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Research Article

Treatment of Non-linear Epidemiological Smoking Model using Evolutionary Padé-approximation

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Abstract: Smoking is the world's biggest public health concern. In epidemiology, the mechanisms of smoking addiction play a crucial role in mathematical models. In this paper Evolutionary Padé Approximation (*EPA*) scheme has been implemented for the treatment of the non-linear epidemiological smoking model. The evolutionary Padé Approximation scheme transforms the nonlinear epidemiology smoking model into an optimization problem by using Padé-approximation. Sufficient parameter settings for *EPA* have been implemented through MATLAB. Simulations represent numerical solutions of the epidemiology smoking model by solving the established optimization problem. First, the convergence solution of *EPA* scheme on population; potential smokers occasional smokers, heavy smokers, temporary quitters, and smokers who quit permanently have been studied and found to be significant. Evolutionary Padé Approximation has provided a convergence solution regarding the relationship among the different population compartments for diseases free equilibrium, it has been observed that the results *EPA* scheme are more reliable and significant when a comparison is drawn with Non-Standard Finite Difference (*NSFD*) numerical scheme. Finally, the *EPA* scheme reduces the contaminated levels for disease-free equilibrium very rapidly and restricts the spread of smoking within the population.

Keywords: Optimization, Non-linear epidemiological smoking model, Padé approximation, Differential Evolution, Penalty function.

1. INTRODUCTION

Mathematics has a wide scope which includes esoteric mathematics and mathematical modeling. The flow of the process, work, predictions, and results can be judged and measured by implementing the theory and mathematical concepts. Consequently, biologists have become dependent on mathematics to find out the mysteries. Mathematical modeling of biological sciences has been executed by multiple dazzling and intelligent scientists [1-3]. There is a connection between simple mathematical modeling which includes integer order of biological system and differential equations that display their subtleties, dynamics, and complex system that shows their change of construction. Multi-scale and nonlinearity behaviors in this model define the mutual connection between parameters [4]. Currently, many biological models have been studying completely by using classical derivatives [5-7].

Smoking is known as one of the main cause in the world which is harming the healthy community. It hurts the dissimilar organs of the human body. It has become the major cause of more than 1 million demises. Compared to non-smokers, the heart attack ratio in a smoker is more than 70 percent [8]. Similarly, lung cancer is 10% higher than in a non-smoker [9]. The visible diseases of short term smoking are stained teeth, bad breath, coughing, and high blood pressure. So far as the main infections and diseases of a long term smoking are throat cancer, stomach ulcer, lung cancer, gum disease, heart disease, and mouth cancer in the current centuries smoker. Besides, the life of a smoker is

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not more than 13 or 14 years of age.

World Health Origination (WHO) reports proving that smoking is the cause of many individuals in this entire world. Each doctor, scientist, and mathematician is striving hard to confront the severe causes of smoking while mathematicians are trying to form different valuable models of smoking to eradicate the smoking effects. Various smoking models are found that the writers planned: examined the Caputo fractional derivative smoking model [10], the examination of optimal control models expresses the dynamics of smoking qualitative analysis [11], Lung cancer and tobacco smoking examination [9], describe recovery and decrease in the mathematical assessment of tobacco dynamics [12], examined a fractional smoking and many others [8]. The interpretations of the description of smoking global dynamics of a mathematical system of equations [13].

These epidemiological models are vital procedures to investigate and acquire improved information about the development with the help of Mathematical tools which are built on arithmetical and numerical analysis, influence, and the deriving mechanisms, particularly when there is not available any analytical solution. Thoughtful information and knowledge about these model aid in adopting preventive actions and to evaluate their efficiency and effectiveness to avert such infections.

Contemporary meta-heuristics are proposed to cope with the maximum hitches by changing them into optimization problems in recent times. As we know Meta-heuristic algorithms have been formulated by natural phenomena as swarm behaviors [14,15], evolution [16,17], sports strategies [18], water dynamics [19], food foraging behavior [20], etc. Consult the survey article for more detailed studies referred to in [21]. Meta-heuristics is based on approaches that resolve differential equations associated with the non-standard mesh-free methods class. Improvisation of these suggested heuristics to differential equations may also be discovered in [22,23], but here is an issue that these meta heuristics applications to extensive and epidemic models are really difficult to see. The complete population in this model is split into five compartments where prospective smokers, occasional smokers, heavy smokers, temporary quitters and permanent quitters are present. Finding reproductive number was used to analyze disease-free equilibrium. Graphically, numerical findings are provided to demonstrate the model's dynamics.

The study presents an innovative scheme known as Padé-approximation [24] which is based on the Differential Evolution Algorithm to handle the numerical treatment of this model. This suggested computational framework includes the following characteristics: 1) by creating an equivalent problem of optimization by manipulating the Padé approximation extrapolation and interpolation techniques, 2) to maintain positivity through original boundaries and contract conditions by outlining issue limitations, 3) an indispensable prerequisite for building fitness/objective function by using the penalty function approach, 4) Differential Evolution introduction to optimize the fitness function.

This whole paper has been established on these grounds. Section 2 has a comprehensive detail of the nonlinear epidemiology smoking model. Section 3 is based on the fundamentals of Padé approximation, differential evolution, and penalty function developed in the structure of EPA scheme to solve the numerical treatment of the nonlinear epidemiological smoking model. While in section 4, revolves around the analyses of the results which have been presented. Finally, in the last section concluding remarks and findings for future directions have been given.

2. MATHEMATICAL MODEL OF SMOKING

The variables of the model at any time t are defined as P(t): potential smokers, L(t): occasional smokers, S(t): heavy smokers, Q(t): temporary quitters, and R(t): smokers who quit permanently. The smoking model referred to in [25] is in the form of a system of a nonlinear differential equation is:

$$\left.\begin{aligned} P'(t) &= a\big(1-P(t)\big) - bP(t)S(t),\\ L'(t) &= -aL(t) + bP(t)S(t) - cL(t)S(t),\\ S'(t) &= -(a+d)S(t) + cL(t)S(t) + fQ(t),\\ Q'(t) &= -(a+f)Q(t) + d(1-e)S(t),\\ R'(t) &= -aR(t) + edS(t). \end{aligned}\right\} \tag{1}$$

$$P(0) \geq 0, L(0) \geq 0, S(0) \geq 0, Q(0) \geq 0, R(0) \geq 0$$

Where a = rate of natural death, b = contact rate between potential smokers and smokers who smoke occasionally, c = contact rate between temporary quitters and smokers who smoke occasionally, d = rate of giving up smoking, e = remaining fraction of smokers who give up smoking permanently, f = contact rate between smokers and temporary quitters who return to smoking, (1 - e) = fraction of smokers who temporarily give up smoking at a rate d.

$$T(t) = P(t) + L(t) + S(t) + Q(t) + R(t)=1 \quad (2)$$

Suppose that:

$$\left.\begin{array}{c} P'(t) = X_1(t) = a\big(1 - P(t)\big) - bP(t)S(t), \\ L'(t) = X_2(t) = -aL(t) + bP(t)S(t) - cL(t)S(t), \\ S'(t) = X_3(t) = -(a + d)S(t) + cL(t)S(t) + fQ(t), \\ Q'(t) = X_4(t) = -(a + f)Q(t) + d(1 - e)S(t), \\ R'(t) = X_5(t) = -aR(t) + edS(t). \end{array}\right\} \quad (3)$$

Subject to the conditions

$$P_0 = P(0) \geq 0, L_0 = L(0) \geq 0, S_0 = S(0) \geq 0,$$
$$Q_0 = Q(0) \geq 0, R_0 = R(0) \geq 0$$

Reproductive number = $R_0 = \frac{df(1-e)}{(a+d)(a+f)}$

In the case of the disease-free equilibrium point, R_0<1 This shows that the disease will die out

Disease-free equilibrium: E_0=(1,0,0,0,0)

In the case of endemic equilibrium, R_0>1 which shows that the disease spreads in the population?
Endemic equilibrium:

$$E_1 = \left(\frac{a}{a + bS^*}, \frac{ab}{(a + bS^*)(a + cS^*)}, \frac{d(1 - e)S^*}{a + f}, \frac{edS^*}{a}\right)$$

Table 1 Exhibited the parameters [25] of the smoking model.

Table 1. Values of physical parameters of the smoking model

Parameter	Value	Parameters	Value
a	0.04	b	0.23
c	0.30	d	0.20
e	0.40	f	0.25

3. EVOLUTIONARY PADÉ APPROXIMATION SCHEME

The evolutionary Padé Approximation scheme was developed and implemented for the numerical treatment of the HIV/AIDS epidemic model with vertical transmission by using evolutionary Padé-approximation [26]. The design of this scheme is based on Padé-approximation [27], Differential Evolution [28],[29] and penalty function [30]. The evolutionary Padé Approximation scheme has been applied to a nonlinear epidemiology smoking model which involves the following steps.

3.1 Padé-approximation

The concept of a Padé-approximation was launched at the end of the 19th century through the classical theory of continuing fractions. The reasonable (N,M) order function of the approximation of Padé referred in [27]

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

The polynomials $\sum_{i=0}^{N} a_i t^i$ and $\sum_{j=0}^{M} b_j t^j$ are known as Padé approximants. By putting $b_0 \neq 0$ normalizing the above expression and attain the following form:

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

The above expression contains (N + M + 1) undetermined coefficients, applying the Maclaurin series expansions of $P_{N,M}(t)$ to get the target referred to in [24].
Suppose that $P(t)$, $L(t)$, $S(t)$, $Q(t)$ and $R(t)$ are approximated by Padé rational functions as

$$P(t) = \frac{\sum_{i=0}^{N} a_i t^i}{1+\sum_{j=1}^{M} b_j t^j}, L(t) = \frac{\sum_{i=0}^{N} c_i t^i}{1+\sum_{j=1}^{M} d_j t^j}, S(t) = \frac{\sum_{i=0}^{N} e_i t^i}{1+\sum_{j=1}^{M} f_j t^j}$$
$$Q(t) = \frac{\sum_{i=0}^{N} g_i t^i}{1+\sum_{j=1}^{M} h_j t^j}, R(t) = \frac{\sum_{i=0}^{N} k_i t^i}{1+\sum_{j=1}^{M} l_j t^j},$$

Imposing initial conditions

$$\left.\begin{array}{c} P(t_0) = P_0 = 0.5, L(t_0) = L_0 = 0.3, S(t_0) = S_0 = 0.1, \\ Q(t_0) = Q_0 = 0.05, R(t_0) = R_0 = 0.05 \end{array}\right\}$$

It is obtained

$$a_0 = P_0, c_0 = L_0, e_0 = S_0, g_0 = Q_0, k_0 = R_0$$

The discrete-time steps are $t_q = t_0 + qh$; q = 0,1,2,3,...,q_{max}, and the above system of equations (3) reduces as:

$$\left.\begin{aligned}\varepsilon_1(t_q)=0\\ \varepsilon_2(t_q)=0\\ \varepsilon_3(t_q)=0\\ \varepsilon_4(t_q)=0\\ \varepsilon_5(t_q)=0\end{aligned}\right\}$$

Here ε_1, ε_2, ε_3, ε_4 and ε_5 are the residuals defined by $\varepsilon_1(t_q)=$

$$\left(1+\sum_{j=1}^{M} b_j t_q^j\right)\left(\sum_{i=0}^{N} i a_i t_q^{i-1}\right)- \left(\sum_{i=0}^{N} a_i t_q^i\right)\left(\sum_{j=1}^{M} j b_j t_q^{j-1}\right)-X_1(t_q)\left(1+\sum_{j=1}^{M} b_j t_q^j\right) \quad (6)$$

$$\varepsilon_2(t_q)= \left(1+\sum_{j=1}^{M} d_j t_q^j\right)\left(\sum_{i=0}^{N} i c_i t_q^{i-1}\right)- \left(\sum_{i=0}^{N} c_i t_q^i\right)\left(\sum_{j=1}^{M} j d_j t_q^{j-1}\right)-X_2(t_q)\left(1+\sum_{j=1}^{M} d_j t_q^j\right) \quad (7)$$

$$\varepsilon_3(t_q)= \left(1+\sum_{j=1}^{M} f_j t_q^j\right)\left(\sum_{i=0}^{N} i e_i t_q^{i-1}\right)- \left(\sum_{i=0}^{N} e_i t_q^i\right)\left(\sum_{j=1}^{M} j f_j t_q^{j-1}\right)-X_3(t_q)\left(1+\sum_{j=1}^{M} f_j t_q^j\right) \quad (8)$$

$$\left(1+\sum_{j=1}^{M} h_j t_q^j\right)\left(\sum_{i=0}^{N} i g_i t_q^{i-1}\right)- \left(\sum_{i=0}^{N} g_i t_q^i\right)\left(\sum_{j=1}^{M} j h_j t_q^{j-1}\right)-X_4(t_q)\left(1+\sum_{j=1}^{M} h_j t_q^j\right) \quad (9)$$

$$\varepsilon_5(t_q)= \left(1+\sum_{j=1}^{M} l_j t_q^j\right)\left(\sum_{i=0}^{N} i k_i t_q^{i-1}\right)- \left(\sum_{i=0}^{N} k_i t_q^i\right)\left(\sum_{j=1}^{M} j l_j t_q^{j-1}\right)-X_5(t_q)\left(1+\sum_{j=1}^{M} l_j t_q^j\right) \quad (10)$$

By solving system (5) having $5q_{max}$ nonlinear simultaneous equations the problem reduces to finding 5(M+N) coefficients of Padé approximants.

3.2. Problem Constraints

The equality constraints of the model are considered as stated in the system (4):

$$h_1(t)=P(t)-P_0=0 \quad (11)$$
$$h_2(t)=L(t)-L_0=0 \quad (12)$$
$$h_3(t)=S(t)-S_0=0 \quad (13)$$
$$h_4(t)=Q(t)-Q_0=0 \quad (14)$$
$$h_5(t)=R(t)-R_0=0 \quad (15)$$

The inequality constraints (16) to (20) are related to positivity:

$$g_{1q}=\frac{\sum_{i=0}^{N} a_i t_q^i}{1+\sum_{j=1}^{M} b_j t_q^j}\geq 0, \quad (16)$$

$$g_{2q}=\frac{\sum_{i=0}^{N} c_i t_q^i}{1+\sum_{j=1}^{M} d_j t_q^j}\geq 0, \quad (17)$$

$$g_{3q}=\frac{\sum_{i=0}^{N} e_i t_q^i}{1+\sum_{j=1}^{M} f_j t_q^j}\geq 0, \quad (18)$$

$$g_{4q}=\frac{\sum_{i=0}^{N} g_i t_q^i}{1+\sum_{j=1}^{M} h_j t_q^j}\geq 0, \quad (19)$$

$$g_{5q}=\frac{\sum_{i=0}^{N} k_i t_q^i}{1+\sum_{j=1}^{M} l_j t_q^j}\geq 0, \quad (20)$$

whereas (21) incorporates the bounded-ness of the numerical solution:

$$g_{1q}+g_{2q}+g_{3q}+g_{4q}+g_{5q}\leq 1 \quad (21)$$

3.3. Objective Function

Suppose that:

$$\boldsymbol{x}=\begin{pmatrix}a_1,a_2,\cdots,a_M,b_1,b_2,\cdots,b_N,c_1,c_2,\cdots,c_M,d_1,d_2,\cdots,d_N,e_1,e_2,\cdots,e_M,\\ f_1,f_2,\cdots,f_N,g_1,g_2,\cdots,g_M,h_1,h_2,\cdots,h_N,k_1,k_2,\cdots,k_M,l_1,l_2,\cdots,l_N\end{pmatrix}^t$$

$\mathbb{R}^{5(M+N)}$, by converting the system (5) into minimization problem as:

$$Minimize\ \phi(\boldsymbol{x})=\frac{1}{5}\sum_{z=1}^{5}\sum_{q=0}^{q_{max}}\left[\varepsilon_z(t_q)\right] \quad (22)$$

3.4. Penalty Function

In penalty function, a large positive number depending on the degree of violation of constraints is added to the objective function. In the following relation, the objective function presented by $\psi(x)$, and the penalty function is presented by $\zeta(x)$ describes penalized function $\varphi(x)$ which was unconstrained defined as follows:

$$\varphi(x)=\begin{cases}\psi(x)+\zeta(x) & \text{if } \boldsymbol{x} \text{ is infeasible}\\ \psi(x) & \text{if } \boldsymbol{x} \text{ is feasible}\end{cases}$$

Here $\zeta(x)\geq 0$ is used for minimization problem and $\zeta(x)\leq 0$ for a maximization problem. By using the following penalty function unconstrained optimization model is obtained as:

$$\zeta(\boldsymbol{x})=\sum_{q=1}^{q_{max}} P_q\times max\left\{\begin{matrix}0,(h_1)^2,(h_2)^2,(h_3)^2,(h_4)^2,(h_5)^2,\\ -g_{1q},-g_{2q},-g_{3q},-g_{4q},-g_{5q},\sum_{s=1}^{5} g_{sq}-1\end{matrix}\right\}$$

Here scalar Pq is a large positive real number of q^{th} discrete-time step acting as a penalty factor then the unconstrained objective function is:

Minimize $\varpi(x)=\phi(x)+\zeta(x)$ (23)

3.5. Optimization process with Differential Evolution (DE)

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 randomly chosen starting points. In this original population, the preset bound parameter describes the domain from which the *Np* 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, u_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 [28],[29].

The following steps are involved to optimize objective function (23) through *EPA* scheme as:

Step 1. Generate population randomly, the population of K solutions $x_j \in \mathbb{R}^{5(M+N)}; 1 \leq j \leq K$.

Step 2. Evaluate the value $\varpi_j = \varpi(\mathbf{x_j})$ of each solution. Collect the best solution with the minimum value of the objective function. Initially set T = 0.

Step 3. Set T = T + 1.

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

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

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

Step 6. If $\varpi(y) < \varpi_j$ then $x_j \leftarrow y$, otherwise discard y.

Step 7. The best solution must be updated.

Step 8. If T > number of iterations, finish with the best solution, otherwise repeat the entire process from step 3.

4. NUMERICAL RESULTS AND DISCUSSION

Set parameters of *DE* algorithm for numerical illustrations:

$$N=50;\ F=0.55;CR=0.91$$
$$\textit{and maximum iterations} = 2000.$$

The approximation order for Padé is set to (N , M) = (2 , 2). The q_{max} the parameter is set to 2000. The penalty factor for all q is set to $L_q = 10^{10}$. The optimized smoking model parameters are shown in Table 1. 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 smoking model was provided. To notice the sound effects of the *EPA* algorithm on potential smokers, occasional smokers, heavy smokers, temporary quitters, and smokers who quit permanently population, comparison with *NSFD* having the property of uniqueness and positivity represent in Figure 1-5. Figures show convergence solution with the relationship between the different population compartments for diseases free equilibrium by using *EPA* scheme and *NSFD* scheme, here it can be easily observed that the results *EPA* scheme are more reliable and better convergence as with numerical scheme. In Figure 6 represents the impact of population compartments for diseases free equilibrium by using *EPA*. The results of different simulations through *EPA* are represented in Figure 7.

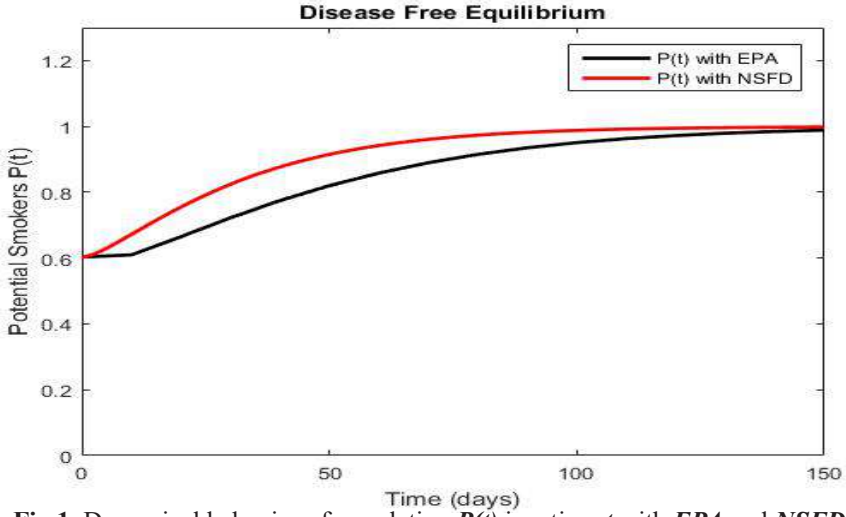

Fig 1. Dynamical behavior of population ***P(t)*** in a time ***t*** with ***EPA*** and ***NSFD***

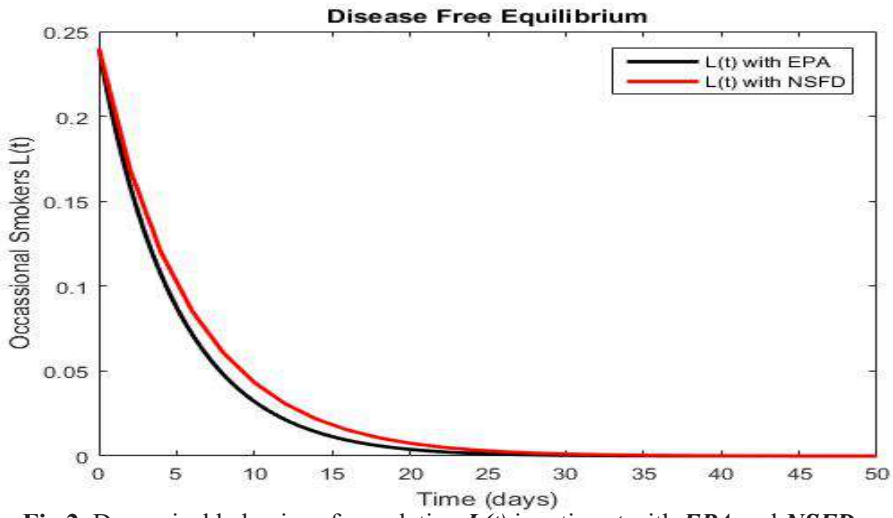

Fig 2. Dynamical behavior of population ***L(t)*** in a time ***t*** with ***EPA*** and ***NSFD***

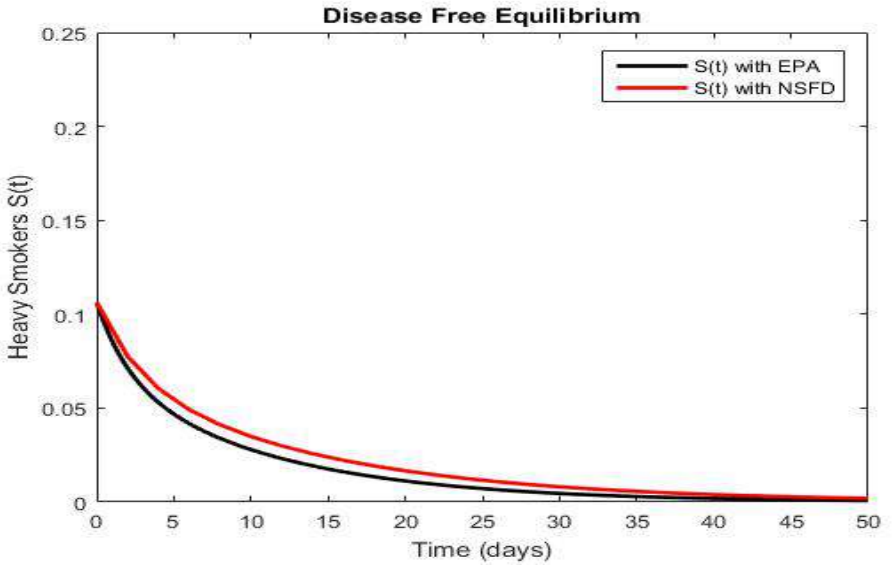

Fig 3. Dynamical behavior of population ***L(t)*** in a time ***t*** with ***EPA*** and ***NSFD***

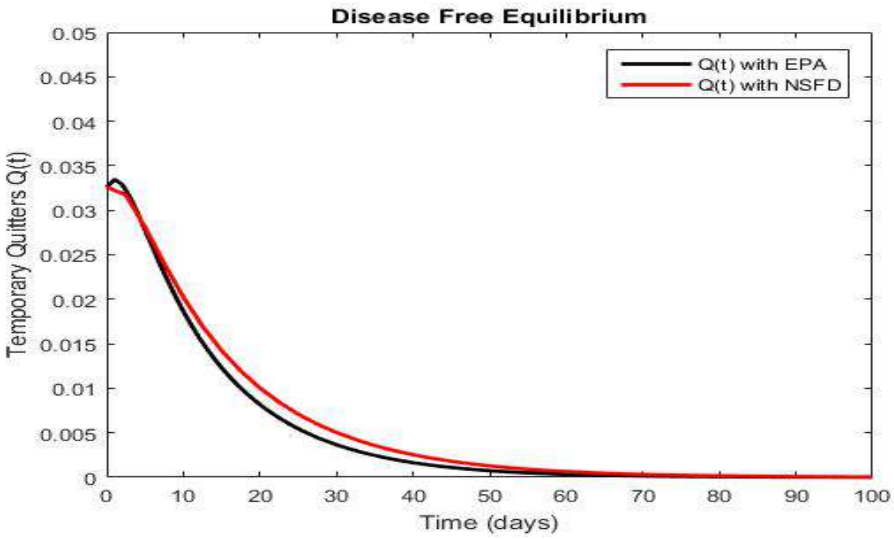

Fig 4. Dynamical behavior of population ***Q(t)*** in a time ***t*** with ***EPA*** and ***NSFD***

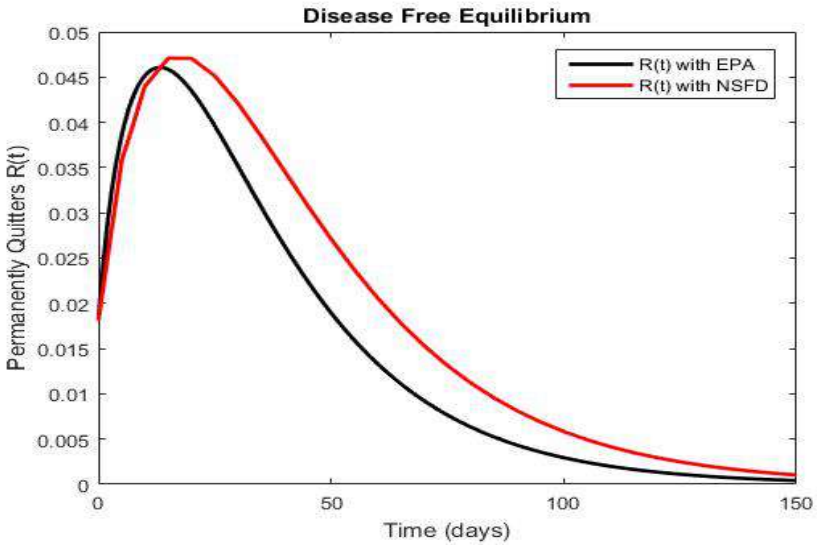

Fig 5. Dynamical behavior of population ***R(t)*** in a time ***t*** with ***EPA*** and ***NSFD***

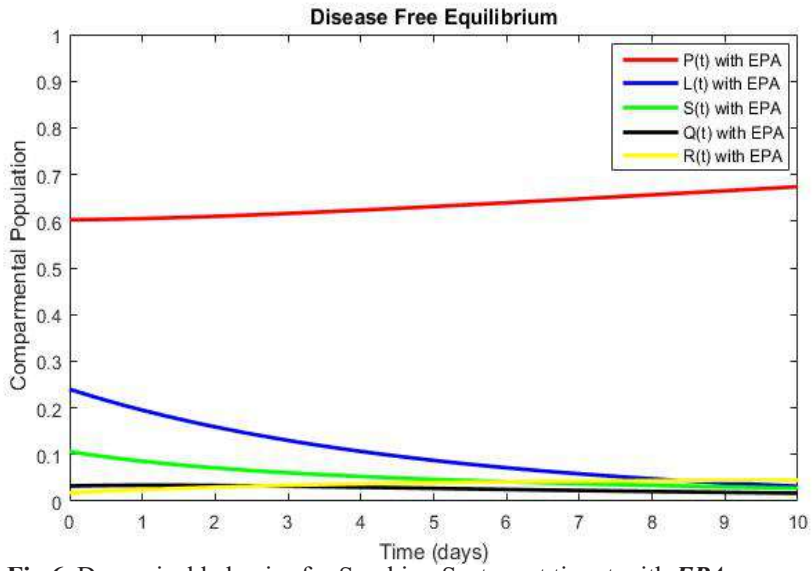

Fig 6. Dynamical behavior for Smoking System at time t with ***EPA***

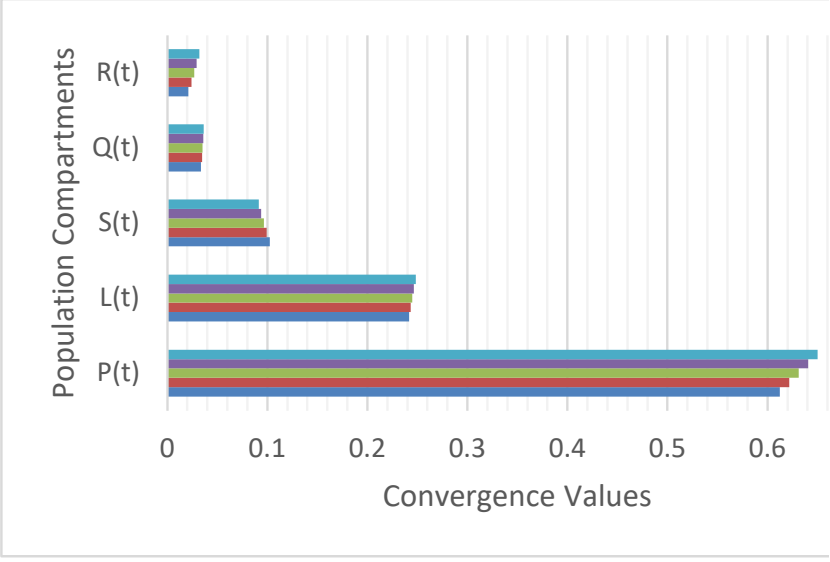

Fig 7. Results of different simulations through ***EPA***

5. CONCLUSIONS

This work proposes an evolutionary Padé approximation scheme for the solution of a nonlinear epidemiological smoking model. The *EPA* scheme efficiently applies to the highly nonlinear smoking model; this scheme produces excellent approximations of state variables that are highly accurate to the governing equations. Initial conditions are converted into problem constraints and then the constraint problem is converted into an unconstraint problem by using the penalty function. The *EPA* scheme has provided a convergence solution regarding the relationship among the different population compartments for diseases free equilibrium, it has been observed that the results *EPA* scheme are more reliable and significant when a comparison is drawn with *NSFD* numerical scheme. Finally, we have presented the numerical simulation and verified all the analytical results numerically by using *EPA* to reduce the infected rates very fast for disease-free equilibria. Further, in strong contrast to *NFSD*, this technique has eliminated the need to provide step size and control the spreading of smoking in the community.

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