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STUDY OF A MATHEMATICAL MODEL FOR MEASLES WITH PERIODIC VARIATION IN SUSCEPTIBILITY

B. Uma Maheswari[1], K.L. Vasundhara[2], Bhagya Lakshmi Kuntumalla[3], P. Mahendra Varma[4], P. Prashanth Kumar[5] and S. Balamuralitharan[6]

[1]Assistant Professor, Department of Mathematics, Matrusri Engineering College, Saidabad, Hyderabad,Telangana, India. gudipatiuma1977@gmail.com

[2]Associate Professor, Department of Mathematics, Stanley College of Engineering and Technology for Women, Hyderabad, Telangana, India. vasundhara.yerasuri@gmail.com

[3]Associate Professor, Department of Mathematics, CMR Technical Campus, Kandlakoya(v), Medchal road, Hyderabad, Telangana, India. bhagyalakshmi.hns@cmrtc.ac.in

[4]Assistant Professor, Department of H&S, Narasimha Reddy Engineering College, Hyderabad, Telangana, India. mahendravarma12@gmail.com .

[5]Professor, Department of Mathematics, ACE Engineering College, Hyderabad, Telangana, India. prashanthkumar@aceec.ac.in

[6]Professor, Adjunct Faculty, Department of Mathematics, Saveetha School of Engineering, SIMATS, Saveetha University, Chennai 602105, Tamil Nadu, India.

Email Id: balamurali.maths@gmail.com

Abstract

In this paper, we proposed a mathematical model for measles which is defined by two ordinary differential equations (ODEs) for childhood disease measles. We analyzed the model by studying the periodic variations in susceptibility and according to the infection oscillates with time. We linearize the model and calculate the equilibrium points at those points how the frequency of the disease varies with time. The model resembles SIR model whether the population is large or small. Transmission occurs through respiratory droplets, making it one of the most contagious human diseases, compared with a basic reproduction number of ranges. The numerical simulation illustrates the validation of the model.

Keywords: Measles, Periodic Variation, Susceptibility, ODE, Reproduction Number.

1. Introduction

Measles is a highly contagious viral disease caused by the measles virus, a single-stranded, negative-sense RNA virus of the *Paramyxoviridae* family. It primarily affects children but can infect individuals of any age who are unvaccinated or immune compromised. Transmission occurs through respiratory droplets, making it one of the most contagious human diseases, with a basic reproduction number (R_0) ranging from 12 to 18.

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Measles is one of the most infectious diseases and has severe complications, including pneumonia, encephalitis (inflammation of the brain) and death especially in malnourished children and those with weakened immune systems [1].

Vaccination, particularly the Measles-Mumps-Rubella (MMR) vaccine, has dramatically reduced global measles incidence and mortality. However, outbreaks still occur due to gaps in immunization coverage, vaccine hesitancy, and healthcare disparities.

Historical Perspective

- Measles has been described for centuries, with early clinical descriptions by Persian physician Rhazes in the 9th century.
- The measles virus was isolated in 1954 by John Enders and colleagues, paving the way for vaccine development.

Epidemiological Studies

- WHO reports indicate a dramatic global decrease in measles deaths—from 872,000 in 1999 to about 60,000 in 2020 due to immunization efforts [2].
- However, resurgence in various countries has been reported, particularly in low-vaccine-coverage regions or communities with vaccine resistance.

Vaccination and Public Health

- Multiple studies have emphasized the effectiveness of the MMR vaccine, which provides approximately 93% protection after one dose and 97% after two.
- The Global Vaccine Action Plan (GVAP) aimed to eliminate measles in five WHO regions by 2020, though the target was not fully achieved due to inconsistent coverage.

Outbreak Analysis

- Outbreak investigations, such as the 2019 outbreaks in the U.S. and parts of Europe, identified unvaccinated populations as the primary risk group.
- Research links recent measles resurgence to misinformation and declining vaccine confidence, as shown in studies by Larson et al in 2018.

Virology and Pathogenesis

- Recent molecular biology research focuses on the immune suppression caused by measles, which can persist for weeks to months after recovery ("immune amnesia").
- Genetic sequencing of virus strains contributes to tracking transmission chains and understanding outbreak dynamics.

Challenges and Future Directions

- Continued surveillance, outbreak response, and global vaccination campaigns are essential.
- Integration of molecular tools, health education, and policy interventions remains critical to achieving long-term eradication.

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2. SIR and SEIR models of measles

The SIR model is given by Kermack and McKendrick [3]. In the SIR models one considers horizontal incidence, which is the rate of infection of susceptible individuals when they meet infected people. S(t) is the number of susceptible at time t, I(t) is the number of infectives taking the total population size to be N, then s(t) = S(t)/N and i(t) = I(t)/N are the susceptible and infectious numbers, that is if one normalizes the parameters with respect to the population size N. The following SIR Model is defined as below:

$$s'(t) = -\beta si,\ \ s(0) = s_0 \geq 0$$
$$i'(t) = \beta si - \gamma i,\ \ i(0) = i_0 \geq 0$$
$$r'(t) = 1 - s(t) - i(t)$$

SEIR model is organized as below:

$$s'(t) = -\beta si, \qquad s(0) = s_0 \geq 0$$
$$e'(t) = \beta si - \epsilon e, \qquad e(0) = e_0 > 0$$
$$i'(t) = \epsilon i, \qquad i(0) = i_0 > 0$$
$$r'(t) = 1 - s(t) - e(t) - i(t)$$

where β is the contact rate, $1/\gamma$ is the average infectious period, $\frac{1}{\epsilon}$ is the average latent period, and σ = β/γ is the contact number and $s + e + i + r = 1$.

3. Modeling Measles Epidemics

The mathematical model for the spread of measles was formulated by Hamer in 1906 [4].

A standard SIR model is used to study the dynamics of measles epidemics (susceptible-infective-recovered) [5], which is driven by variations in susceptibility [6]. Several authors discussed the transmission of measles disease using extension of SIR Model [7]. The dynamics of the disease is investigated through some mechanisms underlying the observed wild-type measles infection dose responses using the Akaike Information Criterion (AIC) to evaluate relevant biological hypotheses and their respective model parameterizations [8]. The measles epidemics also approaches fractional modeling for the transmission dynamics of disease with double-dose vaccination [9]. Using Ethiopian data of measles, the stability analysis of the dynamics of measles was matched and it analyzes the endemic status of the disease using reproduction number [10]. Using computational simulations on graph-based models to analyze how vaccination affects the spread of infectious diseases. The results demonstrate that adequate vaccination coverage is critical for halting outbreaks, with a marked reduction in disease spread observed as the fraction of vaccinated individuals increases [11]. A mathematical model identifies the most influential parameter on the threshold quantities and study of global sensitivity analysis by using the Partial Rank Correlation Coefficient (PRCC). The result from this analysis informs the most impactful parameter that contributes most to the spread and control of the disease [12]. Although measles is one of only a handful of human pathogens that

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could be eradicated, and despite the broad availability of an intervention tool that could make this possible an effective and inexpensive measles vaccine with decades of use and an excellent safety track record, the disease remains a leading cause of childhood disease and death globally [13]. We proposed a mathematical model for measles which is defined by two ordinary differential equations:

$$\frac{dx}{dt} = \mu - \mu x - N\beta xy(1 + \delta\beta \sin \omega t)$$

$$\frac{dy}{dt} = N\beta xy(1 + \delta\beta \sin \omega t) - (\mu + \vartheta)y \tag{1}$$

where

$N =$ Number of populations

$x =$ Number of susceptibles expressed as a fraction of N

$y =$ Number of infectives expressed as a fraction of N

$\mu =$ Death rate = 0.04years^{-1}

$\beta =$ Transmission coefficient (Susceptibility to disease)

$\delta\beta =$ Fractional variation in susceptibility

$\vartheta =$ Rate of recovery from disease = 24 hours^{-1}

$\omega =$ The angular frequency of oscillation in susceptibility = 2π/period of oscillation

We choose two different values of the parameter ω in both the figures. In figure 2 we plot the graph for a longer time t. When there are no variations in susceptibility ($\delta\beta = 0$) the values for the proportion of susceptibles and infectives are

$$x_0 = \frac{\mu+\vartheta}{N\beta}$$

$$y_0 = \frac{\mu}{\mu+\vartheta}(1 - x_0)$$

when $\delta\beta > 0$ is small, Equation (1) can be approximated using the process of linearization. Set

$$x = x_0 + x_1 \tag{2}$$

$$y = y_0 + y_1 \tag{3}$$

where x_1 and y_1 are the change in x and y from their equilibrium values. Putting Equations (2) and (3) in equation (1) and neglecting higher-order terms gives

$$\frac{dx_1}{dt} \approx -(N\beta y_0 + \mu)x_1 - (\mu + \vartheta)y_1$$

$$-(\mu + \vartheta)y_0\delta\beta \sin \omega t \tag{4}$$

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$$\frac{dy_1}{dt} \approx N\beta y_0 x_1 + (\mu + \vartheta) y_0 \delta\beta \sin \omega t \qquad (5)$$

Equations (4) and (5) represent a linear differential system $(\mu + \vartheta) y_0 \delta\beta \sin \omega t$

The damping factor is given by

$$\rho = \frac{N\beta}{2(\mu+\vartheta)} \sqrt{\frac{\mu}{N\beta-(\mu+\vartheta)}} \qquad (6)$$

For the values of the data used ρ is small $(\rho \approx 0.05)$. This indicates that there is a little damping in the system where

$$\omega_r = \sqrt{1 - \rho^2 \omega_n} \approx \omega_n \qquad (7)$$

and
$$\omega_n = \sqrt{\mu[N\beta - (\mu + \vartheta)]} \qquad (8)$$

If the driving term is $\delta\beta \sin \omega t$, the linear reaction of the population fraction is phase-shifted version of the input. There is a different amplitude and different phases and same frequency. Thus, the output is

$$\delta\beta\, A(\omega) \sin[\omega t + \varphi(\omega)],$$

where

$$A(\omega) = \frac{1}{N\beta} \sqrt{\frac{\omega^2+\mu^2}{\left(1-\frac{\omega^2}{\omega_n^2}\right)^2 + 4\rho^2\omega^2/\omega_n^2}}$$

$$\varphi(\omega) = \tan^{-1}\left(\frac{\omega}{\mu}\right) - \tan^{-1}\left(\frac{2\rho\omega\omega_n}{\omega_n^2-\omega^2}\right)$$

The frequency period is

$$T = \frac{2\pi}{\sqrt{\mu[N\beta-(\mu+\vartheta)]}} \qquad (9)$$

The inter epidemic interval is given by $N\beta$

Since $\mu \ll \vartheta$, the expression can be approximated to $T = 2\pi\sqrt{AD}$ (10)

4. Numerical Simulation

In this section, we have discussed numerical analysis study of a mathematical model for measles with periodic variation in susceptibility [14]-[17]. The comparative numerical part for the final susceptible in figure1 and figure 2 respectively.

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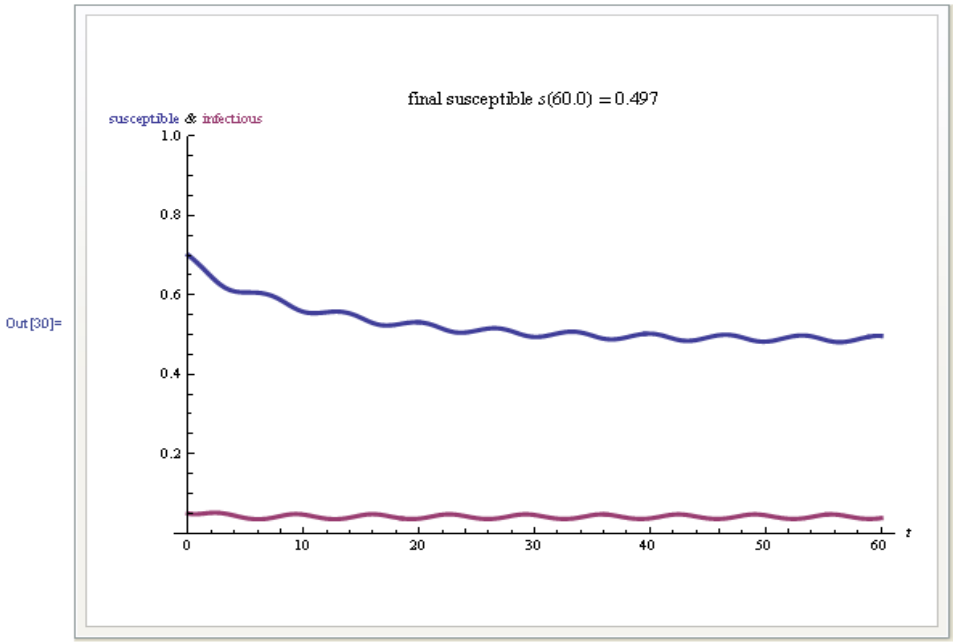

Figure 1: A simulation of the model with $\mu = 0.04$, $\delta\beta = 0.3, N\beta = 1$, $\vartheta = 24$ hours, $\omega = 0.3$ and $t = 60$ hours.

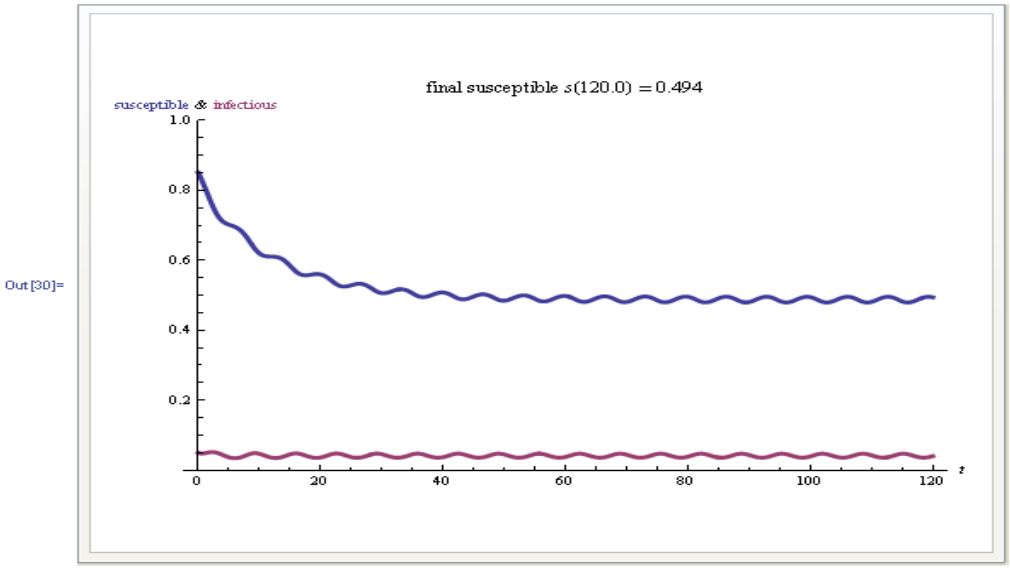

Figure 2: A simulation of the model with $\mu = 0.04, \delta\beta = 0.3, N\beta = 1, \vartheta = 24$ hours, $\omega = 0.15$ and $t = 120$ hours.

From Figures 1 and 2 the periodic variation in susceptibility makes the susceptible and infected oscillate with time. We model the above equations (1) by taking two different considerations into the problem.

There are two cases, such as defined clearly as below:

Case I:

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When N the size of the population is small, this could be modeled on a sparsely populated region (see, Figure 3), then the term $-N\beta xy(1+\delta\beta\sin\omega t)$ can be neglected as it would be much smaller in comparison to the remaining terms in the equations and the equations become:

$$\frac{dx}{dt} = \mu - \mu x$$

$$\frac{dy}{dt} = -(\mu+\vartheta)y$$

Then $\frac{dy}{dx} = \frac{-(\mu+\vartheta)y}{\mu-\mu x}$

The solution of these equations is $x + y - xy = c$

where c is an arbitrary constant.

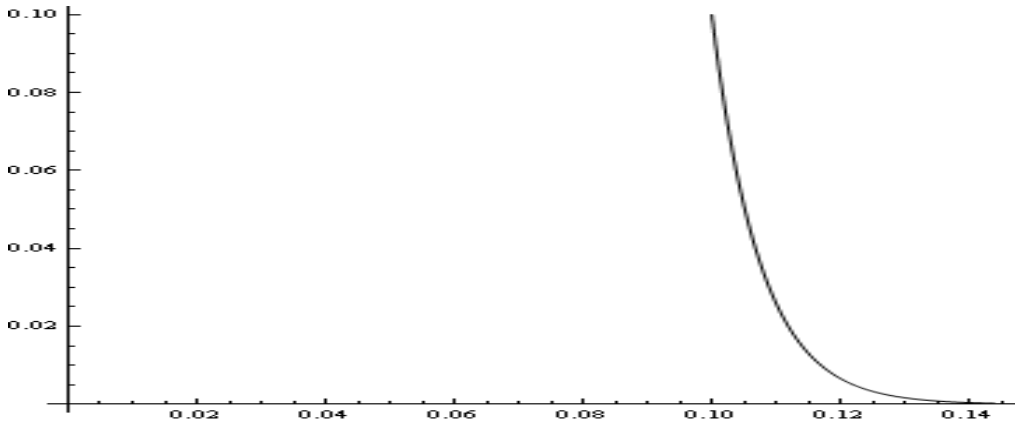

Figure 3: By taking values for $\mu = 0.1$, $\vartheta = 12$, $x(0) = 0.1$, $y(0) = 0.1$, it is the graph shown above.

Case II:

If one considers the epidemic in a densely populated region of the earth (see, Figure 4), then the term $N\beta xy(1+\delta\beta\sin\omega t)$ dominates over the other two terms in the respective equations for x and y. The other terms can be ignored. In this case the equations become:

$$\frac{dx}{dt} = -N\beta xy(1+\delta\beta\sin\omega t)$$

$$\frac{dy}{dt} = N\beta xy(1+\delta\beta\sin\omega t)$$

then $\frac{dx}{dt} + \frac{dy}{dt} = 0$

The solution is $x + y = c_1$ where c_1 is an arbitrary constant.

That is the sum of susceptible and infectives are constant.

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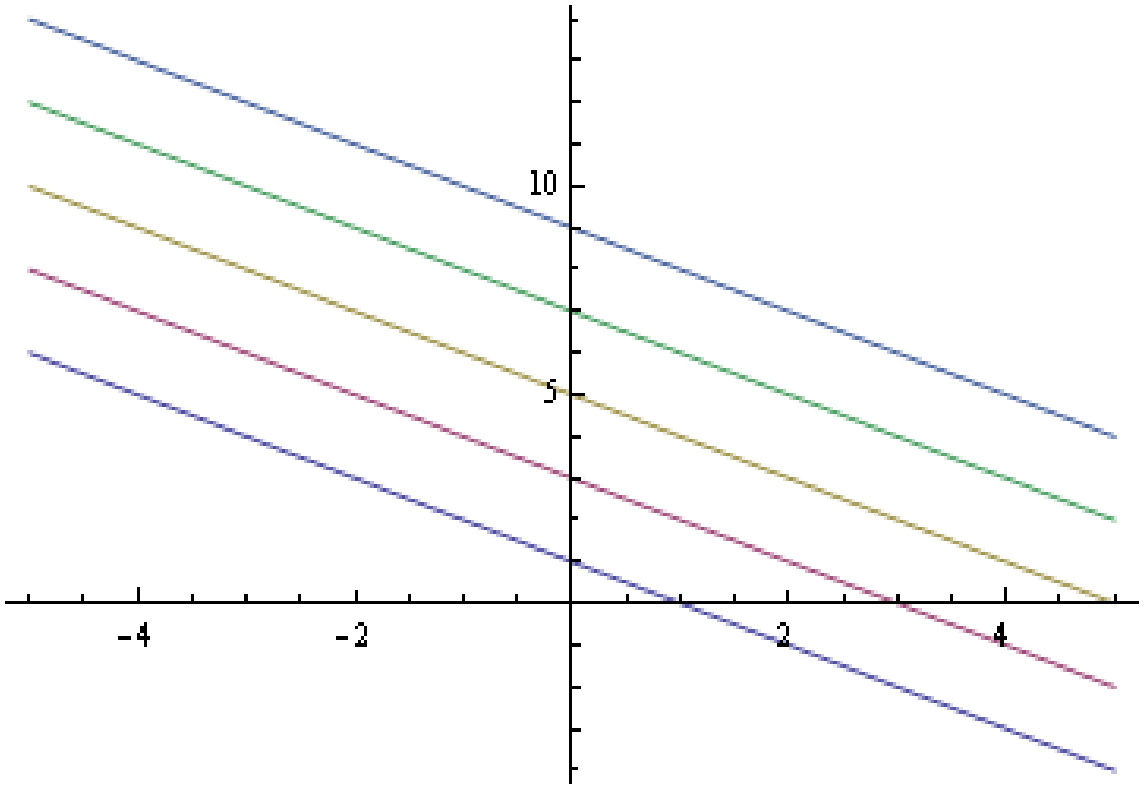

Figure 4: By taking values for $\mu = 0.1$, $\vartheta = 12$, $x(0) = 0.1$, $y(0) = 0.1$, $c_1 = 1$ to 10, the graph is shown above.

5. Conclusion

Measles is a highly contagious disease which can be transmitted easily to susceptible populations through direct contact or from any transmissible media. In this model for measles the control parameter is ω which changes the period of $\sin \omega t$. At a particular value of ω there is a variation in susceptibility, and the disease follows a periodic fashion. We could then say that for a certain periodic variation in susceptibility the disease itself could be periodic. From Case 1 and 2, we conclude that there is no change in periodicity of the coefficient of the variation and both susceptible and infectives are constant in this disease. Furthermore, uncertainty will always exist during the estimation of the parameters, and it will always influence model predictions.

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