$$

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

and

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

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

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

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

Finally, we have

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

$$\begin{aligned} \frac{\partial f_4}{\partial e} &= \sigma, \left| \frac{\partial f_4}{\partial e} \right| = |\sigma| < \infty, \\ \frac{\partial f_4}{\partial i} &= \gamma, \left| \frac{\partial f_4}{\partial i} \right| = |\gamma| < \infty, \\ \frac{\partial f_4}{\partial r} &= -\mu, \left| \frac{\partial f_4}{\partial r} \right| = |-\mu| < \infty. \end{aligned}$$

Clearly, all these partial derivatives are continuous and bounded, hence, by theorem (2), there exist a unique solution of (6)-(10) in the region D' .

The first in our analysis is to find equilibria (S^*, E^*, I^*, R^*) from equations

$$0 = B - \beta SI - \mu S, \tag{15}$$

$$0 = \beta SI - (\mu + \alpha + \sigma)E, \tag{16}$$

$$0 = \mu E - (\mu + \gamma)I, \tag{17}$$

$$0 = \gamma I + \sigma E - \mu R. \tag{18}$$

Model (1)-(4) always has a disease free equilibrium $P_0 = \left(\frac{B}{\mu}, 0, 0, 0\right)$. An endemic equilibrium $P^* = (S^*, E^*, I^*, R^*)$ satisfies $S^*, E^*, I^*, R^* > 0$. From the equilibrium equations, we can show that a unique P^* exist with $S = \frac{(\mu + \alpha + \sigma)(\mu + \gamma)}{\beta \alpha}$. For P^* to exist in the feasible region Ω , it is necessary and sufficient that $0 < S^* \leq \frac{B}{\mu}$, or equivalently, $\frac{B}{\mu S^*} \geq 1$. Define

$$R_0 = \frac{1}{S^*} \frac{B}{\mu} = \frac{B\beta\alpha}{\mu(\mu + \alpha + \sigma)(\mu + \gamma)}. \tag{19}$$

Then R_0 is a threshold parameter that determines the number of equilibria.

4. Stability of the Disease-Free Equilibrium

To examine the local stability of the disease-free equilibrium P_0 we evaluate the Jacobian matrix at $P_0 = \left(\frac{B}{\mu}, 0, 0, 0\right)$

$$J(P_0) = \begin{pmatrix} -\mu & 0 & -\frac{B\beta}{\mu} & 0 \\ 0 & -(\mu + \alpha + \sigma) & \frac{B\beta}{\mu} & 0 \\ 0 & \alpha & -(\mu + \gamma) & 0 \\ 0 & \sigma & \gamma & -\mu \end{pmatrix}. \tag{20}$$

We have the stability result that shows that our model is locally asymptotically stable.

Proposition 2. P_0 is locally asymptotically stable if $R_e(\lambda) < 0$.

Proof.

$$|J(P_0) - \lambda I| = \begin{vmatrix} -\mu - \lambda & 0 & -\frac{B\beta}{\mu} & 0 \\ 0 & -(\mu + \alpha + \sigma) - \lambda & \frac{B\beta}{\mu} & 0 \\ 0 & \alpha & -(\mu + \gamma) - \lambda & 0 \\ 0 & \sigma & \gamma & -\mu - \lambda \end{vmatrix} = 0 \quad (21)$$

The eigenvalues of $J(P_0)$ are

$$\lambda_1 = -\mu,$$

$$\lambda_2 = -\mu,$$

$$\lambda_3 = -(\mu + \alpha + \sigma),$$

and $\lambda_4 = -\left(\frac{B\beta\alpha}{\mu} + \mu + \gamma\right)$.

Therefore, $R_e(\lambda) < 0$ since all the parameters are non negative. This proves the proposition. \square

5. Numerical Solution and Results

We carry out numerical simulations of our model (1)-(4) in a hypothetical population size 1000. We will vary key parameters to investigate the impact of exposed individuals at latent period on the transmission dynamics of measles.

Based on epidemiological data we have estimated the values of our model parameters as follows:

$$B = 0.32, \mu = 0.2, \beta = 0.01, \gamma = 0.2, \alpha = 0.01. \quad (22)$$

In (a), diagnosis and exposed individuals therapy is at rate $\sigma = 0.25$. In (b), $\sigma = 0.50$. Other parameter values are the same as (22). In the first set of simulations, we fix the birth and immigration rate B at 0.32, contact rate β at 0.01, death μ at a rate 0.2, rate at which exposed becomes infected α and recovery rate γ are 0.01 and 0.2 respectively. we vary the parameter σ to see the effect of exposed individuals at latent period therapy on the transmission dynamics of measles.

We see in Figure 2(a) that, if only 25% of exposed individuals at latent period are diagnosed and treated, the number of susceptible individuals decreases

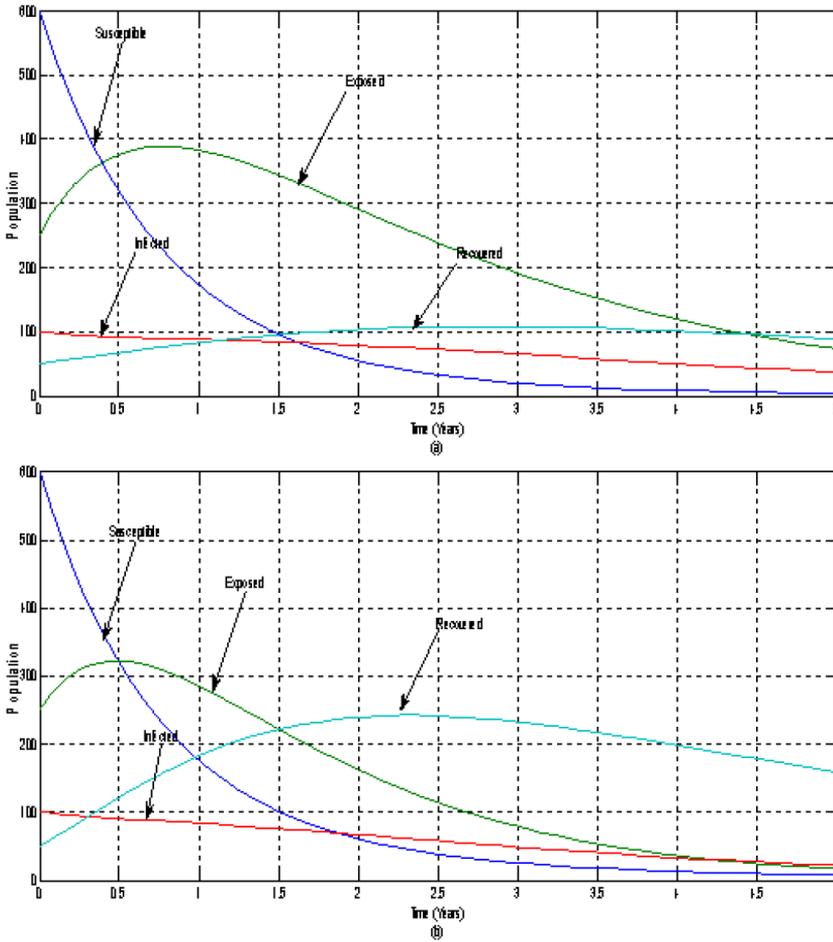


Figure 2: Simulation results showing the impact of testing, diagnosis and therapy of exposed individuals at latent period

significantly, the exposed individuals at latent period increases slightly and decreases steadily. The infected decreases steadily while the recovered increases steadily. If the diagnosis and exposed individuals at latent period therapy is increased from 25% to 50%, a more dramatic change occurs in the disease dynamics: The number of exposed individuals at latent period shows a much greater decline while the number of infected decrease further. This demonstrate that testing, diagnosis and exposed individuals at latent period therapy can be an effective control measure in high measles prevalence countries.

In the second set of simulations, we increased $\sigma = 0.75$ and maintained

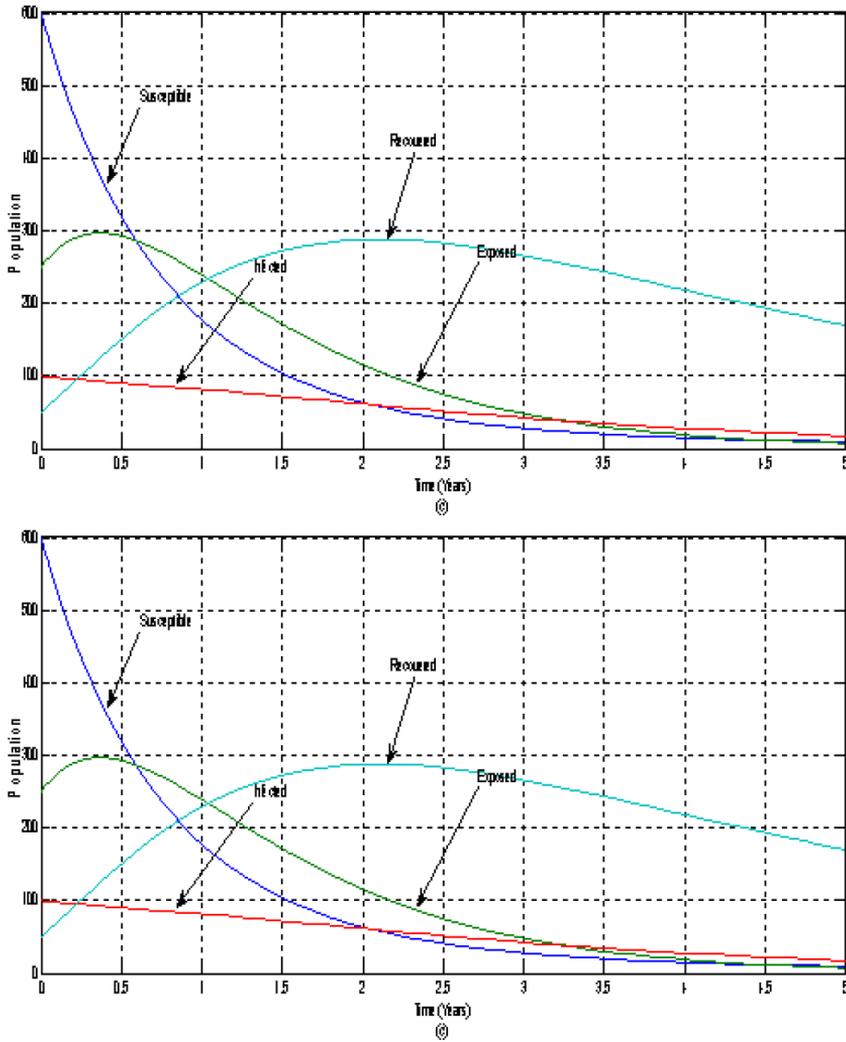


Figure 3: Simulation results showing the impact of testing, diagnosis and therapy of exposed individuals at latent period

other parameters as in (20). We see in Figure 3(a) that the number of exposed individuals at latent period decreases further compared to (b), while recovered increased significantly and infected decrease further compared to Figure 2(b).

Our model simulations demonstrate the challenges of measles infection. The existence of a large number of individuals. Comparing our simulation results in Figure 2(a) and (b), we conclude that, in high measles prevalence countries,

testing, diagnosis and exposed individuals at latent period therapy will have a much greater impact on the disease burden. While this conclusion may have practical implications for the control of measles infections, more realistic models that are specific for measles infection and more detailed data need to be employed to further explore its significance in future study.

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