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Review Article

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Dynamical Transmission and Mathematical Analysis of SIR Measles Epidemiological Model

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Abstract

In this work, we presented a nonlinear time fractional model of measles in order to understand the outbreaks of this epidemic disease. The stability analysis and sensitivity analysis of the model is provided and the certain threshold value of the basic reproduction number $R_0>1$, disease free and endemic equilibrium point of the model is also calculated. We develop unconditionally convergent nonstandard finite difference scheme by applying Mickens approach $Ø(h)=h+O(h^2)$. This method proved to be very efficient technique for solving epidemic models to control the infection diseases. We also discussed the qualitative behavior of the model and numerical simulations are carried out to support the analytic results.

Keywords: Qualitative Analysis; Stability Analysis; Nonstandard Finite Difference Scheme

Introduction

In human population epidemiology studies play an important role to understand the disease in human population. Often the work of mathematical epidemiology consists of model building, estimation of parameters and investigation of the sensitivity of models to change in the parameters and numerical simulations. Epidemiologists use mathematical models to understand previous outbreaks of diseases and to better understand the dynamics of how infections spread through populations [1]. The research of this kind helps to understand the ratio of disease spread in the population and to control their parameters [2, 3]. These types of diseased models are often called infectious diseases (i.e. the disease which transferred from one person to another person). Measles, rubella, chicken pox, mumps, aids and gonorrhea syphilis are the examples of infectious disease [4, 5]. Rubella virus is highly infectious illness which is also known as morbilli or measles. The virus can be found in the mucus of the throat, nose of an infected adult and child. Measles symptoms caused by Rubeola virus always included fever, coryza (runny nose), conjunctivitis and at least one of the three Cs-coughs. Symptoms appear after the initial infection about 9-11 days [6]. Complications of measles are fairly common but the patients have weak immune system are more likely to be worse such as those with HIV/AIDS or leukemia and those with vitamin deficiency. Healthy children over the age of 5 are less likely to have complications than adults over the age of 20. It is the first and worst eruptive fever occurs during childhood [7, 8].

The NSFD schemes preserve main properties of the differential counterparts, such as positivity, monotonicity, periodicity, stability, and some other invariant including energy and geometrical shapes. It should be emphasized that NSFD schemes can preserve all properties of continuous models for any discretization parameters. The discrete models with these properties are called dynamically consistent [9]. For the last two decades NSFD methods have attracted attention for many researchers and achieved significant results [10]. The property of stability of the set of equilibria of differential equation is one of these results because it plays the essential role in the study of asymptotical behavior of the solutions of the differential equations [11-13]. The construction of differences schemes, which preserve the stability of the equilibrium points, is important in numerical simulation of differential equations. There are many works concerning the elementary stable schemes. The typical results are for general dynamical systems and for other specific systems [14, 15].

Mathematical Model

W.O Kermack and A.G.MecKendric are those persons who are inventor of the diseases models, and they played an important role in Mathematical epidemiology. In perposed model population is divided into three groups which denoted by S (susceptible), I (infected) and R (recovered). The class S of susceptible is increased by birth at a rate πN where both rate π and death rate μ are same, so susceptible is increased by μN. It is decreased by infection following contacts with infected individuals I at rate α. This class is decreased by recovery from infection at a rate γ and dimensioned by natural death rate μ. This generate a class R of individuals who have complete protection against disease [16]. The flow chart of the model is representing in (figure 1).

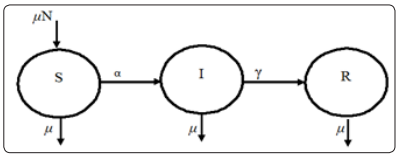

Figure 1: flow chart of model

Following are the equations of the system:

$$\frac{dS}{dt} = \mu N - \alpha SI - \mu S \quad (1)$$

$$\frac{dI}{dt} = \alpha SI - (\gamma + \mu)I \quad (2)$$

$$\frac{dR}{dt} = \gamma I - \mu R \quad (3)$$

Where μ ,α and γ represents the per capita removal rate, transitivity rate ant per capita recovery rate respectively.
where N=S+I+R
With initial conditions
$S(0)$=990, $I(0)$ = 10, $R(0)$=0

Qualitative Analysis of the Model

For equilibrium we have,

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

This state that the population is disease free that is infection dies out of the population and is such that
0=μN-αSI-μS
0=αSI-(γ+μ)I
0=γI-μR

By simplifying the above equations we get disease free equilibrium denoted by E_0 i.e E_0= (N,0,0) and the endemic equilibrium point denoted by E_1 i.e

$$E_1 = \left(\frac{(\gamma+\mu)}{\alpha}, \frac{\alpha\mu N - \mu(\gamma+\mu)}{\alpha(\gamma+\mu)}, \frac{\alpha\gamma\mu N - \mu\gamma(\gamma+\mu)}{\alpha\mu(\gamma+\mu)}\right)$$

Reproductive number

Consider the jacobian matrix J.

$$J = \begin{bmatrix} -(\alpha I + \mu) & -\alpha S & 0 \\ \alpha I & \alpha S - (\gamma + \mu) & 0 \\ 0 & \gamma & -\mu \end{bmatrix}$$

Since the jacobian matrix is $J = F - V$ where

$$F = \begin{bmatrix} -(\alpha I + \mu) & 0 & 0 \\ \alpha I & \alpha S & 0 \\ 0 & \gamma & -\mu \end{bmatrix} \text{ And } V = \begin{bmatrix} -(\alpha I + \mu) & -\alpha S & 0 \\ \alpha I & \alpha S - (\gamma + \mu) & 0 \\ 0 & \gamma & -\mu \end{bmatrix}$$

We know that $K = FV^{-1}$ and using the relations $|K-\lambda I| = 0$ for the Eigen values, which represents reproductive number R_0 i.e

$$R_0 = \frac{\alpha N}{(\gamma + \mu)} > 1$$

Sensitivity analysis of R_0

The sensitivity of R_0 is as follows

$$\frac{\partial R_0}{\partial \alpha} = \frac{N}{(\gamma + \mu)} > 0$$

$$\frac{\partial R_0}{\partial \mu} = \frac{-N\alpha}{(\gamma + \mu)^2} < 0$$

$$\frac{\partial R_0}{\partial \gamma} = \frac{-N\alpha}{(\gamma + \mu)^2} < 0$$

$$\frac{\partial R_0}{\partial N} = \frac{\alpha}{(\gamma + \mu)} > 0$$

It can be seen that R_0 is most sensitive to change of its parameter, here R_0 is increasing with α, N and decreasing with μ, γ. In other words it found that the sensitivity analysis shows that prevention is better than to control the disease.

Stability at the Endemic Equilibrium

To examine the local stability of endemic equilibrium E_1 we evaluate the jocabian matrix at

$$E_1 = \left(\frac{(\gamma+\mu)}{\alpha}, \frac{\alpha\mu N - \mu(\gamma+\mu)}{\alpha(\gamma+\mu)}, \frac{\alpha\gamma\mu N - \mu\gamma(\gamma+\mu)}{\alpha\mu(\gamma+\mu)}\right)$$

The Matrix which is called Jacobian is of the form:

$$J = \begin{bmatrix} -(\alpha I + \mu) & -\alpha S & 0 \\ \alpha I & \alpha S - (\gamma + \mu) & 0 \\ 0 & \gamma & -\mu \end{bmatrix}$$

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

Theorem 1:

The endemic equilibrium E_1 is locally asymptotically stable for R_0>1 otherwise unstable.
Proof:

E_1 is locally asymptotically stable if $Re\ (\lambda)$<0 where λ can be calculated from the relation $|J-\lambda I| = 0$ i.e

$$\begin{vmatrix} -(\alpha I + \mu + \lambda) & -\alpha S & 0 \\ \alpha I & \alpha S - (\gamma + \mu) - \lambda & 0 \\ 0 & \gamma & -(\mu + \lambda) \end{vmatrix} = 0$$

$$i.e\ (\alpha I + \mu + \lambda)[\alpha S - (\gamma + \mu + \lambda)](\mu + \lambda) + \alpha^2 S[I(\mu + \lambda)] = 0$$

The equation which is given above is called characteristic equation. By substituting the values of endemic equilibrium E_1 we get values of λ as follows

$$\left.\begin{aligned} \lambda_1 &= -\mu \\ \lambda_2 &= \frac{-\mu}{2} + \frac{\sqrt{\mu(4\gamma + 9\mu - 4\beta N)}}{2} \\ \lambda_3 &= \frac{-\mu}{2} - \frac{\sqrt{\mu(4\gamma + 9\mu - 4\beta N)}}{2} \end{aligned}\right\}$$

Real parts are $\lambda_1 = -\mu, \lambda_2 = \frac{-\mu}{2}$, $\lambda_3 = \frac{-\mu}{2}$, if $\lambda_i < 0$.Therefore, since all the Eigen values are negative. So E_1 is locally asymptotically stable. This proves the proposition.

Nonstandard finite difference (NSFD) scheme

Nonstandard difference (NSFD) methods for the numerical integration of differential equations had their genesis in a paper published in 1989 [17-20]. The basic rules to construct such schemes and their application to specific nonlinear equations appear in a variety of publications [9, 14]. In recent years, NSFD discrete models have been constructed and or tested for a wide range on nonlinear dynamical systems [21]. In this section, we design the NSFD scheme that replicates the dynamics of continuous model (1)-(3). Let $Y_k=(S_k, I_k, R_k)^t$ denoted the approximation of $X(t_k)$ where $t_k=k\Delta t$, with $k\epsilon N$, $h=\Delta t>0$ be a step size then

$$\frac{S^{k+1}-S^k}{\emptyset} = \mu N - \alpha S^{k+1} I^k - \mu S^{k+1} \quad (4)$$

$$\frac{I^{k+1}-I^k}{\emptyset} = \alpha S^{k+1} I^k - (\gamma+\mu) I^{k+1} \quad (5)$$

$$\frac{R^{k+1}-R^k}{\emptyset} = \gamma I^{k+1} - \mu R^{k+1} \quad (6)$$

Which is the purposed NSFD scheme for the given model, where

$$\emptyset = \emptyset(h) = \frac{1-e^{-(\gamma+\mu)h}}{(\gamma+\mu)}$$

The discrete method (4-6) is indeed an NSFD scheme because it is constructed according to Mickens rules [17] formalized as follows in [16].

Rule 1

The standard denominator $h = \emptyset(t)$ of the discrete derivatives is replaced by the complex denominator function in Equation (35) which satisfies the asymptotic relation

$$\emptyset(h) = h + 0\,(h^2)$$

Note that the denominator function $\emptyset$ is expected to better capture the dynamics of the continuous model through the presence of the underlying parameters γ,μ. In fact, exact schemes for a wide range of dynamical systems involve such complex denominator functions [17-19].

Rule 2

Non-linear terms of the right hand side of equation (1)-(3) are approximated in a non-local way. For instance, we have

$$I(t_k)S(t_k) \cong I_k S_{k+1} \text{ instead of } I(t_k)S(t_k) \cong I_k S_k.$$

Analysis of the Scheme

Theorem 2: The NSFD scheme (4)-(18) is a dynamical system on the biological feasible domain κ of the continuous model (1)-(3).

Proof: First we prove the positivity of the scheme (16)-(18). It is to show that the NSFD scheme (16)-(18) takes the explicit form3

$$S^{k+1} = \frac{\mu N\emptyset + S^k}{1+\alpha\emptyset I^k + \mu\emptyset} \quad (7)$$

$$I^{k+1} = \frac{\alpha\emptyset(S^k + N\mu\emptyset)I^k + I^k(1+\emptyset\alpha I^k + \emptyset\mu)}{(1+\emptyset(\gamma+\mu))(1+\alpha\emptyset I^k + \mu\emptyset)} \quad (8)$$

$$R^{k+1} = \frac{\gamma\emptyset\{\alpha\emptyset(S^k+N\mu\emptyset)I^k+I^k(1+\emptyset\alpha I^k+\emptyset\mu)\}+R^k(1+\emptyset(\gamma+\mu))(1+\alpha\emptyset I^k+\mu\emptyset)}{(1+\mu\emptyset)(1+\emptyset(\gamma+\mu))(1+\alpha\emptyset I^k+\mu\emptyset)} \quad (9)$$

Thus $S^{k+1}\geq 0$, $I^{k+1}\geq 0$, $R^{k+1}\geq 0$, whenever the discrete variables are non-negative at the previous iteration. It remains to prove the positive invariance of κ.

Adding equation 4 & 5 we get

$$H^{k+1}(1+\emptyset\mu) = \emptyset\mu N + H^k - (\emptyset(\gamma+\mu)+1)I^{k+1} \leq \emptyset\mu N + H^k$$

$$H^{k+1}(1+\emptyset\mu) \leq \emptyset\mu N + H^k$$

Therefore

$$H^{k+1} \leq N, \text{ whenever } H^k \leq N$$

The priori bounds for R^{k+1} follow readily from the fact that R^{k+1} and I^{k+1} are less than or equal to H^{k+1}. This complete the proof.

Numerical Simulations

The mathematical analysis of epidemic measles SIR model with non-linear incidence has been presented. To observe the effects of the parameters using in this dynamics measles SIR model (1)-(3), conclude several numerical simulations with parameters values α=0.003, μ=0.05, γ=1 ,N=1000 are given in [16]. Hence R_0=2.85 and unique equilibrium point E_1=(350 ,30.95,619.04) is asymptotically stable, this result enhance Theorem 1. Figure 1-2 shows the convergence solution using NSFD scheme at h=10 and h=1 for true equilibrium point. It can be easily seen that by labeled points in figure 1-2, which shows fast convergence by decreasing the value ofh. Figure 1-2 shows that the model presented gradually approaches the steady states for different values of h. In figure 1-2 clearly shows that infected individuals at latent period are diagnosed and treated, the number of susceptible individuals decreases significantly, the infected decreases steadily while the recovered increased steadily.

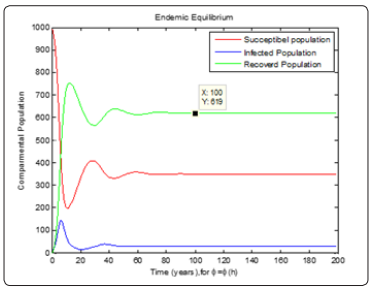

Figure 2: Numerical solutions for susceptible, infected individual, recovered population in a time t for $\emptyset = \emptyset(h)$, where h=10

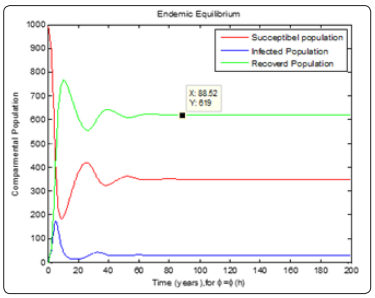

Figure 3: Numerical solutions for susceptible, infected individual, recovered population in a time t for $\emptyset = \emptyset$(h). h = 1

Conclusion

This paper deals with the mathematical model of measles disease to investigate the occurrence of diseases in population. The analysis of the system is well established. Qualitative analysis and stability analysis of the model according to equilibrium point are discussed and where it is locally asymptotically stable forR_0>1. The nonstandard finite difference scheme is dynamically consistent,

easy to implement and show a good agreement to control the spread of measles disease for long period of time. Finally we presented the numerical simulation and verified all the analytical results numerically by using nonstandard finite difference scheme to reduce infected rate for endemic equilibrium. While this conclusion may have practical implications for the control of measles infections, more realistic models that are specific for measles infection.

References

1. S Abubakar, NI Akinwande, Abdulrahman (2012) A Mathematical Model of Yellow Fever Epidemics. Afrika Mathematika 6: 56-58.
2. BT Grenfell (1992) Chance and chaos in measles dynamics. J R Stat Soc B 54: 383-398.
3. PL Panum (1940) Observations Model during the epidemic of Measles on the Faroe Islands in the year 1846. JAMA 115: 1747.
4. Geneva: World Health Organization (2010) WHO vaccine-preventable diseases: monitoring system.
5. Roberts MG, Tobias MI (2000) Predicting and Preventing Measles Epidemic in New Zealand: Application of Mathematical Model. Epidemiol Infect 124: 279-287.
6. JM Ochoche, RI Gweryina (2014) A Mathematical Model of Measles with Vaccination and Two Phases of Infectiousness. IOSR Journal of Mathematics 10: 95-105.
7. HW Hethcote (2000) The Mathematics of Infectious diseases, Society for Industrial and applied Mathematics. SIAM 42: 599-653.
8. JD Murray (2003) Mathematical Biology I. Introduction, 3rd Edition 315.
9. R Anguelov, Y Dumont, JM-S Lubuma, M Shillor (2014) dynamically consistent nonstandard finite differences schemes for epidemiological models. Journal of Computational and Applied Mathematics 225: 161-182.
10. R Anguelov, JMS Lubuma (2003) nonstandard finite difference method by nonlocal approximations, Mathematics and Computers in Simulation 61: 465-475.
11. Ronald E (2003) Mickens on the occasion of his 60th Birthday; Guest Editor, A. B. Gumel, Journal of Difference Equations and Applications 2003: 1059-1112.
12. TD Dimitrov, HV Kojouharov (2006) Positive and elementary stable nonstandard numerical methods with applications to predator –prey models. Journal of Computational and Applied Mathematics 189: 98-108.
13. TD Dimitrov, HV Kojouharov (2008) nonstandard finite-difference methods for predator-prey models with general functional response. Mathematics and Computers in Simulations 78: 1-11.
14. TD Dimitrov, HV Kojouharov (2007) Stability-preserving Finite-Difference Methods for General Multi- Dimensional Autonomous Dynamical Systems. International Journal of Numerical Analysis and Modelling 4: 280-290.
15. Mickens RE (1994) nonstandard finite difference Models of differential equations, World scientific Singapore.
16. FS Akinboro, S Alao, FO Akinpelu (2014) Numerical Solution of SIR Model using Differential Transformation Method and Variational Iteration Method Gen. Math Notes 22: 82-92.
17. R Anguelov, JMS Lubuma (2001) Contribution to the mathematics of the nonstandard finite difference method and applications. Numer Methods Partial Differ Equ 17: 518-543.
18. J MS Lubuma, K Patidar (2007) Non-standard methods for singularity perturbed problems possessing oscillatory/ layer solutions. APP Math Comput 187: 1147-1160.
19. Abba B Gumel (2014) Mathematics of continuous and discrete Dynamics Systems. Contemp Math 618: 147-161.
20. RE Mickens (1989) Exact solutions to a finite difference model of a nonlinear reactions –advection equation: Implications for numerical analysis, Numerical Methods for partial Differential Equations 5: 313-325.
21. RE Mickens (2000) Applications of Nonstandard finite difference Schemes, World Scientific.