



Scientific Inquiry and Review (SIR)

Volume 4, Issue 4, December 2020

ISSN (P): 2521-2427, ISSN (E): 2521-2435

Journal DOI: <https://doi.org/10.32350/sir>

Issue DOI: <https://doi.org/10.32350/sir.44>

Homepage: <https://journals.umt.edu.pk/index.php/SIR/Home>

Journal QR Code:



Article **An Efficient Numerical Method for the Solution of the Polio Virus (Poliomyelitis) Epidemic Model with the Role of Vaccination**

Author(s) Muhammad Rafique, Naveed Shahid, Nauman Ahmed, Tahira Sumbal Shaikh, Muhammad Asif, Muhammad Ozair Ahmad

Online Published December 2020

Article DOI <https://doi.org/10.32350/sir.44.02>

QR Code of Article



Muhammad Rafique

To cite this Article Rafique M, Shahid N, Nauman A, et al. An efficient numerical method for the solution of the polio virus (Poliomyelitis) epidemic model with the role of vaccination. *Sci Inquiry Rev.* 2020;4(4):15–30. [Crossref](#)

Copyright Information This article is open access and is distributed under the terms of Creative Commons Attribution – Share Alike 4.0 International License.



A publication of the School of Science, University of Management and Technology Lahore, Pakistan.

Indexing & Abstracting



An Efficient Numerical Method for the Solution of the Polio Virus (Poliomyelitis) Epidemic Model with the Role of Vaccination

Muhammad Rafiq^{1*}, Naveed Shahid², Nauman Ahmed²,
Tahira Sumbal Shaikh³, Muhammad Asif³, Muhammad Ozair Ahmad⁴

¹Department of Mathematics, Faculty of Sciences,
University of Central Punjab, Lahore, Pakistan

²Department of Mathematics and Statistics,
The University of Lahore, Lahore, Pakistan

³Department of Mathematics,
Lahore College for Women University, Lahore, Pakistan

⁴Department of Mathematics & Statistics,
Minhaj University, Lahore, Pakistan

*m.rafiq@ucp.edu.pk

Abstract

Mathematical modeling of a communicable disease is an effective way to describe the behavior and dynamics of the disease. It builds on our understanding of the transmission of a contagion in a population. In this paper, we explore the transmission dynamics of the polio virus (poliomyelitis) with vaccination using standard methods. We formulate an unconditionally stable Non-Standard Finite Difference (NSFD) scheme for a continuous system of the epidemic polio virus. The designed scheme to approximate the solution is bounded, consistent with the underlying model. The proposed numerical scheme preserves the positivity of the stated variables which is necessary for any population dynamical system. It is used to calculate the numerical solutions of the epidemic model for different step sizes "h". Two other numerical schemes are enforced to find the solution of the proposed system. Finally, the comparison of the NSFD technique with these methods proves its validity and effectiveness.

Keywords: convergence, mathematical modeling, Non-Standard Finite Difference (NSFD) method, polio virus, vaccination

2010 Mathematics Subject Classification: 65P99, 39A30, 92B99

Introduction

Infectious diseases are caused by pathogenic microorganisms such as bacteria, viruses, parasites and fungi. Tuberculosis, influenza, and polio are some examples of infectious diseases. These diseases are also

known as catching or transmissible diseases. Polio, biologically named as poliomyelitis, is a disabling and lifelong disease for human beings which can spread rapidly. It is an epidemic which spreads in the human population from person to person [1]. It damages the human spinal cord which limits the mobility of a person. In biological terms, this situation is called paralysis. In the beginning of the 20th century, polio became a frequent disease in the developed countries and thousands of children were paralyzed due to it. In the mid-20th century, medical specialists successfully developed the vaccination for the polio virus. After the discovery of the vaccination, it was brought under control and vaccination became easily available in 1955 [2].

Poliomyelitis is a highly infectious disease caused by the poliovirus. The word poliomyelitis comes from the Greek words “polios” for gray and “myelos” for marrow [3]. Polio can spread by direct interaction with an affected person or any surface infected with the air-borne droplets of the saliva of an infected person. In the first stage, polio virus enters into the infected person through the nose or mouth and then travels to the guts where it multiplies. In the second stage, the virus enters into the blood stream where “anti-polio” antibodies are created. At that stage, the development of the virus is stopped in most cases and the individual gets permanent protection against the polio virus [4]. Otherwise, the infected person becomes paralyzed or in some cases, some people die due to this disease. However, in most infections caused by the poliovirus, there are few typical signs [5]. Global efforts to eliminate wild polioviruses continue, with types 1 and 3 of wild polioviruses in four countries (Nigeria, India, Afghanistan, and Pakistan) and producing fewer than 2000 cases of paralytic polio per annum, globally [6]. In fact, 95% of all individuals exposed to the polio virus have no signs at all even under common conditions [7,8]. The remaining 5% of the infected people experience bare signs [9,10]. It is expected that about one individual of every 1,000 who contract the disease experience muscular paralysis [7,2].

Polio cases can be divided into the following three types:

- Abortive polio
- Non- Paralytic polio
- Paralytic polio

In the paralytic type of cases, polio is a minor illness with viral like signs such as sore throat and fever. Non-paralytic polio typically manifests the symptoms of abortive polio, such as neck toughness. Most of the patients of non-paralytic polio recover completely from the disease. However, a small number of infected people remain infected for the whole life and become paralytic [11]. Some scholars concluded that a small percentage of individuals who do grow paralytic polio may be physically susceptible to the disease and the remaining people may have a natural immunity or protection against the polio virus [12]. In 1953, Jonas Salk created the first Inactivated Polio Vaccine (IPV) using the virus grown on the kidney cells of monkeys. In 1954, this vaccine (IPV) was administered on a huge population of about sixteen hundred thousand children in three developed countries including United States of America, Canada, and Finland [13]. IPV vaccine was administered all over the USA in April 1955. Consequently, the number of the active cases of poliomyelitis in the country decreased rapidly [14]. Albert Sabin, an American microbiologist, prepared an Oral Live-virus Vaccine (OPV) for human protection against the polio virus. In 1963, an oral vaccine (Sabin's oral vaccine) was discovered. It is inexpensive to produce, buy and take, and it also creates 'herd immunity' in individuals not vaccinated previously [11]. Polio Eradication Inventiveness was launched at a worldwide level following a resolution in 1988 by the World Health Assembly. At that time, more than 1000 kids were paralyzed globally each day due to polio. Since then, 2.5 billion kids have been protected against polio. This became possible only by the collaboration of more than 200 nations and 20 million helpers, supported by an international investment of more than 8 billion US dollars [15]. There are three countries in the whole world where the polio virus is still present. Unfortunately, Pakistan is one of them. Global Polio Eradication Inventiveness states that six healthy heavens for the polio virus are found in the whole world. Sadly, four out of these six said heavens are in Pakistan [16].

Mathematical Model

2.1. Assumptions

- The joining and dropping rates for a person in the total population are λ and μ , respectively.

- The exposed individuals move from the exposed compartment to the infected compartment after spending the incubation time period.
- In the susceptible compartment, those individuals are included that have contact with both the exposed and infected persons.
- The susceptible compartment also consists of individuals that are vaccinated at the time of birth and cannot catch the disease for the whole life.

The total population in the underlying system is partitioned into four classes namely susceptible, exposed, infected and vaccinated. The susceptible compartment contain the individuals having the maximum chances of catching the disease. An exposed individual is the one who is infected but cannot transfer the disease before the incubation period. The infected compartment contains individuals to whom the virus has been blown out. Vaccinated individuals have established protection against the disease.

2.2. Parameters Used in the Model

S = total number of individuals in the susceptible compartment

E = total number of individuals in the exposed compartment

I = total number of individuals in the infected compartment

V = total number of individuals in the vaccinated compartment

A = individuals in the whole population

μ = natural death rate in the total population

β = proportion of catching infection after contact with the infected population in one unit of time

$r\beta$ = proportion of catching infection after contact with the exposed population in one unit of time

r = rate of the blowout of infection by exposed individuals

v = proportion of more individuals that move from the susceptible compartment to the vaccinated compartment

v_1 = rate at which the exposed individuals are given a vaccine as they are asymptomatic for polio

B = rate of transferring the exposed individuals to the infected compartment

a = death rate of all individuals in the population due to polio

The flow diagram given below describes the compartmental dynamics of the blowout of the polio virus.

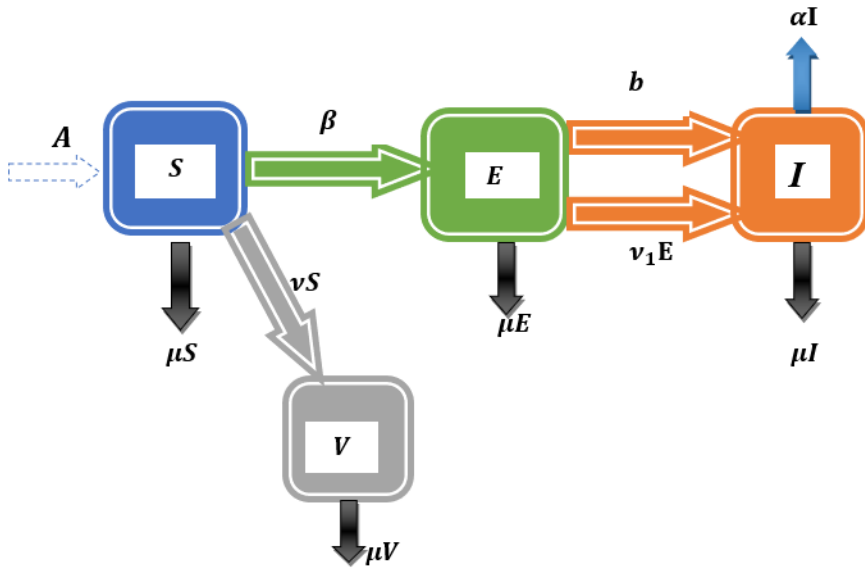


Figure 1. Flow chart of polio

2.3. Transmission Model of the Polio Virus with Vaccination

From the above flow diagram, we can /infer the system of the first order nonlinear ordinary differential equations given below [17].

$$dS/dt = A - \beta SI - r\beta SE - (\mu + \nu)S \tag{1}$$

$$dE/dt = \beta SI + r\beta SE - (b + \mu + \nu_1) E \tag{2}$$

$$dI/dt = (b + \nu_1) E - (\mu + \alpha)I \tag{3}$$

$$dV/dt = \nu S - \mu V \tag{4}$$

The given initial conditions are: $S(0) = s_0 > 0; E(0) = E_0 > 0; I(0) = I_0 > 0; V(0) = V_0 > 0$

3. Analysis of the Mathematical Model

3.1. Fixed Points of the Model

The equilibrium points/fixed points can be originated by setting the equations (1)-(4) equal to zero, then we have

(i) $E_0 = (\frac{A}{(\mu+\nu)}, 0, 0, \frac{\nu A}{\mu(\mu+\nu)})$, a disease-free equilibrium point

(ii) $E_1 = (S^*, E^*, I^*, V^*)$, an endemic equilibrium point

where

$$S^* = \frac{A}{(\mu + \nu)} \frac{1}{R}$$

$$E^* = \frac{A}{(b + \mu + \nu_1)} \left[1 - \frac{1}{R} \right]$$

$$I^* = \frac{(b + \nu_1)}{(\mu + \alpha)} E^*$$

$$V^* = \frac{\nu}{\mu} S^*$$

where $R = \frac{A\beta\{(b+\nu_1) + r(\mu+\alpha)\}}{(\mu+\nu)(\mu+\alpha)(b+\mu+\nu_1)}$, the basic reproductive number

$$R = R_0 + R_1$$

$$= \frac{A\beta(b + \nu_1)}{(\mu + \nu)(\mu + \alpha)(b + \mu + \nu_1)} + \frac{A\beta r}{(\mu + \nu)(b + \mu + \nu_1)}$$

The stability of the endemic equilibrium point is determined by finding the Jacobian matrix of the right-hand side of eq.(1)-(4). To find the eigenvalues, put $\det(J - \xi I) = 0$, where

J =Jacobian matrix at the endemic equilibrium point

ξ =eigenvalue

I =identity matrix

Eigenvalues are obtained by solving $\xi^3 + c_0\xi^2 + c_1\xi + c_2 = 0$

where

$$c_0 = \frac{A}{S^*} + \frac{\beta S^* I^*}{E^*} + (\mu + \alpha)$$

$$c_1 = \frac{A(\mu + \alpha)}{S^*} + \frac{\beta I^* A}{E^*} + r\beta S^* (\beta I^* + r\beta E^*)$$

$$c_2 = \beta S^* (\beta I^* + r\beta E^*) \{r(\mu + \alpha) + b + \nu_1\}$$

According to Routh-Hurwitz [14], the stability of the mathematical models can be analysed as:

“The equilibrium point (fixed point) is stable when the constants c_0, c_1, c_2 obey the inequalities $c_0 > 0, c_1 > 0, c_0 c_1 > c_2$.” This confirms the stability of the endemic equilibrium point for $R > 1$.

3.2. Parameter Values

Table 1. Suggested Values of Parameter

Equilibrium points	A	β	r	μ	v	v_1	b	α
EE	1000	0.002	0.5	0.5	0.6	0.001	0.9	0.6
DFE	584	0.001102	0.5	0.78	1.4	0.001	0.9	3

Using the above values of the parameters, we calculated the values of the Disease Free Equilibrium Point and the Endemic Equilibrium Point.

Table 2. Steady States of the Model

Disease Free Equilibrium Point	Endemic Equilibrium Point
$R = 0.1297 < 1$	$R = 1.7119 > 1$
(S_0, E_0, I_0, V_0) $= \left(\frac{A}{(\mu + v)}, 0, 0, \frac{vA}{\mu(\mu + v)} \right)$	(S^*, E^*, I^*, V^*) $= \left(\frac{A}{(\mu + v)} \frac{1}{R}, \frac{A}{(b + \mu + v_1)} \left[1 - \frac{1}{R} \right], \frac{(b + v_1)}{(\mu + \alpha)} E^*, \frac{v}{\mu} S^* \right)$
(S_0, E_0, I_0, V_0) $= (267.9, 0, 0, 480.8)$	(S^*, E^*, I^*, V^*) $= (531.04, 296.82, 243.12, 637.25)$

3.3. Numerical Method

In the current article, a numerical model of the proposed continuous model of the polio virus disease comprising a system of differential equations is constructed. For a positive real number h , suppose that $m = 0, 1, 2, \dots$ and for any time $t \geq 0$, we define the time taken at each grid point by $t_m = mh$, where h is supposed to be the time interval in the partition of the time taken which is fixed for each interval of time. Also, suppose that $S(t), E(t), I(t), V(t)$ represent the solution of the state variables at any time $t \geq 0$, while $S(t_m), E(t_m), I(t_m), V(t_m)$ are represented by the solution of the proposed system at time grid t_m . According to the numerical computations, the approximate solutions at the same given points t_m are denoted by S^m, E^m, I^m and V^m , respectively.

The Non-Standard Finite Difference (NSFD) technique is among the discrete versions of the system of mathematical model comprising the differential equations that obey the rules defined by Mickens and is extensively used by various researchers [18-20]. The fundamental theory of this discretization method used for mathematical modelling was proposed by Mickens [21]. This technique for approximating the solutions of various physical systems gives better results as compared to other numerical methods. It is dynamically consistent with the concerned continuous model and stability. Moreover, this type of scheme is independent of the grid size and preserves the most important trait of the dynamical systems, that is, the positivity of the values of the stated variables. It converts the continuous model into a discrete model. Mickens presented certain rules for finding the best difference equations. Here, we construct an NSFD scheme for the mathematical modelling of the polio spread with vaccination.

First, we make approximation to $\frac{dS}{dt}$, $\frac{dE}{dt}$, $\frac{dI}{dt}$ and $\frac{dV}{dt}$ using first order forward differences.

$$\frac{dS(t)}{dt} = \frac{1}{h} [S(t+h) - S(t)] + O(h) \quad \text{as } h \rightarrow 0 \quad t = t_m$$

$$\frac{dE(t)}{dt} = \frac{1}{h} [E(t+h) - E(t)] + O(h) \quad \text{as } h \rightarrow 0 \quad t = t_m$$

$$\frac{dI(t)}{dt} = \frac{1}{h} [I(t+h) - I(t)] + O(h) \quad \text{as } h \rightarrow 0 \quad t = t_m$$

$$\frac{dV(t)}{dt} = \frac{1}{h} [V(t+h) - V(t)] + O(h) \quad \text{as } h \rightarrow 0 \quad t = t_m$$

Using these approximations to the derivatives, systems (1)-(4) can be written as follows:

$$\frac{S^{n+1} - S^n}{h} = A - \beta S^{n+1} I^n - r \beta S^{n+1} E^n - (\mu + \nu) S^{n+1}$$

$$\frac{E^{n+1} - E^n}{h} = \beta S^{n+1} I^n + r \beta S^{n+1} E^n - (b + \mu + \nu_1) E^{n+1}$$

$$\frac{I^{n+1} - I^n}{h} = (b + \nu_1) E^{n+1} - (\mu + \alpha) I^{n+1}$$

$$\frac{V^{n+1} - V^n}{h} = \nu S^{n+1} - \mu V^{n+1}.$$

Solving the above equations for S^{n+1} , E^{n+1} , I^{n+1} and V^{n+1} , we have

$$S^{n+1} = \frac{S^n + h A}{1 + h\beta I^n + hr\beta E^n + h(\mu + \nu)} \quad (5)$$

$$E^{n+1} = \frac{E^n + h\beta S^{n+1}I^n + hr\beta S^{n+1}E^n}{1 + h(b + \mu + \nu_1)} \quad (6)$$

$$I^{n+1} = \frac{I^n + h(b + \nu_1)E^{n+1}}{1 + h(\mu + \alpha)} \quad (7)$$

$$V^{n+1} = \frac{V^n + h\nu S^{n+1}}{1 + h\mu} \quad (8)$$

Equations (5)-(8) comprise the discrete version of the polio models(1)-(4)developed by the novel numerical technique, that is, NSFD.

3.4. Convergence Analysis

In this section, we shall discuss the convergence of the proposed numerical model. Let us consider

$$F(S, E, I, V) = \frac{S + h A}{1 + h\beta I + hr\beta E + h(\mu + \nu)}$$

$$G(S, E, I, V) = \frac{E + h\beta SI + hr\beta SE}{1 + h(b + \mu + \nu_1)}$$

$$H(S, E, I, V) = \frac{I + h(b + \nu_1)E}{1 + h(\mu + \alpha)}$$

$$L(S, E, I, V) = \frac{V + h\nu S}{1 + h\mu}$$

The Jacobian for this system is

$$J(S, E, I, V) = \begin{bmatrix} \frac{\partial F}{\partial S} & \frac{\partial F}{\partial E} & \frac{\partial F}{\partial I} & \frac{\partial F}{\partial V} \\ \frac{\partial G}{\partial S} & \frac{\partial G}{\partial E} & \frac{\partial G}{\partial I} & \frac{\partial G}{\partial V} \\ \frac{\partial H}{\partial S} & \frac{\partial H}{\partial E} & \frac{\partial H}{\partial I} & \frac{\partial H}{\partial V} \\ \frac{\partial L}{\partial S} & \frac{\partial L}{\partial E} & \frac{\partial L}{\partial I} & \frac{\partial L}{\partial V} \end{bmatrix}.$$

The numerical scheme given by the equations (5) – (8) will converge to a fixed point of the system if and only if the absolute value

of all eigenvalues λ_i , $i = 1, 2, 3, 4$ computed from the Jacobin matrix and related to the given system is less than 1 at the equilibrium point, that is, $|\lambda_i| < 1$, for $i = 1, 2, 3, 4$. The Jacobin matrix at disease-free equilibrium point

$(S, E, I, V) = \left(\frac{A}{(\mu+v)}, 0, 0, \frac{vA}{\mu(\mu+v)}\right)$ is given by

$$J^* \left(\frac{A}{(\mu+v)}, 0, 0, \frac{vA}{\mu(\mu+v)} \right) = \begin{bmatrix} \frac{1}{1+h(\mu+v)} & -\left(\frac{A}{(\mu+v)} + hA\right)hr\beta & -\left(\frac{A}{(\mu+v)} + hA\right)h\beta & 0 \\ 0 & \frac{1+hr\beta\left(\frac{A}{(\mu+v)}\right)}{1+h(b+\mu+v_1)} & \frac{h\beta\left(\frac{A}{(\mu+v)}\right)}{1+h(b+\mu+v_1)} & 0 \\ 0 & \frac{h(b+v_1)}{1+h(\mu+\alpha)} & \frac{1}{1+h(\mu+\alpha)} & 0 \\ \frac{hv}{1+h\mu} & 0 & 0 & \frac{1}{1+h\mu} \end{bmatrix}$$

$$\lambda_1 = \frac{1}{1+h(\mu+v)} < 1, \quad \lambda_2 = \frac{1}{1+h\mu} < 1.$$

The remaining two eigenvalues are given by the matrix

$$J^* = \begin{bmatrix} \frac{1+hr\beta\left(\frac{A}{(\mu+v)}\right)}{1+h(b+\mu+v_1)} & \frac{h\beta\left(\frac{A}{(\mu+v)}\right)}{1+h(b+\mu+v_1)} \\ \frac{h(b+v_1)}{1+h(\mu+\alpha)} & \frac{1}{1+h(\mu+\alpha)} \end{bmatrix}.$$

The following lemma is established to evaluate the eigenvalues of the Jacobian J^* related to the proposed model.

Lemma: If the quadratic equation (characteristic equation) associated with the Jacobian matrix $\lambda^2 - \lambda A + B = 0$ has the roots λ_1, λ_2 , then both roots have absolute values less than 1 if and only if the conditions mentioned below hold

- (a) $1 - A + B > 0$
- (b) $1 + A + B > 0$
- (c) $B < 1$

Now, suppose that

$$A = \text{Trace } J^*, \quad B = \text{Det} J^*.$$

Therefore,

$$A = \frac{\left[1 + hr\beta \left(\frac{A}{(\mu+v)}\right)\right] [1 + h(\mu + \alpha)] + [1 + h(b + \mu + v_1)]}{[1 + h(b + \mu + v_1)][1 + h(\mu + \alpha)]}$$

$$B = \frac{1 + hr\beta \left(\frac{A}{(\mu+v)}\right) - h^2 \beta \left(\frac{A}{(\mu+v)}\right) (b + v_1)}{[1 + h(b + \mu + v_1)][1 + h(\mu + \alpha)]}$$

The above values of A and B satisfy all the conditions of the above lemma. The absolute value of both eigenvalues of J^* is less than 1 for every value of the step size 'h' when $R < 1$. This shows that the developed numerical scheme (5)-(8) converges unconditionally to the disease-free equilibrium point for any value of h whenever $R < 1$.

3.5. Numerical Experiments

Using the suggested parameter values given in Table 1, a numerical example is illustrated below to verify our results.

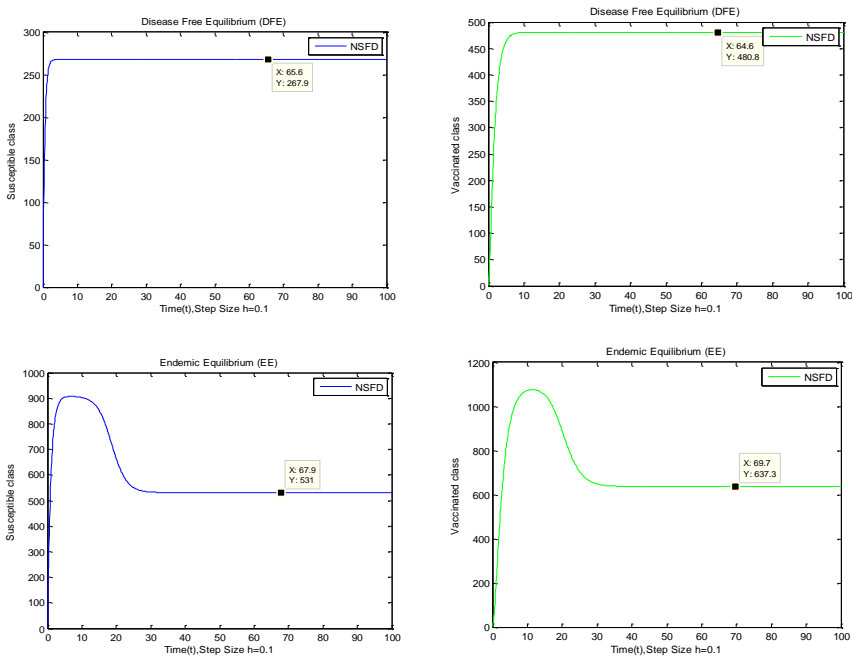


Figure 2. Numerical solution

4. Results and Discussion

In this paper, we have construed a polio model with the role of vaccination. It was found that two parameter values R_0 and R_1 are associated with the interactive contact of the susceptible individuals with the exposed population and with the individuals in the infected class, respectively. The quantity R is the sum of the quantities R_0 and R_1 . The basic reproductive number, stability dynamics of the model and the disease dies out are completely determined by the value of R . It is visualized that when $R \leq 1$, the behavior of the disease-free equilibrium remains stable. It means that the disease does not exist. On the other hand, there exists a unique endemic equilibrium when $R > 1$ and the disease spreads out. We used the discretization technique, that is, Finite Difference scheme with Mickens' non-standard rules to solve the system of ordinary differential equation numerically and the results were obtained using different values of the step size 'h'. We compared these results with Euler and RK 4 and found that these two methods are convergent for small step sizes but diverge for the large value of 'h'. So, these methods are conditionally convergent. The designed discrete numerical technique (NSFD) has an unconditional convergence towards the fixed point of the model, that is, it preserves the convergence condition for a very large grid size 'h' ($h = 100$). The following sketched table describes the behavior of the proposed numerical scheme at each grid point h for both equilibrium points, namely disease-free equilibrium and endemic equilibrium points.

Table 3. Comparison of Euler RK 4 and NSFD Scheme

h	Euler	RK-4	NSFD scheme
0.1	Convergence	Convergence	Convergence
0.9	EE = Convergence DFE = Divergence	EE = Convergence DFE = Divergence	Convergence
10	Divergence	Divergence	Convergence
100	Divergence	Divergence	Convergence
1000	Divergence	Divergence	Convergence

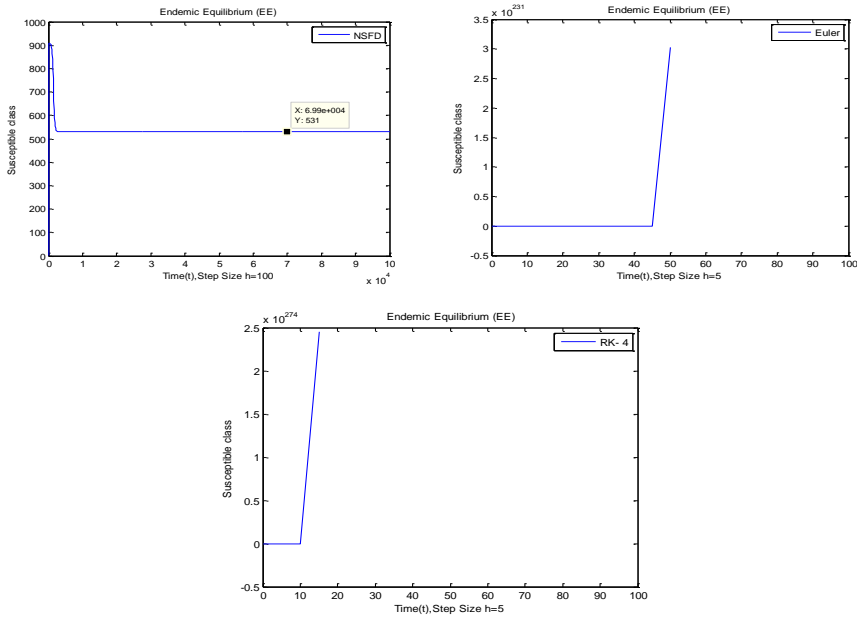


Figure 3. Numerical solutions

5. Conclusion

We adopted the discretization technique, that is, Finite Difference scheme with Mickens’ non-standard rules which is unconditionally convergent for the transmission dynamics of the polio disease. Unlike RK 4 and Euler which diverge for large values of ‘h’, the results of the developed numerical scheme are closed to the fixed points or points of equilibrium for the continuous model at all values of ‘h’. The NSFD scheme is dynamically consistent, easy to implement and show similarity with the analytical results obtained by the dynamic analysis of the model. Moreover, numerical experiments yield that the proposed numerical scheme remains consistent with the biological nature, preserving all of its essential properties.

References

[1] Adu F, Iber J, Bukbuk D, et al. Isolation of recombinant type 2 vaccine-derived poliovirus (VDPV) from a Nigerian child. *Virus Res.* 2007 Jul 1;127(1):17-25.
<https://doi.org/10.1016/j.virusres.2007.03.009>

[2] Agarwal M, Bhadauria AS. Modeling spread of polio with the role of vaccination. *Appl Math Int J.* 2011;6(2):552-71.

- [3] Bunimovich-Mendrazitsky S, Stone L. Modeling polio as a disease of development. *J Theoretical Bio.* 2005 Dec 7;237(3):302-15. <https://doi.org/10.1016/j.jtbi.2005.04.017>
- [4] Okonek BM. Development of polio vaccines. 2001. www.accessexcellence.org/AE/AEC/CC/polio.html
- [5] Wheeler MF, Volk WA. *Basic Microbiology*. Lippincott; 1969. <https://www.worldcat.org/title/basicmicrobiology/oclc/598552504>
- [6] Dutta A. Epidemiology of poliomyelitis—options and update. *Vaccine.* 2008 Oct 23;26(45):5767-73. <https://doi.org/10.1016/j.vaccine.2008.07.101>
- [7] Physicians D, Montvale NJ. *Medical Economics.* 2001:778-785.
- [8] Frank MacFarlane Burnet S, Burnet FM, et al. *Natural History of Infectious Disease.* CUP Archive; 1972 Aug 24.
- [9] Neustaedter R. The Vaccine Guide. *Making an Informed Choice Berkeley, California.* 1996;152:107-108.
- [10] BabyCenter. The Polio Vaccine. http://www.babycenter.com/0_the-polio-vaccine_1566.bc
- [11] The history of Vaccine. History of Polio (Poliomyelitis) <http://www.historyofvaccines.org/content/articles/history-polio-poliomyelitis>
- [12] Moskowitz T. *Immunizations the Other Side.* Mothering Spring.1984.
- [13] Monto AS. Francis field trial of inactivated poliomyelitis vaccine: background and lessons for today. *Epidemiol Rev.* 1999 Jan 1;21(1):7-23.
- [14] Strebel PM, Sutter RW, Cochi SL, et al. Epidemiology of poliomyelitis in the United States one decade after the last reported case of indigenous wild virus-associated disease. *Clin Infect Pract.* 1992 Feb 1;14(2):568-79. <https://doi.org/10.1093/clinids/14.2.568>
- [15] Polio Global education initiative. History of Polio. <http://www.polioeradication.org/polioandprevention/Historyofpolio.aspx>

- [16] Rizwan K, Islam M. Change in strategies of training lead toward effective implementation of polio eradication program in Pakistan, does it matter? *Merit Res J Edu*, 2013;2:8-13.
- [17] May RM. *Stability and Complexity in Model Ecosystems*. Princeton university press; 2019 Dec 31.
- [18] Katz R, Graeden E, Abe K, Attal-Juncqua A, Boyce MR, Eaneff S. Mapping stakeholders and policies in response to deliberate biological events. *Heliyon*. 2018 Dec 1;4(12):e01091. <https://doi.org/10.1016/j.heliyon.2018.e01091>
- [19] Ahmed N, Shahid N, Iqbal Z, et al. Numerical modeling of SEIQV epidemic model with saturated incidence rate. *J Appl Environ Bio Sci*. 2018;8(4):67-82.
- [20] Ahmed N, Tahira SS, Rafiq M, et al. Positivity preserving operator splitting nonstandard finite difference methods for SEIR reaction diffusion model. *Open Math*. 2019 Apr 29;17(1):313-30.
- [21] Piyawong W, Twizell EH, Gumel AB. An unconditionally convergent finite-difference scheme for the SIR model. *Appl Math Comput*. 2003 Dec 31;146(2-3):611-25. [https://doi.org/10.1016/S0096-3003\(02\)00607-0](https://doi.org/10.1016/S0096-3003(02)00607-0)