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Seasonal Effect on the Dynamics Mathematical Model of Measles

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ABSTRACT
In this paper, we proposed a mathematical model to describe the transmission of measles with seasonal effect. Mathematical model is analyzed by using standard method, the equilibrium points and stability of the equilibrium points is determined. The findings conclude that, the mathematical model for the transmission of measles with seasonal effect consisting of a system of four nonlinear differential equations. We obtained two equilibrium points, disease free equilibrium and endemic equilibrium point. If the relative humidity equal to 0.7 then the basic reproductive number $R_0 < 1$, which the disease free equilibrium point is local asymptotically stable and the disease endemic equilibrium will occur if the relative humidity equal to 0.9, we obtained the basic reproductive number $R_0 > 1$, which the endemic equilibrium point is local asymptotically stable.

INTRODUCTION

Measles is caused by the measles virus, a member of the genus *Morbillivirus* of the family *Paramyxoviridae*., which to be found in the nose and throat of the patient. Measles is one of the most contagious diseases ever known and is an important cause of death and disability among young children worldwide. Measles starts with fever, runny nose, cough, red eyes, and sore throat. It's followed by a rash that spreads over the body. Measles virus is highly contagious virus and spreads through the air through coughing and sneezing. (CDC,2015) Mathematical modeling is a useful tool for understanding and describing the transmission of this disease. Worldwide, measles vaccination has been very effective, preventing an estimated 80 million cases and 4.5 million deaths annually (CDC,2015). Although global incidence has been significantly reduced through vaccination, measles remains an important public health problem. In Thailand, the vaccine against measles when making 1984 the incidence of the disease decreased a lot, especially in children under five years old, but still found the disease was sporadic. And there are occasional outbreaks in the country. Since 2004 – 2014, it found that each year there have been reports of patients increased from January to March. After that, the number of cases started to decrease and increase again in June-August. Make 2008, the year with the highest incidence rate almost three times that of the year with the lowest incidence rate compared the number of cases in the year 2014 and 2015. We found that in the year 2014 the number of patients less than last year in every month (IDEP,2015). In this paper, we proposed a mathematical model to describe the transmission of measles with effect seasonal.

2. Model Formulation:

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In our model, we assume that the human is constant. We formulate the model of measles transmission by using basic ideas taken from epidemiology that is in rain or winter season when the relative humidity is high which effect to measles epidemic. The model is obtained by assuming:

The total human population N is divided into four compartments: Susceptible human denote by S (the members of the human population who may become infected). Infected human denoted denote by X (the infectives at the first phase of infectiousness (Asymptomatic)) and Y is the infectives at the second phase of infectiousness (Symptomatic). Recover human denoted by R (the numbers of recovered individuals).
The dynamics of the disease is depicted in the flow chart shown in **Fig. 1.**

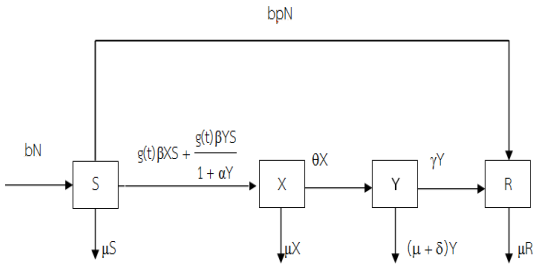

Fig. 1: Flow chart of the dynamics of measles.

The dynamics of measles is described by the following ordinary differential equations:

$$\frac{dS}{dt} = b(1-p)N - g(t)\beta XS - \frac{g(t)\beta YS}{1+\alpha Y} - \mu S \quad (1)$$

$$\frac{dX}{dt} = g(t)\beta XS + \frac{g(t)\beta YS}{1+\alpha Y} - (\theta + \mu)X \quad (2)$$

$$\frac{dY}{dt} = \theta X - (\delta + \gamma + \mu)Y \quad (3)$$

$$\frac{dR}{dt} = \gamma Y + bpN - \mu R \quad (4)$$

with $N = S + X + Y + R$
where

S is the Susceptible individuals,
X is the infective individuals at the first phase of infectiousness (Asymptomatic) ,
Y is the infective individuals at the second phase of infectiousness (Symptomatic) ,
R is the recovered individuals ,
N is the total human population ,
g is the seasonal effects(the relative humidity),
β is the probability of infection upon contact ,
b is the birth rate,
p is the individuals successfully vaccinated against the disease ,
μ is the natural death rate,
θ is the rate at which the asymptomatically infectious become symptomatic,
γ is the recovery rate ,
δ is the death rate induced disease,
α is the measures of the inhibitory effect.

3. ***Analysis of the Model:***

Equilibrium Points:

The system has two equilibrium points; a disease free equilibrium point and an endemic equilibrium point. We obtained these by setting the right hand sides of equations. (1) - (4) to zero. Doing this, we obtained

Disease Free Equilibrium Point: (E_0):

In the absence of the disease**, i.e.,** $X = 0$, $Y = 0$, equation (1), (2) becomes

$$\frac{dS}{dt} = b(1-p)N - g(t)\beta XS - \frac{g(t)\beta YS}{1+\alpha Y} - \mu S \text{ and}$$

$$\frac{dR}{dt} = \gamma Y + bpN - \mu R$$

The solution to this equation is $S = \frac{bN(1-p)}{\mu}$ and $R = \frac{bpN}{\mu}$. The disease free state is

$$E_0 = (\frac{bN(1-p)}{\mu}, 0, 0, \frac{bpN}{\mu})$$

Endemic Equilibrium Point: (E_1):

In the case where the disease is present, by setting

$X^* \neq 0, Y^* \neq 0$. This gives

$$S^* = \frac{\theta bN(1-p)(1+\alpha Y^*)}{g(t)(1+\alpha Y^*)(\delta+\gamma+\mu)\beta Y^* + \theta g(t)\beta Y^* + \theta\mu(1+\alpha Y^*)}$$

$$X^* = \frac{g(t)(1-p)\theta bN\beta Y^*}{(\theta+\mu)[g(t)(1+\alpha Y^*)(\delta+\gamma+\mu)\beta Y^* + \theta g(t)\beta Y^* + \theta\mu(1+\alpha Y^*)] - g(t)\theta bN(1-p)(1+\alpha Y^*)\beta}$$

$$Y^* = \frac{-A_2 \pm \sqrt{A_2^2 - 4A_1A_3}}{2A_1}$$

$$R^* = \frac{\gamma Y^* + bpN}{\mu}$$

Where

$$A_1 = a_3a_4,$$

$$A_2 = a_3a_5 + a_3a_6 + a_7a_8 - a_1a_9,$$

$$A_3 = a_7a_{10} - a_1a_{11} - a_1a_2,\ a_1 = g(t)(1-p),$$

$$a_3 = g(t)(\theta+\mu),\ a_4 = (\delta+\gamma+\mu)^2\alpha\beta,$$

$$a_5 = (\delta+\gamma+\mu)^2\beta,\ a_6 = (\delta+\gamma+\mu)\theta\beta,$$

$$a_7 = (\theta+\mu)(\delta+\gamma+\mu),\ a_8 = \theta\mu\alpha,$$

$$a_9 = (\delta+\gamma+\mu)\theta bN\alpha\beta,\ a_{10} = \theta\mu,\ a_{11} = (\delta+\gamma+\mu)\theta bN\beta$$

Thus

$$E_1(S^*, X^*, Y^*, R^*) = \left(\frac{\theta bN(1-p)(1+\alpha Y^*)}{g(t)(1+\alpha Y^*)(\delta+\gamma+\mu)\beta Y^* + \theta g(t)\beta Y^* + \theta\mu(1+\alpha Y^*)}, X^*, Y^*, \frac{\gamma Y^* + bpN}{\mu}\right)$$

Basic Reproductive Number:

The basic reproductive number is obtained by the next generation matrix. In the notation of Van den Driessche and Watmough (2002), we start with

$$\frac{dx}{dt} = F(x) - V(x) \qquad (5)$$

where $F(x)$ is the matrix of new infectious and $V(x)$ is the matrix of the transfers between the compartments in the infective equations. We obtained

$$F(x)=\begin{bmatrix} 0 \\ g(t)\beta X(\frac{bN(1-p)}{\mu})+\frac{g(t)\beta YbN(1-p)}{\mu(1+\alpha Y)} \\ 0 \\ 0 \end{bmatrix}$$

$$V(x)=\begin{bmatrix} -b(1-p)N+g(t)\beta X(\frac{bN(1-p)}{\mu})+\frac{g(t)\beta YbN(1-p)}{\mu(1+\alpha Y)}+\mu(\frac{bN(1-p)}{\mu}) \\ (\theta+\mu)X \\ -\theta X+(\delta+\gamma+\mu)Y \\ -\gamma Y-bpN+\mu R \end{bmatrix}$$

where $F=\left[\frac{\partial F_i(E_0)}{\partial X_i}\right]$ and $V=\left[\frac{\partial V_i(E_0)}{\partial X_i}\right]$

for all $i,j=1,2,3,4$. This are the Jacobian matrix of $F(x)$ and $V(x)$ at E_0. The basic reproductive number, R_0, is the threshold for indicating the degree of epidemiology of the disease. It can be determined by noting that

$$R_0=\rho(FV^{-1})$$

For our model, the Jacobian matrices are

$$F=\begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & g(t)\beta(\frac{bN(1-p)}{\mu}) & g(t)\beta(\frac{bN(1-p)}{\mu}) & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \text{ and}$$

$$V=\begin{bmatrix} \mu & g(t)\beta(\frac{bN(1-p)}{\mu}) & g(t)\beta(\frac{bN(1-p)}{\mu}) & 0 \\ 0 & \theta+\mu & 0 & 0 \\ 0 & -\theta & \delta+\gamma+\mu & 0 \\ 0 & 0 & -\gamma & \mu \end{bmatrix}$$

The inverse of is V

$$V^{-1}=\begin{bmatrix} \frac{1}{\mu} & \frac{g(t)(\gamma-\theta)\beta bN(1-p)}{\mu^3(\theta+\mu)(\delta+\gamma+\mu)} & \frac{g(t)\beta bN(1-p)}{(\delta+\gamma+\mu)\mu^2} & 0 \\ 0 & \frac{1}{\theta+\mu} & 0 & 0 \\ 0 & \frac{\theta}{(\theta+\mu)(\delta+\gamma+\mu)} & \frac{1}{\delta+\gamma+\mu} & 0 \\ 0 & \frac{\theta\gamma}{(\theta+\mu)(\delta+\gamma+\mu)} & \frac{\gamma}{\delta+\gamma+\mu} & \frac{1}{\mu} \end{bmatrix}$$

This leads to

$$FV^{-1}=\begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & \frac{g(t)\beta bN(1-p)[(\delta+\gamma+\theta)]}{\mu(\theta+\mu)(\delta+\gamma+\theta)} & \frac{g(t)\beta bN(1-p)}{\mu(\delta+\gamma+\theta)} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

Thus,

$$R_0=\frac{g(t)\beta bN(1-p)[(\delta+\gamma+\mu)+\theta]}{\mu(\theta+\mu)(\delta+\gamma+\theta)} \qquad (6)$$

Local Asymptotically Stability:

The local stability of an equilibrium point is determined from the Jacobian matrix of the ordinary differential equation (1) – (4) evaluated at E_0 . The Jacobian matrix at E_0 is

$$J_0=\begin{bmatrix} -\mu & \frac{-g(t)\beta bN(1-p)}{\mu} & \frac{-g(t)\beta bN(1-p)}{\mu} & 0 \\ 0 & -\left[\frac{\mu(\theta+\mu)-g(t)\beta bN(1-p)}{\mu}\right] & \frac{g(t)\beta bN(1-p)}{\mu} & 0 \\ 0 & \theta & -(\delta+\gamma+\mu) & 0 \\ 0 & 0 & \gamma & -\mu \end{bmatrix}$$

The eigenvalues of the J_0 are obtained by solving $det(J_0-\lambda I)=0$. From this, we obtain the characteristic equation,

$$(-\mu-\lambda)^2[\lambda^2+(b_2+b_3)\lambda+(b_2b_3-\theta b_1)]=0$$

$$(-\mu-\lambda)^2[\lambda^2+A\lambda+B]=0$$

where

$$b_1=\frac{g(t)\beta bN(1-p)}{\mu},$$

$$b_2=\frac{\mu(\theta+\mu)-g(t)\beta bN(1-p)}{\mu},$$

$$b_3=(\delta+\gamma+\mu)$$

$$A=b_2+b_3,\ B=b_2b_3-\theta b_1$$

From the characteristic equation, we see that two eigenvalues are $\lambda_{1,2}=-\mu<0$. The other two are the solutions of the characteristic equation The roots of this equation will be negative if two coefficients satisfied with the Routh-Hurwitz criteria (Allen,2006). 1) $A>0$ 2) $B>0$

Disease Endemic Equilibrium Point:

To determine the stability of the endemic equilibrium point. We examine the eigenvalues of Jacobian matrix at E_1 , which is

$$J_1=\begin{bmatrix} -\left[g(t)\beta X+\frac{g(t)\beta Y}{1+\alpha Y}+\mu\right] & -g(t)\beta S^* & -\frac{g(t)\beta S^*}{(1+\alpha Y^*)^2} & 0 \\ g(t)\beta X^*+\frac{g(t)\beta Y^*}{1+\alpha Y^*} & -[(\theta+\mu)-g(t)\beta S^*] & \frac{g(t)\beta S^*}{(1+\alpha Y^*)^2} & 0 \\ 0 & \theta & -(\delta+\gamma+\mu) & 0 \\ 0 & 0 & \gamma & -\mu \end{bmatrix}$$

Where are given by equations (5) . The characteristic equation of Jacobian matrix at E1 given by equations (1) – (4), becomes,

$$(-\mu-\lambda)[\lambda^3+D_1\lambda^2+D_2\lambda+D_3]=0$$

where

$$C_1=g(t)\beta X^*+\frac{g(t)\beta Y^*}{1+\alpha Y^*}+\mu,\ C_2=g(t)\beta X^*+\frac{g(t)\beta Y^*}{1+\alpha Y^*},$$

$$C_3=-g(t)\beta S^*,\ C_4=[(\theta+\mu)-g(t)\beta S^*],\ C_5=-\frac{g(t)\beta S^*}{(1+\alpha Y^*)^2},\ C_6=\frac{g(t)\beta S^*}{(1+\alpha Y^*)^2},\ C_7=\delta+\gamma+\mu$$

$$D_1=C_1+C_4+C_7$$

$$D_2=C_1C_4+C_1C_7+C_4C_7-\theta C_6$$

$$D_3=C_1C_4C_7-\theta C_6C_1$$

The three eigenvalues of $\lambda^3+D_1\lambda^2+D_2\lambda+D_3=0$ will have negative real part if they satisfy the Routh - Hurwitz criteria (Allen,2006), that is $D_1D_2>D_3$.

4 Numerical Results:

The value of parameters used in the numerical simulation are given in Table 1.

Stability of the disease free state: Using the values of parameters listed in Table 1. We find the eigenvalues and basic reproductive number to be:

$\lambda_1 = -0.1,\ \lambda_2 = -0.1,\ \lambda_3 = -0.934729,\ \lambda_4 = -0.105271$ and $R_0 = 0.506374 < 1$

Since all of the eigenvalues are negative and the basic reproductive number is less than one, the equilibrium state will be the disease free state E_0, as seen in **Fig. 2**

Table 1: Parameter values used in numerical simulations at disease free state.

Parameter	Values	Unit
N	10,000	person
g	0.006	-
β	0.08	-
b	0.1	day^{-1}
p	0.9	-
μ	0.1	day^{-1}
θ	0.9	day^{-1}
γ	0.9	day^{-1}
δ	0.02	day^{-1}
α	0.08	day^{-1}

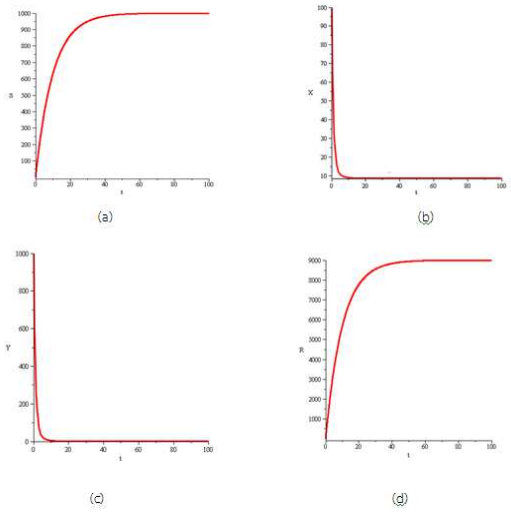

Fig. 2: The time series of (a) the susceptible human , (b) the infectives at the first phase of infectiousness (Asymptomatic), (c) infectives at the second phase of infectiousness (Symptomatic) and (d) recovered individuals. As shown, all the state variables approach to the disease free state .is seen, all the state variables approach their disease free state values $E_0 = (1000, 0, 0, 9000)$.

Stability of the endemic state: Using the values of parameter listed in Table 1. except the value of g(t) we set to be equal 0.7. This values represented the value of the relative humidity in rainy or winter season.

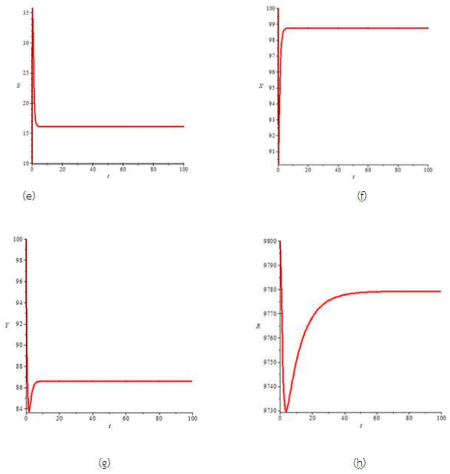

Fig. 3: The time series of (a) the susceptible , (b) the infectives at the first phase of infectiousness (Asymptomatic), (c) infectives at the second phase of infectiousness (Symptomatic) and (d) recovered individuals. Only the value of g(t) has been changed to g(t) = 0.7. All the state variables approach to endemic state $E_1 = (16.0723, 98.3928, 86.8172, 9781.3544)$

The eigenvalues and basic reproductive number become greater than one and the outcome is quite different:

$\lambda_1 = -0.1,\ \lambda_2 = -1.038694,\ \lambda_3 = -1.173663,\ \lambda_4 = -5.129488$ and $R_0 = 105.4117647 > 1$

Since all of the eigenvalues are to be negative and the basic reproductive number is greater than one, the equilibrium state will be the endemic state, E_1 as demonstrated in **Fig. 3.**

5 Discussion and Conclusion:

In this paper, we proposed a transmission model of measles by take into account the seasonal effect. From Fig. 2, we can see that when seasonal effects g = 0.006 the basic reproductive number $R_0 = 0.506374$ which less than one in this case the disease will not occur. But when we change the value of g = 0.7 the basic reproductive number $R_0 = 105.4117647$ which is greater than one in this case the disease will persist as the study of Suksawat and Naowarat(2014). Then the disease will persist in the community. We conclude that if the relative humidity is high, the number of measles infection will be increase.

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