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Treatment of dynamical nonlinear Measles model: An evolutionary approach

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Abstract

Measles is a respiratory system infection caused by a Morbillivirus genus virus. The disease spreads directly or indirectly through respiration from the infected person's nose and mouth after contact with fluids. The vast population of infects in developing countries is yet at risk. Generally, the mathematical model of Measles virus propagation is nonlinear and therefore changeable to solve by traditional analytical and finite difference schemes by processing all properties of the model like boundedness, positivity feasibility. In this paper, an unconditionally convergent semi-analytical approach based on modern Evolutionary computational technique and Padé-Approximation (EPA) has been implemented for the treatment of non-linear Measles model. The convergence solution of EPA scheme on population: susceptible people, infective people, and recovered people have been studied and found to be significant. Eventually, EPA reduces contaminated levels very rapidly and no need to supply step size. A robust and durable solution has been established with the EPA in terms of the relationship between disease-free equilibrium in the population. When comparing the Non-Standard Finite Difference (NSFD) approach, the findings of EPA have shown themselves to be far superior.

Keywords: Optimization, Epidemiological Measles Model, Padé-approximation , Differential Evolution, Penalty Function
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1. Introduction

Studies of epidemiology in the human population play an important role in understanding the human population's disease. Mathematical epidemiology's work often consists of model construction, parameter estimation, and model sensitivity investigation to change parameters and numerical simulations. Mathematical models are used by epidemiologists to understand the nature of how diseases spread in the past. Measles disease is highly contagious with an individual's 90 percent chance of infection. Every year, Measles infects about 30 to 40 million kids. The disease was and remains a major killer of the world's children. Despite the introduction of the Measles vaccine in 1963, as far back as 1980, Measles caused an estimated 2.6 million deaths per year. The surviving children eventually experience blindness, loss of sight, damage to the brain, and death [28]. Worldwide, Measles vaccination was highly efficient, preventing an estimated 80 million instances and 4.5 million fatalities per year [16]. While there has been a significant reduction in worldwide incidence through vaccination, Measles continues a significant public health issue. Since vaccination coverage is not evenly high globally, Measles is the world's leading vaccine-preventable child killer; Measles is estimated to have caused 614000 fatalities annually globally in 2002, with more than half of the fatalities from Measles in sub-Saharan Africa [5]. The research of this kind helps to understand the ratio of disease spread in the population and to control their parameters [7]. These types of diseased models are often called infectious diseases Measles, rubella, chickenpox, mumps, aids, and gonorrhea syphilis are examples of infectious disease [15]. Rubella virus is a highly infectious illness which is also known as morbilli or Measles. The virus can be found in the mucus of the throat, the nose of an infected adult and child. Measles symptoms caused by the Rubella virus always included fever, coryza, conjunctivitis, and at least one of the three Cs-coughs. Symptoms appear after the initial infection about 9-11 days [14]. The epidemiological models are crucial processes for investigating and acquiring improved development information using a mathematical tool based on arithmetic and numerical analysis, influence, and the derivative mechanisms, especially where an analysis solution is not available. Some recent studies like [9, 11, 24] also investigate these kinds of developments. Most of the methods like traditional analytical and finite difference schemes does not handle the properties of the model like boundedness, positivity, feasibility. There is the dire need of developing such a method that may be capable of handling these properties and give true insights into model dynamics. In recent years a lot of sophisticated meta-heuristics have been introduced to solve the most complex problems by transforming them into problems of optimization [17, 20, 18, 2]. Improvisations to differential equations of these suggested metaheuristics can be seen in [12, 19] as well, but the application of these meta-heuristics [26, 25, 22, 21] to widely distributed and disease models are difficult to see. The study conducted by Farhan et al. for the treatment of the HIV/AIDS epidemic model with vertical transmission [6], Hepatitis-B Model [23] and Smoking Model [1] by using evolutionary Padé-approximation, extend this work to solve under line measles dynamical model. The contribution of the current work can be summarized as under: Points of equilibrium (virus equilibrium (VE) and virus-free equilibrium (VFE)) have been calculated and analyzed for their stability analyses. A new approach based on Padé-Approximation has been implemented for solving Measles dynamical model. All the necessary initial conditions (boundedness, positivity, feasibility) properties have been modeled as problem constraints. The control equations are converted to the constraint function and then a penalty function approach has been used to resolve the optimization problem. The solution purposed by this scheme is unconditionally convergent to steady-state and meets all the model requirements. For these reasons, this entire paper has been drawn up. The nonlinear dynamic Measles model having comprehensive detail in Section 2. Section 3 is based on the Padé approach, the differential development, the penalty function, and the structure of the EPA

scheme for the solution of the nonlinear epidemiologic Measles model. Throughout section 4, the interpretation and simulations of the findings presented are the objects of attention. Finally, remarks and recommendations for future directions were discussed in the last segment.

2. Dynamical Measles Model

The considered model of Measles that was proposed by Allen et al in [27] described in Figure 1.

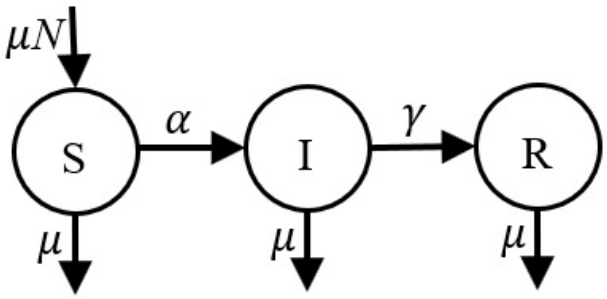

Figure 1: SIR model for Measles treatment

In proposed model population is divided into three following groups. The models' variables are defined at any time t as:
$S(t)$: Susceptible people never come into contact with Measles
$I(t)$: Infective people with Measles and can transmit the disease
$R(t)$: Recovered people from Measles
The model parameters are:
$N(t)$: Total population, μN : Recruitment rate into susceptible class, α : Transitivity, γ : Per-capita recovery rate, μ : Per-capita removal rate.

$$S'(t) = \mu N - \alpha S(t)I(t) - \mu S(t), \tag{2.1}$$

$$I'(t) = \alpha S(t)I(t) - (\gamma + \mu)I(t), \tag{2.2}$$

$$R'(t) = \gamma I(t) - \mu R(t), \tag{2.3}$$

The initial conditions for the model are follows

$$N(t) = S(t) + I(t) + R(t). \tag{2.4}$$

The Measles model may be rewritten as

$$S'(t) = X_1(t) = \mu N - \alpha S(t)I(t) - \mu S(t), \tag{2.5}$$

$$I'(t) = X_2(t) = \alpha S(t)I(t) - (\gamma + \mu)I(t), \tag{2.6}$$

$$R'(t) = X_3(t) = \gamma I(t) - \mu R(t), \tag{2.7}$$

The initial conditions are :

$$S_0 = S(0) = 990; I_0 = I(0) = 50; R_0 = R(0) = 10.$$

The basic reproduction number R_0 of the system (2.1-2.3)

$$R_0 = \frac{\alpha N}{\gamma \ + \mu}$$

Model (2.1-2.3) has a distinctive endemic equilibrium whenever $R_0 > 1(2.85)$, the model's endemic equilibrium is given by E_1=(S*,I*,R*)=(350,30.95,619.04)

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

$$I* = \frac{\alpha\mu N - \mu(\gamma \ + \mu)}{\alpha(\gamma \ + \mu)}$$

$$R* = \frac{\alpha\mu N - \mu(\gamma \ + \mu)}{\alpha\mu(\gamma \ + \mu)}$$

The disease-free equilibrium of the system (1) denoted by E_0=(N,0,0)

3. Evolutionary Pade Approximation Scheme

The design of this scheme is based on Pade-approximation [13], Differential Evolution [3, 4] and penalty function [27]. Evolutionary Pade Approximation scheme has been applied on a nonlinear epidemiology Measles model which involves the following steps.

3.1. Pade Approximation

At the end of the 19th century, in the classical theory of the continuing fraction, the concept of Pade approximation was started. The reasonable (N,M) order function of the approximation of Pade referred to in [13].

$$P_{N,M}(t) = \frac{\sum_{i=0}^{N} a_i t^i}{\sum_{j=0}^{M} a_j t^j}$$

Pade approximation are $\sum_{i=0}^{N} a_i t^i$ and $\sum_{j=0}^{M} a_j t^j$ and by putting $b_0 \neq 0$ the above expression becomes:

$$P_{N,M}(t) = \frac{\Sigma_{i=0}^{N} a_i t^i}{1 + \Sigma_{j=1}^{M} b_j t^j}$$

The above equation having (N+M+1) undetermined coefficients, by using the Maclaurine series $P_{N,M}(t)$ referred in [28] we get the value of coefficient. Suppose that S(t), I(t) and R(t) are Pade rational functions approximated as

$$S(t) = \frac{\Sigma_{i=0}^{N} a_i t^i}{1 + \Sigma_{j=1}^{M} b_j t^j}$$

$$I(t) = \frac{\Sigma_{i=0}^{N} c_i t^i}{1 + \Sigma_{j=1}^{M} d_j t^j}$$

$$R(t) = \frac{\Sigma_{i=0}^{N} e_i t^i}{1 + \Sigma_{j=1}^{M} f_j t^j}$$

By imposing initial conditions we obtain

$$a_0 = S_0, c_0 = I_0, e_0 = R_0. \tag{3.1}$$

The discrete-time steps are $t_q = t_0 + qh; q = 0, 1, 2, 3, ..., q_{\max}$, then the system (2.5-2.7) becomes:

$$\begin{cases} \varepsilon_1(t_q) = 0, \\ \varepsilon_2(t_q) = 0, \\ \varepsilon_3(t_q) = 0, \end{cases} \tag{3.2}$$

Here $\varepsilon_1, \varepsilon_2$ and ε_3 are the residuals defined by

$$\varepsilon_1(t_q) = (1 + \Sigma_{j=1}^{M} b_j t_q^j)(\Sigma_{i=0}^{N} i a_i t_q^{i-1}) - (\Sigma_{i=0}^{N} a_i t_q^i)(\Sigma_{j=1}^{M} j b_j t_q^{j-1}) - X_1(t_q)(1 + \Sigma_{j=1}^{M} b_j t_q^j). \tag{3.3}$$

$$\varepsilon_2(t_q) = (1 + \Sigma_{j=1}^{M} d_j t_q^j)(\Sigma_{i=0}^{N} i c_i t_q^{i-1}) - (\Sigma_{i=0}^{N} c_i t_q^i)(\Sigma_{j=1}^{M} j d_j t_q^{j-1}) - X_2(t_q)(1 + \Sigma_{j=1}^{M} d_j t_q^j). \tag{3.4}$$

$$\varepsilon_3(t_q) = (1 + \Sigma_{j=1}^{M} f_j t_q^j)(\Sigma_{i=0}^{N} i e_i t_q^{i-1}) - (\Sigma_{i=0}^{N} e_i t_q^i)(\Sigma_{j=1}^{M} j f_j t_q^{j-1}) - X_3(t_q)(1 + \Sigma_{j=1}^{M} f_j t_q^j). \tag{3.5}$$

The problem reduces the problem to find 3(M+N) coffieciently of approximators in Pade by solving system (3.2) with $3q_{\max}$ nonlinear simultaneous equations.

Problem Constraints

The model's constraints on equality are seen as stated in the system (3.1):

$$h_1(t) = S(t) - S_0 = 0. \tag{3.6}$$

$$h_2(t) = I(t) - I_0 = 0. \tag{3.7}$$

$$h_3(t) = R(t) - R_0 = 0. \tag{3.8}$$

The inequality constraints have to do with positivity

$$g_{1q} = \frac{\Sigma_{i=0}^{N} a_i t_q^i}{1 + \Sigma_{j=1}^{M} b_j t_q^j} \geq 0.$$

$$g_{2q} = \frac{\Sigma_{i=0}^{N} c_i t_q^i}{1 + \Sigma_{j=1}^{M} d_j t_q^j} \geq 0.$$

$$g_{3q} = \frac{\Sigma_{i=0}^{N} e_i t_q^i}{1 + \Sigma_{j=1}^{M} f_j t_q^j} \geq 0.$$

whereas represents the bounded-ness of the solution.

$$g_{1q} + g_{2q} + g_{3q} \leq N.$$

Objective Function

Suppose that

$$x = \begin{bmatrix} a_1, a_2, ..., a_m, b_1, b_2, ..., b_N, c_1, c_2, ..., c_M, \\ d_1, d_2, ..., d_m, e_1, e_2, ..., e_N, f_1, f_2, ..., f_M, \end{bmatrix}^t \in \mathbb{R}^{3(M+N)}.$$

After converting the above model the minimization becomes:

Minimize $\phi(x) = \frac{1}{3}\Sigma_{z=1}^{3}\Sigma_{q=0}^{q_{\max}}[\varepsilon_Z(t_q)]$.

Penalty Function

A large number of positive factors are added to the objective function in accordance with the degree of violation of the constraints.

$$\theta = \begin{cases} \psi(x) + \zeta(x) if x is infeasible, \\ \psi(x) if x is infeasible. \end{cases} \tag{3.9}$$

Here objective function $= \psi(x)$ and the penalty function $= \zeta(x)$ describes penalized function $= \phi(x)$. Here '$\zeta(x) \geq 0$ is for minimization and $\zeta(x) \leq 0$ for maximization, the unconstrained optimization model becomes:

$$\zeta(x) = \sum_{q=1}^{q_{\max}} P_q \times \max 0, (h_1)^2, (h_2)^2, (h_3)^2, -g_{1q}, -g_{2q}, -g_{3q}, \sum_{s=1}^{3} g_{sq} - N.$$

Here scalar P_q is a large positive real number then the unconstrained objective function is Minimize

$$\overline{\omega}(x) = \phi(x) + \zeta(x). \tag{3.10}$$

4. Optimization process with differential evolution

Price and Storn have created DE as a function optimizer that is easy to use, safe, and flexible. The first published paper on DE appeared as a technical document in 1995. Like nearly all evolutionary algorithms, DE is a population-based optimizer that attacks the starting point problem by sampling the objective function at various randomly chosen starting points? In this original population, the preset bound parameter describes the domain from which the vectors are chosen. Each vector is indexed between 0 and Np-1. DE produces fresh points that interfere with current points. Instead,DE disturbs vectors that have the scaled distinction with two randomly chosen population vectors. To generate the trial vector, μ_0 DE adds the scaled, random vector difference to a third randomly chosen population vector. In the selection phase, the trial vector competes against the same index population vector, which is number 0 in this case. The step of selecting and saving that marks the vector as a next-generation member with the reduced objective function value. The technique repeats until all vectors of the Np population compete against a randomly generated trial vector. After testing the last test vector, the Np survivors become siblings in the next generation's evolutionary process [3, 4]. To optimize objective function (3.10) using EPA scheme the following steps are involved:

Step 1. Generate population randomly, population of K solution $x_j \in R^{3(M+N)}; 1 \leq j \leq K$.

Step 2. Evaluate the value $\bar{\omega_j} = \bar{\omega}(X_j)$ of each of solution. Collect the best solution with the minimum value of objective function. Initially set T=0

Step 3. Set T=T+1

Step 4. Choose three distinct solutions $x_A, x_B and x_C$ from the population excluding x_j for each of j=1,2,3,4,.....,K, Set $y = x_j$

Step 5. For each of dimensions $i = 1, 2, 3, ...3(M + N)$, alter the i^{th} coordinate according to

$$y_i = \begin{cases} x_{Ai} + F \times (x_{Bi} - x_{Ci}) if rand_i[0, 1] < CR \\ x_{ji} otherwise \end{cases} \tag{4.1}$$

Step 6. If $\bar{\omega}(y) < \bar{\omega_j}$ then $x_j \leftarrow y$, otherwise discard y.

Step 7. Best solution must be updated.

Step 8. If T > number of iterations, then terminate, by maintaining the best solution, other wise repeat all the process from step 3.

5. Numerical Results

Set parameters of DE algorithm for numerical illustrations: $N = 50$; $F = 0.55; CR = 0.91$ and $maximum\ iterations = 2000$. The approximation order for Padé is set to $(N, M) = (2, 2)$.

The q_{max} parameter is set to 2000. The penalty factor for all q is set to $L_q = 10^{10}$. The optimized Measles model parameters are $\alpha = 0.003$, $\mu = 0.05$, $\gamma = 1$, $N = 1000$. In all simulations 10 independent runs have been taken and chosen the best one, Intel Core i3 with 4GB RAM computer was used for experimentation with Microsoft windows 10. The source code was executed by using $MATLAB$ (R2015b). The mathematical analysis of the non-linear epidemic Measles model was provided. Figures 2-4 demonstrate the sound effects of the EPA algorithms on the population which is sensitive, infected, and recovered compared to $NSFD$ which is special and healthy. Figures show a convergence solution in relation to disease-free equilibrium among different population groups, with the aid of EPA algorithm and $NSFD$, it can be easily observed that the EPA algorithm results are more reliability and better than the numerical scheme. Figure 5 illustrates the rapidly decreasing effect of vaccination on infected and chronic populations. The resilient and recovered population growth also shows that the effect of vaccines can easily be managed after calculating the disease.

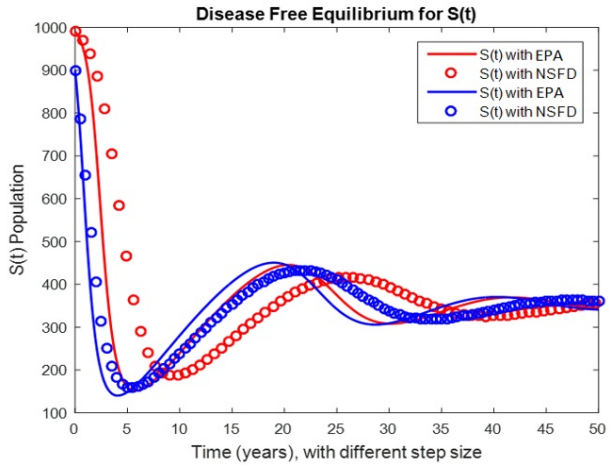

Figure 2: Dynamical behavior for susceptible population S(t) in a time t with EPA and NSFD

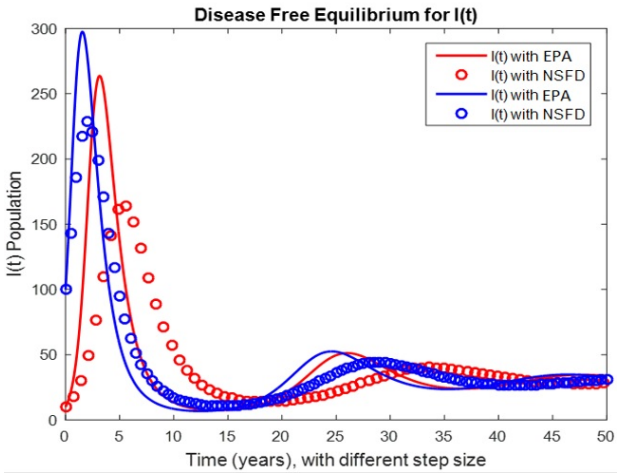

Figure 3: Dynamical behavior for infected population I(t) in a time t with EPA and NSFD

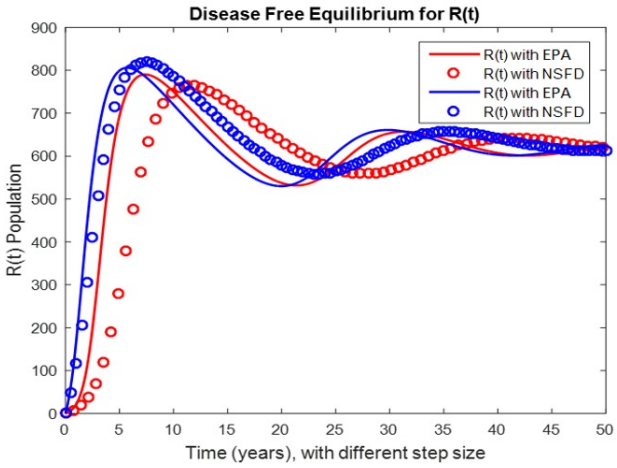

Figure 4: Dynamical behavior for recovered population R(t) in a time t with EPA and NSFD

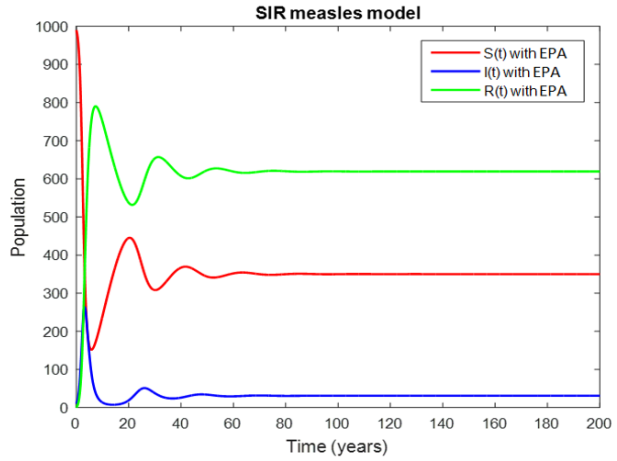

Figure 5: Dynamical behavior of S(t), I(t) and R(t) in a time t with EPA

6. Conclusion

In this study, to get the numerical solutions of the non-linear epidemic Measles model, the evolutionary Padé approach was implemented. EPA has given strong state variables approximations, which fulfill the high accuracy of the governing equations. The solution obtained is very quickly convergent and superior then NSFD, as regards the relationship between different population compartments to disease-free equilibrium. We also implemented the numerical simulation and tested all analytical findings by using EPA to reduce contaminated disease-free equilibrium levels very quickly. The Padé approximation of order (2, 2) was used in the current work, it is worth mentioning. By using the higher-order and robust optimization strategy, the accuracy of the numerical solution can be improved. Finally, this technique removing the need to supply a step size and we can control the spreading of Measles in the community.

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