NIGERIAN JOURNAL OF MATHEMATICS AND APPLICATIONS, VOL. 20, PP. 75-81 (2010).

A DETERMINISTIC MATHEMATICAL MODEL OF MEASLES IN NEWBORN(S) USING THE MSEIR ENDEMIC MODEL.

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ABSTRACT: *In this study we use a Compartmental Mathematical Model (MSEIR) to examine the dynamics of measles spread within a population with constant size. We rely on a compartmental model expressed by a set of differential equations based on the dynamics of measles infection. We examine the stability of the equilibria states with respect to the basic reproductive number R_0 (number of secondary infections); the disease free-state is locally and globally stable and the endemic state is also stable. The model is mathematically and epidemiologically well posed.*

Keywords: Differential equation, Measles, Stability, equilibrium and Basic reproduction number.

1. INTRODUCTION.

Measles is best known for causing a rash in childhood, but measles can affect other parts of the body and sometimes occurs in adults. There are two types of measles, each caused by different viruses. Although both produce a rash and fever, they are really different diseases:

The rubeola virus causes "red measles," also known as "hard measles" or just "measles." Although most people recover without problems, rubeola can lead to pneumonia or inflammation of the brain (encephalitis).

The rubella virus causes "German measles," also known as "three-day measles." This is usually a milder disease than red measles. However, this virus can cause significant birth defects if an infected pregnant woman passes the virus to her unborn child.

Both the rubeola and rubella viruses are spread through the respiratory route. This means they are contagious through coughing and sneezing. In fact, the rubeola virus is one of the most contagious viruses known to man. As a result, it can spread rapidly in a susceptible population. Infected people carry the virus in their respiratory tract before they get sick, so they can spread the disease without being aware of it.

If people are immune to the virus (either through vaccination or by having had measles in the past), they cannot get the disease caused by that virus. For example, someone who had rubeola as a child would not be able to get the disease again. It is to be noted that rubella and rubeola are different viruses. An infection with one of these viruses does not protect against infection with the other.

Measles is a disease that still has the tendency of killing people in developing countries, in fact in March 2010, over 40 children died in a renewed measles outbreak that ravaged nine communities in Southern Ijaw local government area of Bayelsa State in Nigeria.
There are prevention measures like vaccination for this disease but there is no specific treatment or cure for measles.

In this study we will not concern ourselves with the prevention measures or the cure but rather we will center our study on the dynamics of the disease using the well known MSEIR model with modifications to gain insights into the disease. The model we will be proposing will be applicable to the type of measles caused by rubella virus since we will be assuming that mothers infected in the past or with vaccination will pass Immunoglobulins class G (IgG) to their children.

2. THE MODEL

We represent the population density at any time t of the passively-immune newborns, susceptibles, exposed, infected and recovered population by M(t), S(t), E(t), I(t) and R(t) respectively. We also assume that the birth rate μ and natural death rate α are constant, so that we will be able to keep our population N(t) (say) constant too. We can represent the dynamism of the disease with the following diagram;

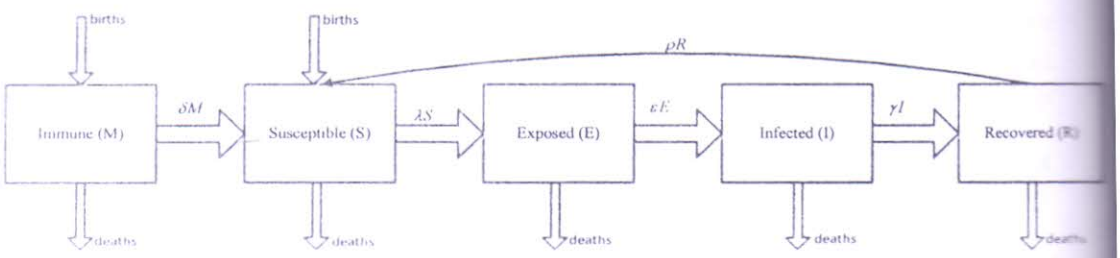

Figure 1 Transfer diagram between the epidemiology classes

Newborns will enter the susceptible class at the rate μS where S is the susceptible class corresponding to newborns whose mothers are susceptible and some will enter the passive immune state at the rate $\mu(N-S)$ corresponding to passive immune class M.

Death will occur to individuals in the susceptible class at the rate αS also newborns in the immune class will migrate to susceptible class at the rate δM . All women would definitely be out of the passively immune class long before their childbearing years; theoretically a passively immune mother would transfer some IgG antibodies to her newborn child, so the infant would have passive immunity. Again death will occur to those in the immune class at the rate μM .

For viral diseases, it is useful to define both a contact rate and the fraction of contacts that can result into transmission, but for directly-transmitted diseases spread primarily by aerosol droplets, transmission may occur by entering a room, hallway, building, etc, that is currently or has been occupied by an infective. Since there is no clear definition of a contact or a transmission fraction, they are replaced by a definition that includes both. An adequate contact is a contact that is sufficient for transmission of infection from an infective to a susceptible. Let the contact rate β be the average number of adequate contacts per person per unit time, so that the force of infection $\omega = \frac{\beta I}{N}$ is the average number of contacts with infectives per unit time. Then the incidence (the number of new cases per unit time) is ωS , i.e, $\frac{\beta IS}{N}$, since it is the number of contacts with infectives per unit time of the S susceptibles. This standard representation $\frac{\beta IS}{N}$ for the incidence is consistent with numerous studies which show that the contact rate β is nearly independent of the

tion size. The people in the exposed class will migrate to the infected class at the rate εE, will occur to the exposed class at the rate αE, the recovery rate from the infectious class is natural death will occur to individuals in the infected class at the rate αI and infection d death at the rate φI. Due to treatment, the rate a which individual in the infected class ve to recovered class is ρR, which is the same thing as the rate of loss of immunity. Then amics of the interaction of the classes is governed by following system of differential ns:

$$\begin{aligned}
&\mu(N-S)-(\delta+\alpha)M \\
&-\frac{\beta SI}{N}+\delta M+(\mu-\alpha)S+\rho R \\
&\frac{\beta SI}{N}-(\varepsilon+\alpha)E \qquad (1.1)\\
&\varepsilon E-(\gamma+\alpha+\varphi)I \\
&\gamma I-(\rho+\alpha)R
\end{aligned}$$

nvenience we will follow the approach of Hethcote (2000) in converting (1.1) to fractions of sses. This can be done by dividing through by the population constant number N and ating s by setting $s=1-m-e-i-r$, where m,s,e,i,r are the proportions (fractions) of pulation that are in the passive-immune, susceptible, exposed, infected, recovered classes tively.

ng so, we will obtain equation (1.2) from equation (1.1).

$$\begin{aligned}
&\mu(m+e+i+r)-(\delta+\alpha)m \\
&\beta i(1-m-e-i-r)-(\varepsilon+\alpha)e \qquad (1.2)\\
&\varepsilon e-(\gamma+\alpha+\varphi)i \\
&\gamma i-(\rho+\alpha)r
\end{aligned}$$

that in equation (1.2) $(m,e,i,r)=(\frac{M}{N},\frac{E}{N},\frac{I}{N},\frac{R}{N})$ respectively and M,E,I,R and N are as ed in equation (1.1) above.

condition for equation (1.2) to hold is that the death rate must be equal to the birth rate so he flow in and out of the population is constant and so the population will be constant, so tion (1.2) becomes;

$$\frac{dm}{dt} = \alpha(e+i+r) - \delta m$$
$$\frac{de}{dt} = \beta i(1-m-e-i-r) - (\varepsilon+\alpha)e$$
$$\frac{di}{dt} = \varepsilon e - (\gamma+\alpha+\varphi)i \qquad (1.3)$$
$$\frac{dr}{dt} = \gamma i - (\rho+\alpha)r$$

in domain Φ

The definition of domain Φ is

$\Phi = \{(m,e,i,r)\}$; $m \geq 0, e \geq 0, i \geq 0$ and $r \geq 0$ with $(m+e+i+r) \leq 1$

The domain Φ is positively invariant, because no solution paths leave through any boundary. The right hand sides of (1.3) are smooth, so that initial value problems have unique solutions that exist on maximal intervals (Hale, 1969).

Since paths cannot leave Φ, solutions exist for all positive time. Thus, the model is mathematically and epidemiologically well posed (Hethcote, 2000).

For our model (Measles dynamics) the linear transfer terms $\delta, \varepsilon, \gamma, \rho$ in (1.3) correspond to waiting times with negative exponential distributions, so that when births and deaths are ignored, the mean passively immune period is $\frac{1}{\delta}$, the mean latent period is $\frac{1}{\varepsilon}$, the mean infectious period is $\frac{1}{\gamma}$, and the mean period of infection-induced immunity is $\frac{1}{\rho}$ (Hethcote etal, 1981). These periods would be $\frac{1}{\delta}$ = 12 months, $\frac{1}{\varepsilon}$ = 15 days, $\frac{1}{\gamma}$ = 8 days and $\frac{1}{\rho}$ =10years .

3. THE BASIC REPRODUCTION NUMBER AND THE EQUILIBRIUM.

3.1 Basic Reproduction Number R_0

The basic reproduction number R_0 (the average number of secondary infection due to introduction of an infected individual into a disease free population) for this model is the same as the contact number σ given by the product of the contact rate β and the average death-adjusted infectious period $\frac{1}{\gamma+\alpha+\varphi}$ times the fraction $\frac{\varepsilon}{\varepsilon+\alpha}$ of exposed people surviving the latent class E.

Thus, the reproductive number R_0 is given as:

$$R_0 = \sigma = \frac{\beta\varepsilon}{(\gamma+\alpha+\varphi)(\varepsilon+\alpha)} \qquad (1.4)$$

This shows that R_0, for this model, is still the average number of secondary infections due to an infective during the infectious period, when everyone in the population is susceptible.

3.2 Disease Free Equilibrium.

The disease free equilibrium is when the immuned class, exposed class, infected class and recovered class are all zero, i.e, $m^0 = 0, e^0 = 0, i^0 = 0, r^0 = 0$ and so $s^0 = 1$.

3.3 Endemic Equilibrium.

At equilibrium, (1.3) becomes:

$$m = \frac{\alpha}{\delta}\left(\frac{\delta(\rho+\alpha)(\gamma+\alpha+\varphi)}{(\alpha+\delta)[(\rho+\alpha)(\gamma+\alpha+\varphi)]+\varepsilon\gamma} + \frac{\varepsilon\delta(\rho+\alpha)}{(\alpha+\delta)[(\rho+\alpha)(\gamma+\alpha+\varphi)]+\varepsilon\gamma} + \frac{\delta\varepsilon\gamma}{(\alpha+\delta)[(\rho+\alpha)(\gamma+\alpha+\varphi)]+\varepsilon\gamma}\right)\left(\frac{\beta\varepsilon-(\gamma+\alpha+\varphi)(\varepsilon+\alpha)}{\beta\varepsilon}\right) \quad (1.5)$$

$$e = \frac{\delta(\rho+\alpha)(\gamma+\alpha+\varphi)[\beta\varepsilon-(\gamma+\alpha+\varphi)(\varepsilon+\alpha)]}{\beta\varepsilon(\alpha+\delta)[(\rho+\alpha)(\gamma+\alpha+\varphi+\varepsilon)+\varepsilon\gamma]} \quad (1.6)$$

$$i = \frac{\varepsilon\delta(\rho+\alpha)(\gamma+\alpha+\varphi)[\beta\varepsilon-(\gamma+\alpha+\varphi+)(\varepsilon+\alpha)]}{\beta\varepsilon(\alpha+\delta)(\gamma+\alpha+\varphi)[(\rho+\alpha)(\gamma+\alpha+\varphi+\varepsilon)+\varepsilon\gamma]} \quad (1.7)$$

$$r = \frac{\gamma\varepsilon\delta[\beta\varepsilon-(\gamma+\alpha+\varphi)(\varepsilon+\alpha)]}{\beta\varepsilon(\alpha+\delta)[(\rho+\alpha)(\gamma+\alpha+\varphi+\varepsilon)+\varepsilon\gamma]} \quad (1.8)$$

From equations (1.4) - (1.8) we have the following unique endemic equilibrium:

$$\begin{aligned} m_e &= \frac{\alpha}{(\alpha+\delta)}\left(1-\frac{1}{R_0}\right) \\ e_e &= \frac{\delta(\rho+\alpha)(\gamma+\alpha+\varphi)}{(\alpha+\delta)[(\rho+\alpha)(\gamma+\alpha+\varphi+\varepsilon)+\varepsilon\gamma]}\left(1-\frac{1}{R_0}\right) \\ i_e &= \frac{\varepsilon\delta(\rho+\alpha)}{(\alpha+\delta)[(\rho+\alpha)(\gamma+\alpha+\varphi+\varepsilon)+\varepsilon\gamma]}\left(1-\frac{1}{R_0}\right) \\ r_e &= \frac{\varepsilon\delta\gamma}{(\alpha+\delta)[(\rho+\alpha)(\gamma+\alpha+\varphi+\varepsilon)+\varepsilon\gamma]}\left(1-\frac{1}{R_0}\right) \end{aligned} \quad (1.9)$$

and

$$s_e = \frac{1}{R_0} \text{ for } R_0 > 0$$

Lemma 3.1

The disease will be eradicated from the population if the basic reproduction number R_0 is one (Hethcote, 2000).

3.4 Local and Global stability of disease-free and endemic equilibra.

Using linearization, the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$ and is an unstable hyperbolic equilibrium with a stable manifold outside Φ and an unstable manifold tangent to a vector into Φ when $R_0 > 1$. On using the Liapunov functional $V = \varepsilon e + (\varepsilon+\alpha)i$ the disease-free equilibrium can be shown to be globally asymptotically stable in domain Φ if $R_0 \le 1$. The Laipunov derivative of V is

$V = [\beta\varepsilon s - (\varepsilon+\alpha)(\gamma+\alpha+\varphi)]i \le 0$ since $\beta\varepsilon \le (\varepsilon+\alpha)(\gamma+\alpha+\varphi)$. Following the approach of (Hethcote etal, 1981), the set where $V = 0$ is the face of Φ with $i = 0$; but $\frac{di}{dt} = \varepsilon e$ on this

See, so that $\dot{i}$ moves off the face unless $e = 0$. When $e = i = 0$, $\frac{dr}{dt} = -\mu r$, so that $r \to 0$.

When $e = i = r = 0$, then $\frac{dm}{dt} = -\delta m$, so $m \to 0$. By definition of Φ, the origin is the only positively invariant subset of the set and so $\dot{V} = 0$; so by Liapunov-Lasalle theorem (Hale, 1969) all paths in Φ approach the origin and so the disease-free equilibrium is globally asymptotically stable in Φ if $R_0 \leq 1$.

The characteristic polynomial of the Jacobian at the endemic equilibrium is of order four and it can be analyze to show that the Routh-Hirwitz criteria are satisfied if $R_0 > 1$ (Li etal, 1995, Simon etal, 1992 and Thieme, 1983). Therefore the endemic equilibrium is locally asymptotically stable when it is in Φ. Lastly, if $R_0 > 1$, then the disease-free equilibrium is unstable and the endemic equilibrium is locally asymptotically stable.

4. CONCLUSION.

By using the concept of reproduction number, we were able to determine the equilibra states (disease-free and endemic) and their stability. The parameter R_0 had enabled us to show that when it is equal to1, the disease will die out in the population and all the individuals in population will be in the susceptible class which is consistent with other studies.

Acknowledgement.

I am grateful to Dr O.D Olayiwola formally of Federal Medical Centre, Bida for his professional guidance during the conduct of this study.

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