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Nonstandard finite difference scheme for control of measles epidemiology

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ABSTRACT

This paper is based on the analysis of SEIR measles models, which are used to study the integrating vaccination as a control strategy and taking the two stages of infectiousness and transmission dynamics of infectious diseases in a population. Measles is a higher contagious that can spread in a community population depending on the number of people susceptible or infected and also depending on their movement in a community. We construct an unconditionally convergent nonstandard finite difference (NSFD) scheme for SEIR measles model. NSFD preserve the positivity of all values of h. This method proved to be a very efficient technique for solving epidemic models. We obtained disease-free equilibrium (DFE) point, Endemic equilibrium (EE), reproduction number for the model. Moreover, the analysis of the epidemic models using nonstandard finite difference scheme reveals that the method provides a rapidly convergent series solution by little iteration and avoids the massive computational work. Numerical simulations show that the rate of infection is decreased with the passage of time and disease will die out in the community. The results are compared to the Differential Transformation Method to show this scheme is efficient and better accuracy for epidemic models.

1. Introduction

In human population epidemiology studies play an important role to understand the disease in human population. The research of this kind help to understand the ratio of disease spread in the population and to control their parameters (Grenfell, 1992). 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. 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-cough. Symptoms appear after the initial infection about 9-11 days. Virus grows in lymphatic system and lungs after entering the body and. Blood vessels, central nervous system, urinary tract, imitates in the eyes are chance to be infected. According to the experts, the virus created itself within 1-3 weeks. Measles reinfection are very rare. Measles is an air borne disease so the ratio of 90 percent peoples who haven't got the immunity against measles cause to spread this disease from one person to another person if they live together at the same house (Ochoche and Gweryina, 2014). 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 (Hethcote, 2000; Murray, 2003). It produces infection of eyes, bronchitis, vomiting, bronchitis is inflammation of the inner walls of airways and laryngitis is inflammation of the voice box.

In recent study the differential transform method is the one of the most effective and efficient numerical techniques for solving system of linear and non-linear differential equations, integral and Integro differential equations. Till now this method solve various type of linear, non-linear differential equations as well as integral equations (Pukhov, 1978). DTM provides an efficient numerical and explicit solution with least calculations and high

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accuracy. DTM is very effective technique because it gives well approximately solution in a very small region (Pukhov, 1981; 1982; 1986).

Now we discuss nonstandard finite difference scheme (NSFD) which works correctly for large time step size. In recent years, construction of NSFD discrete models has been tested for a wide range on nonlinear dynamical systems (Moaddy et al., 2011; Dimitrov and Kojouharov, 2005; Taylor et al., 1986).

The NSFD have been widely used for system of differential equations that are describing problems in mathematical biology and the other different areas. These Method showed the superiority in preserving the passivity (Jang, 2005; Gumel et al., 2005; Mounim and de Dormale, 2004; Mickens, 2003) when comparing all others well known numerical method of state variable of the system under study.

In this paper, we design and analyze a NSFD for mathematical model of the transmission of measles in the society. The dynamics of this model are studied using the qualitative theory of dynamical system. This method also preserves positivity of the solution which is one of the essential requirement when modeling epidemic disease. Comparison also made with other conventional approaches that are routinely used for such problems.

2. Material and method

To describe the transmission dynamics of measles we formulate a deterministic, compartmental, mathematical model. In order to describe the model equations, the total population (N) is divided into four classes: S, E, I, R represented as Susceptible, Exposed, infected and recovered population respectively. We divided our compartmental model into four compartment as shown in flow chart given in Fig. 1. The susceptible population which are not infected and are able to catch the disease and able to transmit others is in S class with infection contact rate βand increased by birth or immigration at a rate B with mortality rate μ (i.e., natural death rate). The compartment E shows the class of those peoples which are exposed but not able to transfer the disease to another individuals in the period of incubation. The class E is break through into infected class at a rate α, decreased by testing and measles therapy at a rate σ and moderated at a rate μ which is natural death rate. The compartment I which is class of infected peoples is decreased at the infection rate γ and decreased at the mortality rate μ. In this model we assumes that both recovered and infected peoples and recovered , exposed peoples have got the permanently immune from the disease where R is the compartment of recovered peoples reduced with mortality rate μ (Momoh et al., 2013).

Therefore, the flow chart of deterministic measles model is shown in Fig. 1.

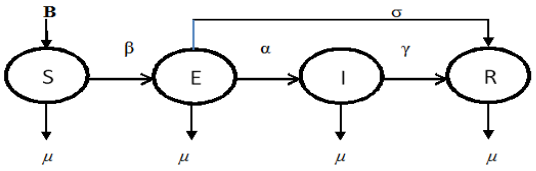


Fig. 1: The flow chart Of SEIR model

Following are the equations of the model:

$$\frac{dS}{dt} = B - \beta SI - \mu S \qquad t > 0 \qquad (1)$$
$$\frac{dE}{dt} = \beta SI - \mu E - \alpha E - \sigma E \qquad t > 0 \qquad (2)$$
$$\frac{dI}{dt} = \alpha E - \mu I - \gamma I \qquad t > 0 \qquad (3)$$
$$\frac{dR}{dt} = \gamma I + \alpha E - \mu R \qquad t > 0 \qquad (4)$$

We will assume the total population is constant for size N $i.e.,\ S + E + I + R = N$.

The system is qualitatively analyzed by two ways i.e. disease Free Equilibrium and endemic Equilibrium.

3. Qualitative analysis

3.1. Disease free equilibrium

Firstly we normalize the model by dividing by N for Eqs. 1-4.

$$s = \frac{S}{N}, e = \frac{E}{N}, i = \frac{I}{N}, r = \frac{R}{N}$$
$$f_1 = B - \beta si - \mu s \qquad (5)$$
$$f_2 = \beta si - \mu e - \alpha e - \sigma e \qquad (6)$$
$$f_3 = \alpha e - \mu i - \gamma i \qquad (7)$$
$$f_4 = \gamma i + \alpha e - \mu r \qquad (8)$$

When naturally, the disease die out then the solution of the above system asymptotically approaches a disease free population or equilibrium is of the form

$$S = \frac{\beta}{\mu}, E = 0, I = 0$$
$$i.e\ (S, E, I, R) = \left(\frac{\beta}{\mu}, 0, 0, 0\right)$$

3.2. Endemic equilibrium

It means that disease never dies out. When disease free equilibrium is unstable that is disease persist in the population then Endemic equilibrium takes the form:

$$(S^*, E^*, I^*, R^*) = \left[\frac{(\mu+\sigma+\alpha)(\mu+\gamma)}{\beta\alpha}, \frac{B}{\mu+\alpha+\sigma} - \frac{\mu(\mu+\gamma)}{\alpha}, \frac{B\alpha}{(\mu+\sigma+\alpha)(\mu+\gamma)} - \mu, \frac{1}{\mu}\left\{\gamma - \frac{\sigma}{\alpha}(\mu+\gamma)\right\}\left\{\frac{B\alpha}{(\mu+\alpha+\sigma)(\mu+\gamma)} - \mu\right\}\right]$$

3.3. Reproductive number

The reproductive number is:

$$R_0 = \frac{B}{\mu S} = \frac{B\beta\alpha}{\mu(\mu+\sigma+\alpha)(\mu+\gamma)} = 0.00087 < 1$$

So this is disease free equilibrium.

3.4. Stability analysis

We evaluate the Jacobian matrix to examine the local stability of the disease free equilibrium.

$$(s, e, i, r) = \left(\frac{\beta}{\mu}, 0,0,0\right)$$

Theorem 1: The given system of non-linear differential equation is locally asymptotically stable at disease-free equilibrium if this satisfies the following condition $R_0 < 1$ while E_0 is unstable saddle point if $R_0 > 1$.

Theorem 2: If $R_0 \leq 1$, then the model (1)-(4) is globally asymptotically stable at disease-free equilibrium, $E_0 = (S_0, 0,0,0)$ and unstable otherwise.

Theorem 3: The endemic equilibrium state E1 = (S_; E_; I_; R_) of the model (1)-(4) is globally asymptotically stable, if$R_0 > 1$, otherwise unstable. Proof of these theorems will be given in.

3.5. Differential transform method

This method is transformation technique based on Taylor series expansion (Pukhov, 1978; 1981; 1982; 1986) offers a convenient means for obtaining analytic solutions of differential equations. Zhou was the first man who introduced this technique in 1986 (Zhou, 1986), and is commonly used for the solution of electric circuit problems, it seems to be largely unknown to the research community. After that Pukhov (1986) worked in this technique and further developed these techniques for partial differential equations. This is a semi analytic technique and the solution of this technique is in the form of series also this method give closed form solution unlike other numerical methods.

Definition 1: A differential Transformation $U(k)$of a function $u(x)$ is in the form (Chen and Sy-Hong, 1996; Chen and Wu, 1996; Jang and Chen, 1997; Chen and Ho, 1996).

$$U(k) = \frac{1}{k!}\left[\frac{d^k u(x)}{dx^k}\right]_{x=x_0} \quad (9)$$

here, $u(x) =$ Original function and $U(k)$ =Transformed function. The Inverse of differential Transformation is defined as:

$$u(x) = \sum_{k=0}^{\infty} U(k)(x - x_0)^k \quad (10)$$

when x_0 is taken as zero, then the above original function $u(x)$ will be defined in the form of finite series and above function can be expressed in the form as:

$$u(x) = \sum_{k=0}^{\infty} U(k)\, x^k \quad (11)$$

$$u(x) = \sum_{k=0}^{\infty} U(k)\frac{x^k}{k!}\left[\frac{d^k u(x)}{dx^k}\right]_{x=x_0}. \quad (12)$$

Using the fundamental result of DTM we obtained the following equation. From the above equation we can see that the basic idea of DTM is based on Taylor series.

$$S(k+1) = \frac{1}{(k+1)}\left[B\delta(k) - \beta\sum_{l=0}^{k} S(l)I(k-l) - \mu S(k)\right] \quad (13)$$

$$E(k+1) = \frac{1}{(k+1)}\left[\beta\sum_{l=0}^{k} S(l)I(k-l) - \mu E(k) - \sigma E(k) - \alpha E(k)\right] \quad (14)$$

$$I(k+1) = \frac{1}{(k+1)}\left[\alpha E(k) - \mu I(k) - \gamma I(k)\right] \quad (15)$$

$$R(k+1) = \frac{1}{(k+1)}\left[\gamma I(k) + \sigma E(k) - \mu R(k)\right] \quad (16)$$

The inverse differential transform of S(k) is defined as:

$$s(t) = \sum_{k=0}^{\infty} S(k)(t - t_0)^k$$

when $t_0$is taken as zero, the given function y(x) is declared by a finite series and above equation can be written in the form

$$s(t) = \sum_{k=0}^{\infty} S(k)(t)^k.$$

By solving the above equation for $S(k+1)$, $E(k+1)$, $I(k+1)$ and $R(k+1)$ up to order 5, we get the function of $S(k)$, $E(k)$, $I(k)$ and $R(k)$ respectively

$$s(t) = \sum_{k=0}^{5} S(k)(t)^k \quad (17)$$

$$e(t) = \sum_{k=0}^{4} E(k)(t)^k \quad (18)$$

$$i(t) = \sum_{k=0}^{5} I(k)(t)^k \quad (19)$$

$$r(t) = \sum_{k=0}^{5} R(k)(t)^k \quad (20)$$

3.6. Nonstandard finite difference scheme (NSFD) for SEIR model

In this section, we design the NSFD scheme that replicates the dynamics of continuous model (1)-(4). Let $Y_k = (S_k\,,\ E_k\,,\ I_k\,,\ R_k)^t$ denoted the approximation $X(t_k)$ where $t_k = k\Delta(t)$ with $k \in N$, $h = \Delta(t) > 0$ be a step size then

$$\frac{S^{k+1} - S^k}{\emptyset} = B - \beta S^{k+1} I^k - \mu S^{k+1} \quad (21)$$

$$\frac{E^{k+1} - E^k}{\emptyset} = \beta S^{k+1} I^k - (\mu + \alpha + \sigma)E^{k+1} \quad (22)$$

$$\frac{I^{k+1} - I^k}{\emptyset} = \alpha E^{k+1} - (\mu + \sigma)I^{k+1} \quad (23)$$

$$\frac{R^{k+1} - R^k}{\emptyset} = \gamma I^{k+1} + \sigma E^{k+1} - \mu R^{k+1} \quad (24)$$

$$S^{k+1} = \frac{B\emptyset + S^k}{1 + \beta\emptyset I^k + \mu\emptyset}$$

$$E^{k+1} = \frac{\beta\emptyset S^{k+1} + E^k}{1 + \emptyset(\mu + \alpha + \sigma)}$$

$$I^{k+1} = \frac{\alpha\emptyset E^{k+1} + I^k}{1 + \emptyset(\mu + \sigma)}$$

$$R^{k+1} = \frac{\emptyset\gamma I^{k+1} + \sigma h E^{k+1} + R^k}{1 + \mu\emptyset}$$

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 (13 -16) is constructed by using Mickens rules (Akinboro et al., 2014) which was formalized by Anguelov and Lubuma (2001).

3.7. Analysis of the scheme

Theorem 4: The NSFD scheme (5 - 8) is a dynamical system on the biological feasible domain of the continuous model (13 - 16).

Proof: First we prove the positivity (13-16). For this we take the explicit form.

$$S^{k+1} = \frac{B\emptyset + S^k}{1+\beta\emptyset I^k + \mu\emptyset} \quad (25)$$

$$E^{k+1} = \frac{\beta\emptyset(B\emptyset + S^k) + E^k(1+\emptyset(\mu+\alpha+\sigma))(1+\beta\emptyset I^k+\mu\emptyset)}{(1+\emptyset(\mu+\alpha+\sigma))(1+\beta\emptyset I^k+\mu\emptyset)} \quad (26)$$

$$I^{k+1} = \frac{\alpha\emptyset\{\beta\emptyset(B\emptyset+S^k)+E^k(1+\emptyset(\mu+\alpha+\sigma))(1+\beta\emptyset I^k+\mu\emptyset)\}+I^k(1+\emptyset(\mu+\sigma))(1+\emptyset(\mu+\alpha+\sigma))(1+\beta\emptyset I^k+\mu\emptyset)}{(1+\emptyset(\mu+\sigma))(1+\emptyset(\mu+\alpha+\sigma))(1+\beta\emptyset I^k+\mu\emptyset)} \quad (27)$$

Adding Eqs. 21-22 we get

$H^{k+1}(1+\emptyset\mu) = \emptyset B + H^k - (\emptyset(\mu+\alpha+\sigma)+1)E^{k+1} \leq \emptyset B + H^k$

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

Therefore

$H^{k+1} \leq \frac{B}{\mu}$, $whenever \quad H^k \leq \frac{B}{\mu}$

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

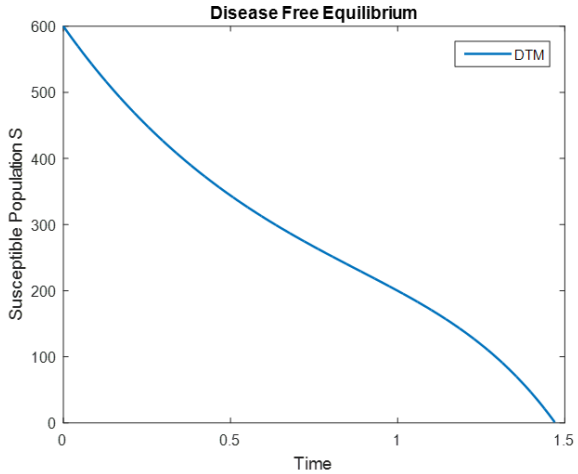
4. Results and discussion

Case 1: For numerical results, we used the followings Table 1 values of parameters are considered from Momoh et al. (2013).

Table 1: Parameters values of the model for case 1

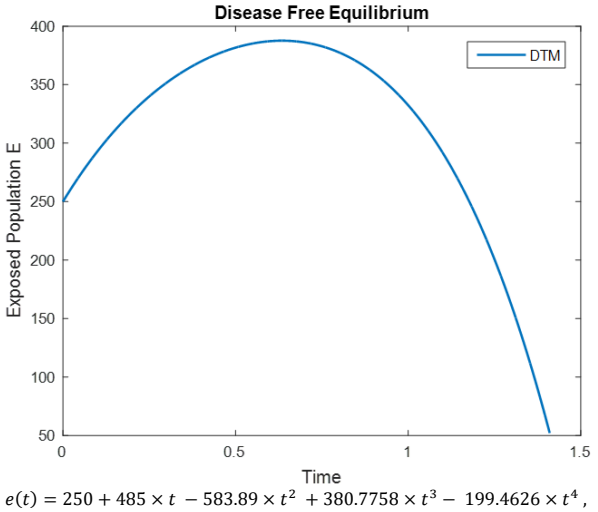
Parameters	Values	Parameters	Values
β	0.01	B	0.32
μ	0.2	α	0.01
σ	0.25	$S(0)$	600
γ	0.2	$E(0)$	250
$I(0)$	100	$R(0)$	50

The tabular form and graphs are shown in Figs. 2-4.



$s(t) = 600 - 719.68t + 544.308 \times t^2 - 327.5332 \times t^3 + 174.04995t^4 - 71.3383 \times t^5$, t=1.5

Fig. 2: Susceptible peoples; $s(t)$, $B = 0.32$, $\mu = 0.2$, $\beta = 0.01, \gamma = 0.2, \alpha = 0.01, \sigma = 0.25$



$e(t) = 250 + 485 \times t - 583.89 \times t^2 + 380.7758 \times t^3 - 199.4626 \times t^4$, t=1.5

Fig. 3: Exposed peoples; $e(t)$, $B = 0.32$, $\mu = 0.2, \beta = 0.01, \gamma = 0.2, \alpha = 0.01, \sigma = 0.25$

$i(t) = 100 - 37.5 \times t + 9.925 \times t^2 - 3.2696 \times t^3 + 1.2789 \times t^4 - 0.50124 \times t^5$, t= 1.5

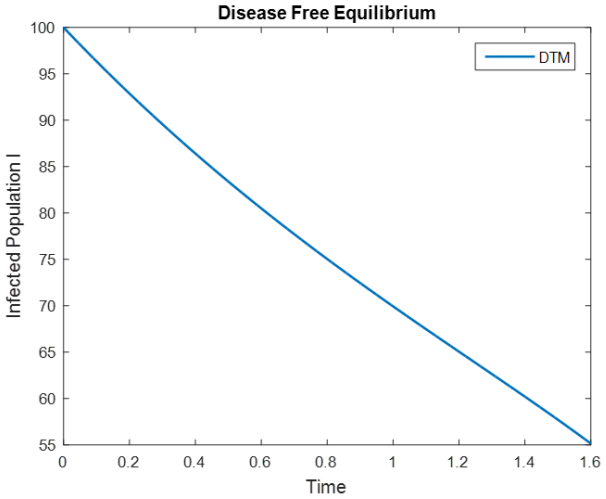
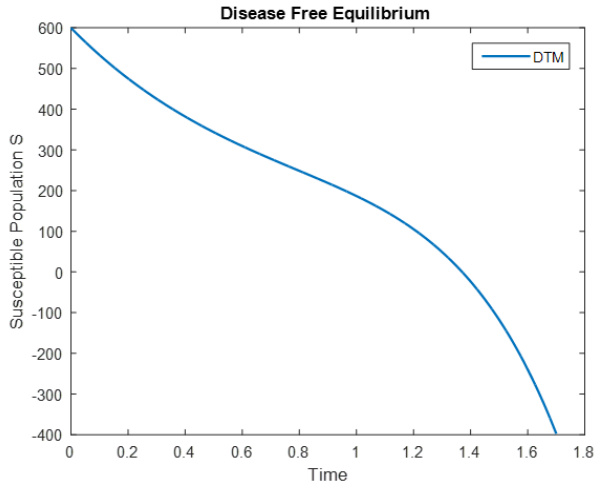


Fig. 4: Infected peoples $i(t)$, $B = 0.32$, $\mu = 0.2, \beta = 0.01, \gamma = 0.2, \alpha = 0.01, \sigma = 0.25$

Case 2: For numerical results, we used the followings Table 2 values of parameters are considered form. The tabular form and graphs are shown in Figs. 5-7.

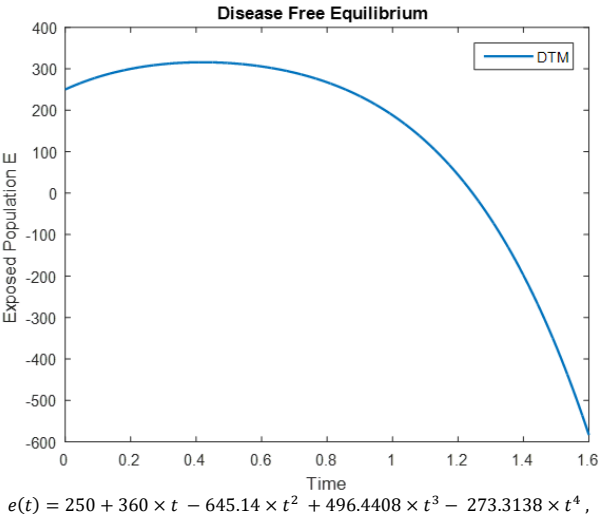
Table 2: Parameters values of the model for case 2

Parameters	Values	Parameters	Values
β	0.01	B	0.32
μ	0.2	α	0.01
σ	0.75	$S(0)$	600
γ	0.2	$E(0)$	250
$I(0)$	100	$R(0)$	50



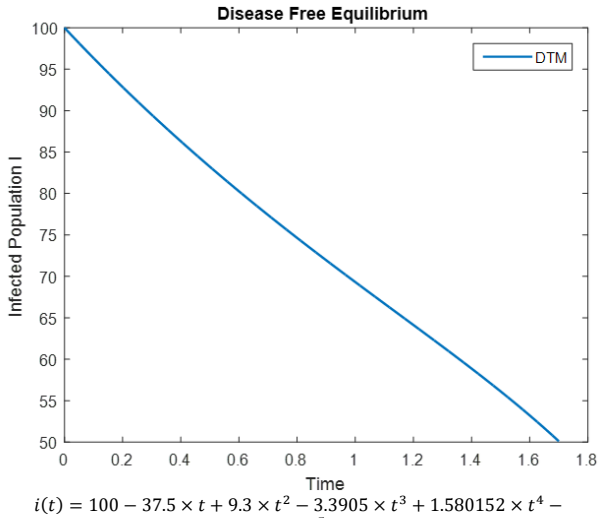
$s(t) = 600 - 719.68t + 544.308 \times t^2 - 326.2832 \times t^3 + 170.48215t^4 - 82.27416 \times t^5$, t=1.5

Fig. 5: Susceptible peoples; $s(t)$ $B = 0.32,\ \mu = 0.2, \beta = 0.01, \gamma = 0.2, \alpha = 0.01, \sigma = 0.75$



$e(t) = 250 + 360 \times t - 645.14 \times t^2 + 496.4408 \times t^3 - 273.3138 \times t^4$, t=1.2

Fig. 6: Exposed peoples; $e(t)$ $B = 0.32, \mu = 0.2, \beta = 0.01, \gamma = 0.2, \alpha = 0.01, \sigma = 0.75$



$i(t) = 100 - 37.5 \times t + 9.3 \times t^2 - 3.3905 \times t^3 + 1.580152 \times t^4 - 0.67304 \times t^5$, t=1.5

Fig. 7: Infected peoples; $i(t)$ $B = 0.32$ $\mu = 0.2, \beta = 0.01, \gamma = 0.2, \alpha = 0.01, \sigma = 0.75$

Graphs of S(t) by using NSFD For case 1 are shown in Figs. 8-10. Fig. 8 shows that the relation between susceptible and time in a year and Fig. 9 shows the relation between exposed and time in a year also Fig. 10 shows the relation between infected and time in a year. Graphs of S(t) by using NSFD For case 2 are shown in Figs. 11-13. Fig. 11 shows that the relation between susceptible and time in a year and Fig. 12 shows the relation between exposed and time in a year also Fig. 13 shows the relation between infected and time in a year.

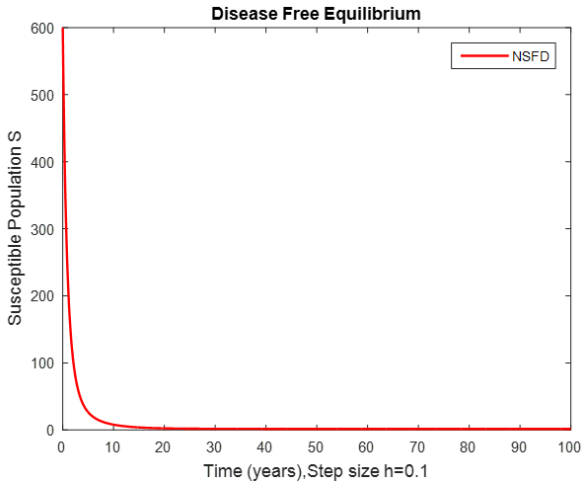


Fig. 8: Susceptible peoples; $B = 0.32,\ \mu = 0.2,\ \beta = 0.01,\ \gamma = 0.2, \alpha = 0.01,\ \sigma = 0.25$

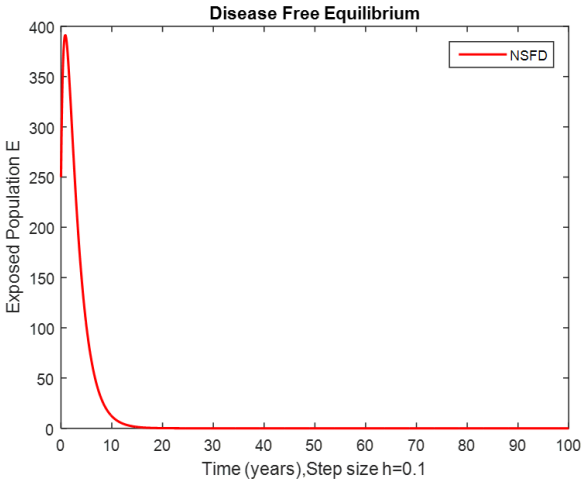


Fig. 9: Exposed peoples; $B = 0.32,\ \mu = 0.2, \beta = 0.01, \gamma = 0.2, \alpha = 0.01, \sigma = 0.25$

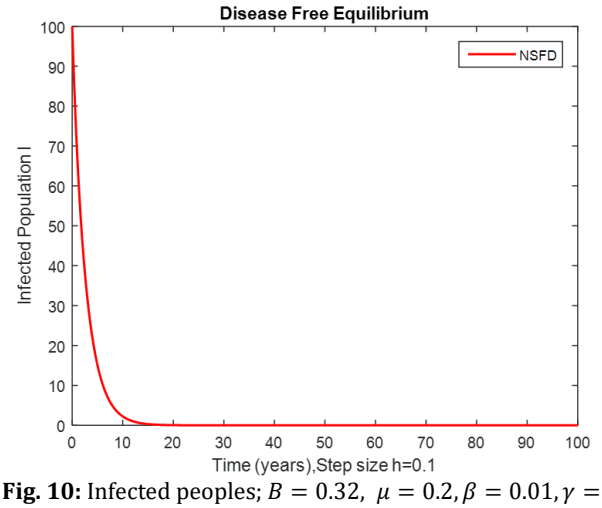


Fig. 10: Infected peoples; $B = 0.32,\ \mu = 0.2, \beta = 0.01, \gamma = 0.2, \alpha = 0.01, \sigma = 0.25$

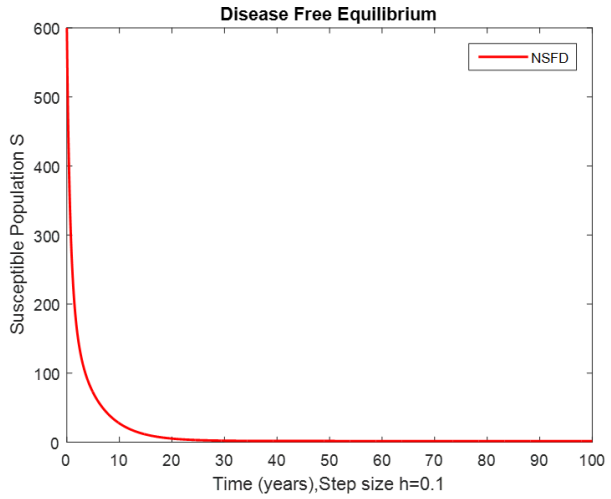


Fig. 11: Susceptible peoples; $B = 0.32,\ \mu = 0.2, \beta = 0.01, \gamma = 0.2, \alpha = 0.01, \sigma = 0.75$

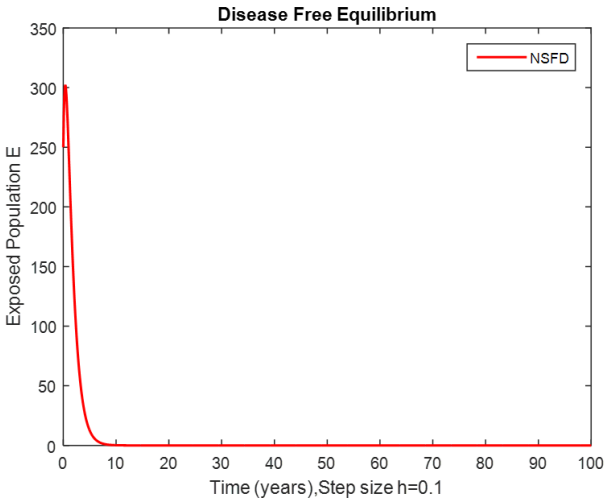


Fig. 12: Exposed peoples; $B = 0.32,\ \mu = 0.2, \beta = 0.01, \gamma = 0.2, \alpha = 0.01, \sigma = 0.75$

5. Conclusion

Many researchers apply many numerical techniques to solve many mathematical models. To analyze SEIR measles model, we use two different techniques Differential transformation method and Non Standard finite difference scheme. After comparison our results we found that rather DTM is very efficient technique, is really worth, offers promoting approach and easy to implement also reliable for complicated systems of linear and non-linear equations. Secondly we construct an unconditionally convergent nonstandard finite difference (NSFD) scheme for SEIR measles model. NSFD preserve the positivity of all values of h (step size).

The NSFD scheme is dynamically consistent, easy to implement and show a good accidence with analytical consequences obtained by dynamics analysis of the model. This method proved to be very efficient technique for solving epidemic models. To analyze SEIR measles model, we use two different cases of measles therapy rate σ and plot the S, E, I against time in years. In each case, we discussed two different cases for the different values of measles therapy rate σ to analyze for what reason the exposed peoples are effected.

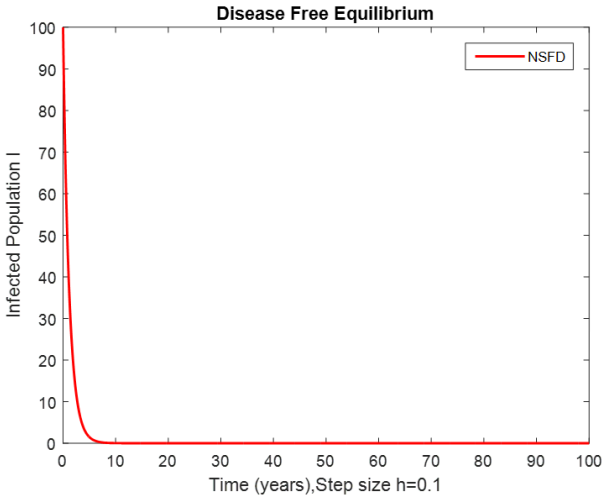


Fig. 13: Infected peoples; $B = 0.32,\ \mu = 0.2, \beta = 0.01, \gamma = 0.2, \alpha = 0.01, \sigma = 0.75$

We conclude that, in high measles prevalence countries, testing diagnosis and exposed individuals at latent period therapy will have a much greater impact on the disease burden. By the isolation of the infections individuals from the other ones, the spread of disease into a population can be controlled as the therapy rate σ will be decreased. For this purpose, appropriate measures should be taken so that the interaction between infectious and susceptible children should be minimized. Moreover, the analysis of epidemic models using nonstandard finite difference scheme reveals that, the method provides rapidly convergent series solution by little iteration and avoids the massive computational work.

Compliance with ethical standards

Conflict of interest

The authors declare that they have no conflict of interest.

References

Akinboro FS, Alao S, and Akinpelu FO (2014). Numerical solution of SIR model using differential transformation method and variational iteration method. General Mathematics Notes, 22(2): 82-92.

Anguelov R and Lubuma JMS (2001). Contributions to the mathematics of the nonstandard finite difference method and applications. Numerical Methods for Partial Differential Equations: An International Journal, 17(5): 518-543. https://doi.org/10.1002/num.1025

Chen CJ and Wu WJ (1996). Application of the Taylor differential transformation method to viscous damped vibration of hard and soft spring system. Computers and Structures, 59(4): 631-639. https://doi.org/10.1016/0045-7949(95)00304-5

Chen CK and Ho SH (1996). Application of differential transformation to eigenvalue problems. Applied Mathematics and Computation, 79(2-3): 173-188. https://doi.org/10.1016/0096-3003(95)00253-7

Chen CL and Sy-Hong L (1996). Application of Taylor transformation to nonlinear predictive control problem. Applied Mathematical Modelling, 20(9): 699-710. https://doi.org/10.1016/0307-904X(96)00050-9

Dimitrov DT and Kojouharov HV (2005). Analysis and numerical simulation of phytoplankton–nutrient systems with nutrient loss. Mathematics and Computers in Simulation, 70(1): 33-43. https://doi.org/10.1016/j.matcom.2005.03.001

Grenfell BT (1992). Chance and chaos in measles dynamics. Journal of the Royal Statistical Society. Series B (Methodological), 54(2): 383-398. https://doi.org/10.1111/j.2517-6161.1992.tb01888.x

Gumel AB, Patidar KC, and Spiteri RJ (2005). Asymptotically consistent non-standard finite-difference methods for solving mathematical models arising in population biology. In: Mickens RE (Ed.), Advances in the applications of nonstandard finite difference schemes: 385-421. World Scientific, Singapore. https://doi.org/10.1142/9789812703316_0009

Hethcote HW (2000). The mathematics of infectious diseases. Society for Industrial and Applied Mathematics Review, 42(4): 599-653. https://doi.org/10.1137/S0036144500371907

Jang MJ and Chen CL (1997). Analysis of the response of a strongly nonlinear damped system using a differential transformation technique. Applied Mathematics and Computation, 88(2-3): 137-151. https://doi.org/10.1016/S0096-3003(96)00308-6

Jang SRJ (2005). Nonstandard finite difference methods and biological models. In: Mickens RE (Ed.), Advances in the applications of nonstandard finite difference schemes: 423-457. World Scientific, Singapore. https://doi.org/10.1142/9789812703316_0010

Mickens RE (2003). A nonstandard finite-difference scheme for the Lotka–Volterra system. Applied Numerical Mathematics, 45(2-3): 309-314. https://doi.org/10.1016/S0168-9274(02)00223-4

Moaddy K, Hashim I, and Momani S (2011). Non-standard finite difference schemes for solving fractional-order Rössler chaotic and hyperchaotic systems. Computers and Mathematics with Applications, 62(3): 1068-1074. https://doi.org/10.1016/j.camwa.2011.03.059

Momoh AA, Ibrahim MO, Uwanta IJ, and Manga SB (2013). Mathematical model for control of measles epidemiology. International Journal of Pure and Applied Mathematics, 87(5): 707-717. https://doi.org/10.12732/ijpam.v87i5.4

Mounim AS and de Dormale BM (2004). A note on Mickens' finite-difference scheme for the Lotka–Volterra system. Applied Numerical Mathematics, 51(2-3): 341-344. https://doi.org/10.1016/j.apnum.2004.06.014

Murray J (2003). Mathematical biology: I. An introduction. 3rd Edition, Springer, Berlin, Germany.

Ochoche JM and Gweryina RI (2014). A mathematical model of measles with vaccination and two phases of infectiousness. IOSR Journal of Mathematics, 10(1): 95-105. https://doi.org/10.9790/5728-101495105

Pukhov GE (1978). Computational structure for solving differential equations by Taylor transformations. Cybernetics, 14(3): 383-390. https://doi.org/10.1007/BF01074670

Pukhov GE (1981). Expanison formulas for differential transforms. Cybernetics, 17(4): 460-464. https://doi.org/10.1007/BF01082476

Pukhov GE (1982). Differential transforms and circuit theory. International Journal of Circuit Theory and Applications, 10(3): 265-276. https://doi.org/10.1002/cta.4490100307

Pukhov GE (1986). Differential transformations and mathematical modelling of physical processes. Naukova Dumka, Kiev, Ukraine. **PMid:2944207**

Taylor AH, Harris JRW, and Aiken J (1986). Distribution of phytoplankton under stratification. Marine Interfaces Ecohydrodynamics, 42: 313-330. https://doi.org/10.1016/S0422-9894(08)71052-3

Zhou JK (1986). Differential transformation and its applications for electrical circuits. Huarjungs University Press, Wuuhahn, China. **PMCid:PMC341339**