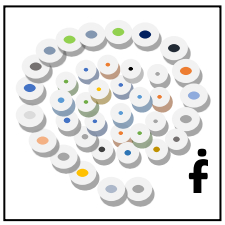

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Modeling and numerical analysis of saturation incidence and saturated treatment of dental caries infection

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Abstract

Dental caries are holes in teeth that form when acid in the mouth erodes tooth enamel. In this study, a SEIR model of dental caries infection is constructed with saturation incidence rate and saturated treatment function and we analyze the numerical behavior of the basic reproduction number and stability. Non-standard finite differences (NSFD) scheme was constructed for the SEIR model of dental caries infection and were compared with the Runge Kutta fourth order (RK-4) scheme and Forward Euler (FD) scheme. The NSFD scheme preserves positivity property and also proved to be an effective and efficient scheme for numerical solution of epidemic models. The dental caries (delay) free equilibrium and endemic equilibrium were obtained. Results of the numerical testing reveals that RK-4 scheme and forward Euler (FD) scheme is conditionally convergent and may loss some important properties of continuous system for certain values of step size h. Also, the numerical result further discloses that if medical resources are maximized and treatment not delayed, dental caries infection dynamics will decrease with transient time and the infection will eventually be eradicated from the population.

Keywords: SEIR model; NSFD scheme; Runge Kutta rk-4 scheme; forward Euler (fd) scheme; dental caries infection; positivity

1 Introduction

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Dental caries is a bacterial infection and bacteria must have the proper environment to multiply and thrive. Until teeth are present in a baby's mouth, the bacteria proliferate. Once teeth erupt, however, biofilm forms and adheres to the tooth surface. This matrix (Plaque) increases in amount unless disrupted daily. If allowed to remain, and other cofactors are present, dental disease is likely to result

$$Carbohydrate + Plaque = Acid + Tooth = Decay.$$

The bacteria within the plaque are able to ferment the sugars and carbohydrates that they contact. They form lactic acid, which can cause demineralization of the enamel of the tooth. Following exposure to sugar (the breakdown of carbohydrate) for 20 minutes, the enamel of the tooth continues to be attacked by acid. Each repeated snack leads to another 20 minutes of acid formation that can quickly weaken the tooth surface [1]. Once teeth have erupted into the mouth, there are surfaces that are more likely to develop decay at an earlier stage than others. The area along the gum line tends to collect plaque easily. This is where decalcification begins. The pits and fissures on molars tend to have a thinner layer of enamel and are prone to collect food and plaque. These are common sites for decay to occur. Plaque also tends to remain between teeth where the tooth brush cannot reach it. These areas then become susceptible to decay if the plaque is not removed by flossing. Biofilm does not form evenly on all teeth, thus, some teeth are more vulnerable than others to decay [24]. If decay is not treated at an early stage, it is likely that it will extend from the hard outer surface of the tooth to the main living tissue known as the pulp.

The etiological agents of dental caries are many and may be grouped into the exciting or instigating and the predisposing factors. The predisposing factors are diet, general health, oral hygiene, heredity and age, which diet has proven to be the leading contributing factor. The exciting factor is now known to be an acid producing organism [16]. Hahn and Liewehr [14] affirmed that the dental pulp is equipped to mount an innate response to invading caries bacteria which can theoretically slow down bacteria invasion. However the unique location of caries bacteria seems to prevent their being killed or eliminated by phagocytes. Instead, persistent infection leads to the activation of adaptive immunity and overwhelming inflammation and resultant increased edema and intra pulpal pressure, which becomes detrimental to the pulp encase in a low compliance environment. On the other hand, clinical experience indicates that inflamed pulps can recover if the majority of the antigens are removed early enough [16]

Farges et al [11] in their work confirmed that the crowns of the erupted human teeth are covered by symbiotic microbial communities mainly composed of Gram-positive saprophytic bacteria which are normally harmless to the tooth. These communities adhere as biofilms to the highly demineralized enamel that constitutes a barrier which is impermeable to microorganisms and protects the underlying mineralized dentin and the loose connective tissue situated at the centre of the tooth, the dental pulp. However, when placed in a sugar rich environment, specific bacteria populations from these communities release acids that progressively demineralize enamel [15, 10]. This leads to the appearance of a carious lesion characterized by a cavity within which "cariogenic" bacteria proliferate and release additional acids that progressively deepen the lesion. When the enamel barrier is disrupted, dentin becomes degraded by Gram-positive bacteria, including streptococci, lactobacilli and actinomyces that largely dominate the dentin caries microflora [23].

The proliferation and metabolic activity of these microorganisms lead to release of bacterial components into the dentinal tubules and their diffusion towards the peripheral pulp. Dentin

demineralization may also enable the release of bacterial components by host cells at the dentin matrix [6]. Recognition of bacterial components by host cells at the dentin –pulp interface triggers host protective events including antibacterial, immune and inflammatory responses [11]. These events may eliminate early stage bacterial infection and block the route of its progression when accompanied by dentin formation at pulp-dentin interface. Unchecked bacterial invasion results in irreversible chronic pulp inflammation. Subsequently, pulp necrosis, infection of the root canal system and periapical disease may occur [23, 18].

Dental caries management depends on the extent of dental caries and can range from the insertion of a restorative material, endodontic treatment, to tooth extraction [7]. Reversible pulpitis treatment with pulp protection and restorative materials is usually sufficient. Irreversible pulpitis treatment involves a root canal and direct or indirect restorations according to the degree of tooth remnant. There is insufficient evidence to recommend antibiotics [3]. Pulp necrosis is also treated with a root canal or tooth extraction as the last resource. A periapical abscess can complicate pulpitis. A periapical abscess is treatable with endodontic treatment and if they have migrated to the mucosa surface and incision and drainage can also be performed. Periapical abscess complicated by systemic symptoms, cellulitis, or in immunocompromised patients should receive antibiotics in addition to drainage [31, 17].

Gingivitis is a reversible condition that can be treated with professional plaque removal. The use of chlorhexidine rinses is suggested only as an adjuvant to mechanical treatment and for a short period of time given its side effects and complications [12]. Periodontitis may be treated with scaling and may require antibiotics in specific cases but not routinely. Antibiotic therapy for dental infections is necessary for systemic symptoms, fascial space infections, and infections that spread to the bony cortex and surrounding soft tissue [25]. Gram-negative organisms, facultative anaerobes, and strict anaerobes are common organisms found in dental infections, with anaerobes outnumbering aerobic bacteria by a factor of three [8]. Good oral hygiene like regular brushing, flossing and dental cleanings with a dentist can prevent and reverse caries. Using fluoride is a tried and tested way to prevent caries (decay) and cavities. High fluoride tooth paste or direct fluoride application helps remineralize the tooth enamel and prevent caries (decay) and cavities. It usually takes 12 – 24 hours for enough plaque buildup to support bacteria. By brushing and flossing at least once a day, one can remove most of this buildup, with twice a year dental cleaning to remove hard reach plaque that may have been missed.

Ndii et al, [30] constructed nonstandard finite difference scheme (NSFDS) for a water related disease mathematical model. They compared the numerical solutions of NSFDs, Eulers method and MATLAB's ode 45. It is shown numerically that the resulting discrete model preserves essential properties of the continuous model such as positivity and stability. Ashraf and Ahmad [5] in their SEIR Measles model used to study the integrating vaccination as control strategy, constructed an unconditional convergent nonstandard finite difference (NSFD) scheme and it preserve the positivity of all values of h. Also many researchers have compared different numerical schemes with the nonstandard finite difference (NSFD) scheme [21, 4].

The saturated incidence rate tend to a saturated level when I get large and βI measures the infection force when the disease is entering a fully susceptible population while $\frac{1}{1+\alpha I}$ measures the behavioral change of susceptible individuals when their number increased [33, 9] and many other epidemic model have been making use of saturated incidence rate [33, 9, 19, 32, 26, 22, 2]. Abdul – Satar and Naji [1] incorporated saturated recovery in their model while Naji and Abdulateef [29]

stated in their model that the treatment for the infected individual follows the saturated treatment function.

2 Model formulation

Many researchers applied many numerical techniques to solve many mathematical models. In this paper we are interested in the in the numerical solution of dental caries infection using SEIR epidemic model with saturation incidence rate and saturated treatment function and we shall apply three different numerical schemes such as Runge Kutta RK-4 method, Forward Euler (FD) scheme and Non Standard Finite Difference (NSFD) scheme. The rationale of NSFD scheme is to improve general finite difference scheme by including some of the exact forms and testing would determine which of the scheme is better than the other.

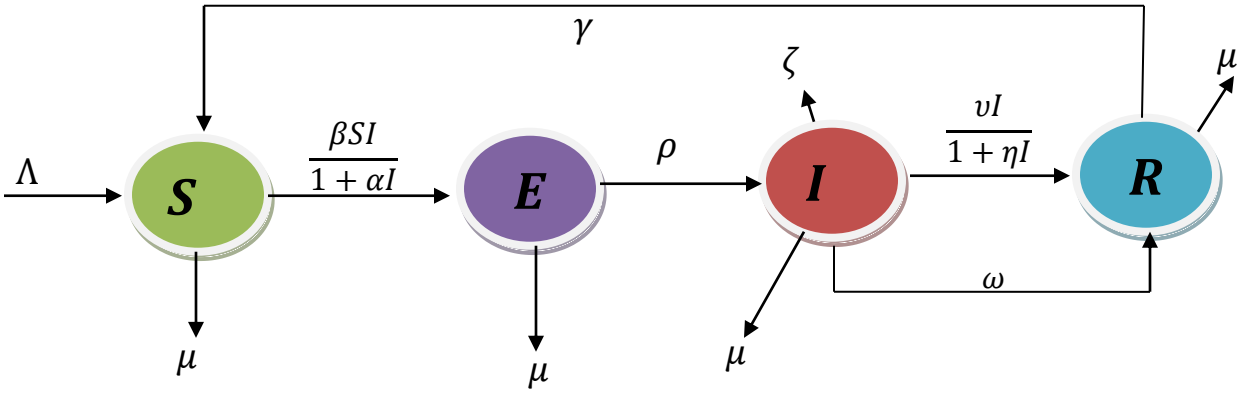

Figure 1: The model flow diagram

$$\left.\begin{aligned}
\frac{dS(t)}{dt} &= \Lambda + \gamma \mathrm{R}(t) - \left(\mu + \frac{\beta I(t)}{1+\alpha I(t)}\right) S(t) \\
\frac{dE(t)}{dt} &= \frac{\beta I(t) S(t)}{1+\alpha I(t)} - (\mu+\rho) E(t) \\
\frac{dI(t)}{dt} &= \rho E(t) - \left(\mu + \zeta + \omega + \frac{v}{1+\eta I}\right) I(t) \\
\frac{dR(t)}{dt} &= \left(\omega + \frac{v}{1+\eta I(t)}\right) I(t) - (\mu+\gamma) R(t)
\end{aligned}\right\} \qquad (1)$$

Description of Variables and Parameters
Variables

$S(t)$= Susceptible individuals at time t.
$E(t)$= Exposed individuals yet to be infectious at time t.
$I(t)$= Infectious individuals at time t.
$R(t)$= Treated and recovered individuals at time t.

Parameters
β= Disease transmission coefficient (i.e., effective contact rate).
Λ= Recruitment rate of the population.
μ= Natural death rate of the population and cut across all the compartments.
γ = The rate at which treated and recovered individuals move back to susceptible class.
α = The parameter that measures the inhibitory effects.
ρ=The rate at which the exposed individuals become infectious.
ω= recovery rate of infected individuals due to immune response.
η= The saturated factor that measures the effect of the infected being delayed for treatment.
υ= Maximum medical resources supplied per unit time.
ζ= The death rate due to the infection.

The most significant and efficient method to prevent and control the spread of dental caries (decay) is through treatment. In conventional epidemic models, the treatment rate of the infection is presumed to be proportional to the number of the infective individuals, which is applicable in dental caries (decay), but in general, the recovery rate depends on the medical resources, such as drugs, hospital personnel's, hospital equipments and efficiency of the treatment. Since most communities or countries have limited capacity for treatment of dental caries (decay), it is necessary to implement a suitable treatment function. Motivated by these facts, and to better understand the effects of the spread of dental caries (decay) with a successful and effective treatment, we proposed a model with saturation incidence rate and saturated treatment function.

The paper is systemized in five sections as follows; in section 2, we have the analysis of the model in which the two eqiulibria were discussed with the basic reproduction number. In section 3, we solved the proposed model using three (3) different methods. The numerical experiments for the three numerical schemes were performed in section 4 and finally, section was the conclusion.

3 Analysis of the model

From system (1) it follows that

$$N(t) = S(t) + E(t) + I(t) + R(t) \tag{2}$$

$$N(t) \leq \Lambda - \mu\big(S(t) + E(t) + I(t) + R(t)\big) \tag{3}$$
$$\leq \Lambda - \mu(S + E + I + R).$$

Then,

$$\lim_{t\to\infty} \sup(S + E + I + R), \leq \frac{\Lambda}{\mu}.$$

Thus, the feasible region for system (1) is

$$\Omega = \left\{\big(S(t) + E(t) + I(t) + R(t)\big): S + E + I + R \leq \frac{\Lambda}{\mu}, S \geq 0, E \geq 0, I \geq 0, R \geq 0\right\} \quad (4)$$

It can be verified that the region Ω is positively invariant with respect to system (1). Thus we have that the basic reproduction number which is the average number of secondary infection activated by the initial infection is given as

$$R_0 = \frac{\beta\Lambda\rho}{\mu(\mu+\rho)(\mu+\zeta+\omega+\upsilon)} \quad (5)$$

The two equilibria of the system are the dental caries (decay) free equilibrium (DCFE) and the dental caries (decay) endemic equilibrium (DCEE) points. The dental caries (decay) free equilibrium (DCFE) point is given by

$$\mathrm{T}^0 = (S^0, E^0, I^0, R^0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right) \quad (6)$$

while the endemic equilibrium (DCEE) point is given as

$$\mathrm{T}^* = (S^*, E^*, I^*, R^*) \quad (7)$$

Where

$$S^* = \frac{[\Lambda(1+\eta I^*)(\mu+\gamma)+\gamma(\omega(1+\eta I^*)+\upsilon)I^*](1+\alpha I^*)}{(1+\eta I^*)(\mu+\gamma)[\mu(1+\alpha I^*)+\beta I^*]},$$

$$E^* = \frac{\beta I^*[\Lambda(1+\eta I^*)(\mu+\gamma)+\gamma(\omega(1+\eta I^*)+\upsilon)I^*]}{(1+\eta I^*)(\mu+\gamma)[\mu(1+\alpha I^*)+\beta I^*]},$$

$$R^* = \frac{(\omega(1+\eta I^*)+\upsilon)I^*}{(1+\eta I^*)(\mu+\gamma)}$$

$$\frac{\rho\big[\beta I^*[\Lambda(1+\eta I^*)(\mu+\gamma)+\gamma(\omega(1+\eta I^*)+\upsilon)I^*]\big]}{(1+\eta I^*)(\mu+\gamma)[\mu(1+\alpha I^*)+\beta I^*]} = \frac{[(\mu+\zeta+\omega)(1+\eta I^*)+\upsilon]I^*}{(1+\eta I^*)}$$

$$\begin{aligned}\Longrightarrow \beta\Lambda\rho(1+\eta I^*)(\mu+\gamma) + \beta\rho\gamma\omega(1+\eta I^*)I^* + \beta\rho\gamma\upsilon I^* \\ = [\mu(\mu+\rho)(\mu+\gamma)(1+\alpha I^*) \\ + \beta(\mu+\rho)(\mu+\gamma)I^*]\big[[(\mu+\zeta+\omega)(1+\eta I^*)+\upsilon]\big]\end{aligned}$$

which after expansion yields

$$\begin{aligned}&[\beta\rho\gamma\omega\eta - \mu\alpha\eta(\mu+\rho)(\mu+\gamma)(\mu+\zeta+\omega) - \eta\beta(\mu+\rho)(\mu+\gamma)(\mu+\zeta+\omega)]I^{*2} \\ &\quad + [\beta\rho\gamma\omega + \beta\rho\gamma\upsilon + \beta\Lambda\rho\eta(\mu+\gamma) - \mu\eta(\mu+\rho)(\mu+\gamma)(\mu+\zeta+\omega) \\ &\quad - \mu\alpha(\mu+\rho)(\mu+\gamma)(\mu+\zeta+\omega) - \mu\upsilon\alpha(\mu+\rho)(\mu+\gamma) \\ &\quad - \beta(\mu+\rho)(\mu+\gamma)(\mu+\zeta+\omega) - \upsilon\beta(\mu+\rho)(\mu+\gamma)]I^* + [(\mu+\gamma)(R_0-1)] \\ &\quad = 0 \quad (8)\end{aligned}$$

This can be written as

$$k_2 I^{*2} + k_1 I^* + k_0 = 0 \quad (9)$$

$$\left.\begin{array}{c} k_2 = \beta\rho\gamma\omega\eta - \mu\alpha\eta(\mu+\rho)(\mu+\gamma)(\mu+\zeta+\omega) - \eta\beta(\mu+\rho)(\mu+\gamma)(\mu+\zeta+\omega) \\ k_1 = \beta\rho\gamma\omega + \beta\rho\gamma\upsilon + \beta\Lambda\rho\eta(\mu+\gamma) - \mu\eta(\mu+\rho)(\mu+\gamma)(\mu+\zeta+\omega) - \mu\alpha(\mu+\rho)(\mu+\gamma)(\mu+\zeta+\omega) \\ -\mu\upsilon\alpha(\mu+\rho)(\mu+\gamma) - \beta(\mu+\rho)(\mu+\gamma)(\mu+\zeta+\omega) - \upsilon\beta(\mu+\rho)(\mu+\gamma) \\ k_0 = (\mu+\gamma)(R_0-1) \end{array}\right\} \quad (10)$$

Thus, for the existence of the dental caries (decay) endemic equilibrium, equation (9) must have positive real roots. Therefore, we determine the sign of k_0 by expressing it as

$$k_0 = \beta\Lambda\rho(\mu+\gamma) - \mu(\mu+\rho)(\mu+\gamma)(\mu+\zeta+\omega+\upsilon), \quad (11)$$

since

$$k_0 = (\mu+\gamma)(R_0-1).$$

We show that at DCEE, $R_0 > 1$, then, we obtain

$$R_0 = \frac{\beta\Lambda\rho}{\mu(\mu+\rho)(\mu+\zeta+\omega+\upsilon)} > 1.$$

It can easily be seen that

$$\beta\Lambda\rho > \mu(\mu+\gamma)(\mu+\zeta+\omega+\upsilon)$$

Since system (9) is a quadratic equation with respect to I^*, it follows from (11) that k_0 is non positive $(k_0 < 0)$ whenever $R_0 < 1$. This proves that $k_0 > 0$ if and only if $R_0 > 1$. Thus, the number of positive real roots depends on the sign of equation (9). This can be analyzed using Descartes rule of sign of equation. Let

$$F(I^*) = k_2 I^{*2} + k_1 I^* + k_0$$

The different possibilities for the roots of $F(I^*)$ are tabulated in Table 1 below.

Table 1: Number of possible positive roots of $F(I^*)$ for $R_0 > 1$.

Cases	k_2	k_1	k_0	R_0	*No of sign change*	*No of positie roots*
1	+	+	−	R_0	1	1
2	+	−	−	R_0	1	1

Model system (1) has a unique endemic equilibrium point where one of the cases 1 and 2 in table 1 are satisfied.

4 Numerical modeling

We shall use three finite difference (FD) methods to solve model system (1) in this section. The three methods are Runge Kutta Method of Order 4 (RK4), Forward Euler (FD) Scheme and Non-Standard Finite Difference (NSFD) Method for numerical solution of system (1) and then we will compare our results with a Standard RK4 method.

4.1 Runge- Kutta Method of Order 4

Here, we shall develop the R-K 4 finite difference (FD) scheme for model system (1), thus;

$$\left.\begin{aligned} S_{n+1} - S_n &= h\left[\Lambda + \gamma R_n - \frac{\beta S_n I_n}{1+\alpha I_n} - \mu S_n\right] \\ E_{n+1} - E_n &= h\left[\frac{\beta S_n I_n}{1+\alpha I_n} - (\mu+\rho)E_n\right] \\ I_{n+1} - I_n &= h\left[\rho E_n - (\mu+\zeta+\omega)I_n - \frac{\upsilon I_n}{1+\eta I_n}\right] \\ R_{n+1} - R_n &= h\left[\omega I_n + \frac{\upsilon I_n}{1+\eta I_n} - (\mu+\gamma)R_n\right] \end{aligned}\right\} \qquad (12)$$

$$\left.\begin{aligned} S_{n+1} &= S_n + h\left[\Lambda + \gamma R_n - \frac{\beta S_n I_n}{1+\alpha I_n} - \mu S_n\right] \\ E_{n+1} &= E_n + h\left[\frac{\beta S_n I_n}{1+\alpha I_n} - (\mu+\rho)E_n\right] \\ I_{n+1} &= I_n + h\left[\rho E_n - (\mu+\zeta+\omega)I_n - \frac{\upsilon I_n}{1+\eta I_n}\right] \\ R_{n+1} &= R_n + h\left[\omega I_n + \frac{\upsilon I_n}{1+\eta I_n} - (\mu+\gamma)R_n\right] \end{aligned}\right\}$$

Finding $S_{n+1}, E_{n+1}, I_{n+1}$ *and* R_{n+1} requires four intermediate calculations such as

Step I

$$\left.\begin{aligned} U_1 &= h\left[\Lambda + \gamma R_n - \frac{\beta S_n I_n}{1+\alpha I_n} - \mu S_n\right] \\ X_1 &= h\left[\frac{\beta S_n I_n}{1+\alpha I_n} - (\mu+\rho)E_n\right] \\ Y_1 &= h\left[\rho E_n - (\mu+\zeta+\omega)I_n - \frac{\upsilon I_n}{1+\eta I_n}\right] \\ Z_1 &= h\left[\omega I_n + \frac{\upsilon I_n}{1+\eta I_n} - (\mu+\gamma)R_n\right] \end{aligned}\right\}$$

Step II

$$\left.\begin{aligned} U_2 &= h\left[\Lambda + \gamma(R_n) - \frac{\beta\left(S_n + \frac{U_1}{2}\right)\left(I_n + \frac{Y_1}{2}\right)}{1 + \alpha\left(I_n + \frac{Y_1}{2}\right)} - \mu\left(S_n + \frac{U_1}{2}\right)\right] \\ X_2 &= h\left[\frac{\beta\left(S_n + \frac{U_1}{2}\right)\left(I_n + \frac{Y_1}{2}\right)}{1 + \alpha\left(I_n + \frac{Y_1}{2}\right)} - (\mu + \rho)\left(E_n + \frac{X_1}{2}\right)\right] \\ Y_2 &= h\left[\rho\left(E_n + \frac{X_1}{2}\right) - (\mu + \zeta + \omega)\left(I_n + \frac{Y_1}{2}\right) - \frac{\upsilon\left(I_n + \frac{Y_1}{2}\right)}{1 + \eta\left(I_n + \frac{Y_1}{2}\right)}\right] \\ Z_2 &= h\left[\omega\left(I_n + \frac{Y_1}{2}\right) + \frac{\upsilon\left(I_n + \frac{Y_1}{2}\right)}{1 + \eta\left(I_n + \frac{Y_1}{2}\right)} - (\mu + \gamma)\left(R_n + \frac{Z_1}{2}\right)\right] \end{aligned}\right\}$$

Step III

$$\left.\begin{aligned} U_3 &= h\left[\Lambda + \gamma(R_n) - \frac{\beta\left(S_n + \frac{U_2}{2}\right)\left(I_n + \frac{Y_2}{2}\right)}{1 + \alpha\left(I_n + \frac{Y_2}{2}\right)} - \mu\left(S_n + \frac{U_2}{2}\right)\right] \\ X_3 &= h\left[\frac{\beta\left(S_n + \frac{U_2}{2}\right)\left(I_n + \frac{Y_2}{2}\right)}{1 + \alpha\left(I_n + \frac{Y_2}{2}\right)} - (\mu + \rho)\left(E_n + \frac{X_2}{2}\right)\right] \\ Y_3 &= h\left[\rho\left(E_n + \frac{X_2}{2}\right) - (\mu + \zeta + \omega)\left(I_n + \frac{Y_2}{2}\right) - \frac{\upsilon\left(I_n + \frac{Y_2}{2}\right)}{1 + \eta\left(I_n + \frac{Y_2}{2}\right)}\right] \\ Z_3 &= h\left[\omega\left(I_n + \frac{Y_2}{2}\right) + \frac{\upsilon\left(I_n + \frac{Y_2}{2}\right)}{1 + \eta\left(I_n + \frac{Y_2}{2}\right)} - (\mu + \gamma)\left(R_n + \frac{Z_2}{2}\right)\right] \end{aligned}\right\}$$

Step IV

$$\left.\begin{aligned} U_2 &= h\left[\Lambda + \gamma(R_n) - \frac{\beta(S_n + U_3)(I_n + Y_3)}{1 + \alpha(I_n + Y_3)} - \mu(S_n + U_3)\right] \\ X_1 &= h\left[\frac{\beta(S_n + U_3)(I_n + Y_3)}{1 + \alpha\left(I_n + \frac{Y_1}{2}\right)} - (\mu + \rho)(E_n + X_3)\right] \\ Y_1 &= h\left[\rho(E_n + X_3) - (\mu + \zeta + \omega)(I_n + Y_3) - \frac{\upsilon(I_n + Y_3)}{1 + \eta(I_n + Y_3)}\right] \\ Z_1 &= h\left[\omega(I_n + Y_3) + \frac{\upsilon(I_n + Y_3)}{1 + \eta(I_n + Y_3)} - (\mu + \gamma)(R_n + Z_3)\right] \end{aligned}\right\}$$

We shall observe that the incremental in the value of $S_{n+1}, E_{n+1}, I_{n+1}$ *and* R_{n+1} is then

$$\left.\begin{aligned} S_{n+1} &= S_n + \frac{1}{6}[U_1 + 2U_2 + 2U_3 + U_4] \\ E_{n+1} &= E_n + \frac{1}{6}[X_1 + 2X_2 + 2X_3 + X_4] \\ I_{n+1} &= I_n + \frac{1}{6}[Y_1 + 2Y_2 + 2Y_3 + Y_4] \\ R_{n+1} &= R_n + \frac{1}{6}[Z_1 + 2Z_2 + 2Z_3 + Z_4] \end{aligned}\right\} \quad (13)$$

4.2 Forward Euler (FD) Scheme

The Forward Euler Finite Difference Scheme were developed for model system (1) as follows:

$$\left.\begin{aligned} S_{n+1} &= S_n + h\left[\Lambda + \gamma R_n - \frac{\beta S_n I_n}{1 + \alpha I_n} - \mu S_n\right] \\ E_{n+1} &= E_n + h\left[\frac{\beta S_n I_n}{1 + \alpha I_n} - (\mu + \rho)E_n\right] \\ I_{n+1} &= I_n + h\left[\rho E_n - (\mu + \zeta + \omega)I_n - \frac{\upsilon I_n}{1 + \eta I_n}\right] \\ R_{n+1} &= R_n + h\left[\omega I_n + \frac{\upsilon I_n}{1 + \eta I_n} - (\mu + \gamma)R_n\right] \end{aligned}\right\} \quad (14)$$

4.3 Non Standard Finite Difference Scheme (NSFD)

In this section we design the non standard finite difference (NSFD) scheme that replicates the dynamics of continuous model system (1). Non standard finite difference scheme was proposed by Micken's in 1989 [21] and now is 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 [13, 20, 27, 28] when comparing all others well known numerical method of state variable of the system under study. NSFD scheme maintains dynamic

consistency as well as numerical stability in terms of initial restriction with irregular step length. The model system (1) is developed according to the laws given by Mickens

$$\frac{S_{n+1}-S_n}{h}=\Lambda+\gamma R_n-\frac{\beta S_{n+1}I_n}{1+\alpha I_n}-\mu S_{n+1}$$

$$S_{n+1}=S_n+h\Lambda+h\gamma R_n-\frac{h\beta S_{n+1}I_n}{1+\alpha I_n}-h\mu S_{n+1}$$

$$S_{n+1}+\frac{h\beta S_{n+1}I_n}{1+\alpha I_n}+h\mu S_{n+1}=S_n+h\Lambda+h\gamma R_n$$

$$S_{n+1}\left[1+\frac{h\beta I_n}{1+\alpha I_n}+h\mu\right]=S_n+h\Lambda+h\gamma R_n$$

$$S_{n+1}=\frac{S_n+h\Lambda+h\gamma R_n}{1+\frac{h\beta I_n}{1+\alpha I_n}+h\mu}$$

$$\frac{E_{n+1}-E_n}{h}=\frac{\beta S_n I_n}{1+\alpha I_n}-(\mu+\rho)E_{n+1}$$

$$E_{n+1}+h(\mu+\rho)E_{n+1}=E_n+\frac{h\beta S_n I_n}{1+\alpha I_n}$$

$$E_{n+1}[1+h(\mu+\rho)]=E_n+\frac{h\beta S_n I_n}{1+\alpha I_n}$$

$$E_{n+1}=\frac{E_n+\frac{h\beta S_n I_n}{1+\alpha I_n}}{1+h(\mu+\rho)}$$

$$\frac{I_{n+1}-I_n}{h}=\rho E_n-(\mu+\zeta+\omega)I_{n+1}-\frac{\upsilon I_{n+1}}{1+\eta I_n}$$

$$I_{n+1}+h(\mu+\zeta+\omega)I_{n+1}+\frac{h\upsilon I_{n+1}}{1+\eta I_n}=I_n+h\,\rho E_n$$

$$I_{n+1}=\left[1+h(\mu+\zeta+\omega)I_{n+1}+\frac{h\upsilon I_{n+1}}{1+\eta I_n}\right]=I_n+h\,\rho E_n$$

$$I_{n+1}=\frac{I_n+h\,\rho E_n}{1+h(\mu+\zeta+\omega)I_{n+1}+\frac{h\upsilon I_{n+1}}{1+\eta I_n}}$$

$$\frac{R_{n+1}-R_n}{h}=\omega I_n+\frac{\upsilon I_n}{1+\eta I_n}-(\mu+\gamma)R_{n+1}$$

$$R_{n+1}+h(\mu+\gamma)R_{n+1}=R_n+h\omega I_n+\frac{h\upsilon I_n}{1+\eta I_n}$$

$$R_{n+1}[1+h(\mu+\gamma)]=R_n+h\omega I_n+\frac{h\upsilon I_n}{1+\eta I_n}$$

$$R_{n+1}=\frac{R_n+h\omega I_n+\frac{h\upsilon I_n}{1+\eta I_n}}{1+h(\mu+\gamma)}$$

$$\left.\begin{aligned} S_{n+1} &= \frac{S_n + h\Lambda + h\gamma R_n}{1 + \frac{h\beta I_n}{1+\alpha I_n} + h\mu} \\ E_{n+1} &= \frac{E_n + \frac{h\beta S_n I_n}{1+\alpha I_n}}{1 + h(\mu + \rho)} \\ I_{n+1} &= \frac{I_n + h\,\rho E_n}{1 + h(\mu + \zeta + \omega) I_{n+1} + \frac{h\upsilon I_{n+1}}{1+\eta I_n}} \\ R_{n+1} &= \frac{R_n + h\omega I_n + \frac{h\upsilon I_n}{1+\eta I_n}}{1 + h(\mu + \gamma)} \end{aligned}\right\} \quad (15)$$

To obtain success in NSFD system (15) should be sequentially computed since the value of S_{n+1} will be applied in calculating E_{n+1}, E_{n+1} is used to calculate the value of I_{n+1} which in turn is then used in computing R_{n+1}. The process is repeated until we finally approaches the time of interest.

Non-Negativity of the Solution

The model is centered on the transmission dynamics of dental caries in human population, therefore, the proposed numerical scheme cannot produce negative value and we assume that the initial conditions are non negative such that

$$S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0 \quad (16)$$

These variables have approximate values which also are non-negative due to the following

$$S_n \geq 0, E_n \geq 0, I_n \geq 0, R_n \geq 0$$

We shall observe that for time steps greater than zero, that is, $h \geq 0$and $S_n, E_n, I_n, R_n \geq 0$, numerators and denominators are nonnegative and hence $S_{n+1}, E_{n+1}, I_{n+1}, R_{n+1} \geq 0$.

4.3.1 Convergence analysis of the proposed NSFD scheme

The NSFD scheme converges to the equilibrium point of the absolute of the eigenvalues is less than one. At this point, we shall examine the convergence of the proposed non standard finite difference scheme (NSFD) using

$$\left.\begin{aligned} L &= \frac{S + h\Lambda + h\gamma R}{1 + h\mu + \frac{h\beta I}{1+\alpha I}} \\ P &= \frac{E + \frac{h\beta SI}{1+\alpha I}}{1 + h(\mu + \rho)} \\ Q &= \frac{I + h\rho E}{1 + h(\mu + \zeta + \omega) + \frac{\upsilon h}{1+\eta I}} \\ T &= \frac{R + h\left(\omega + \frac{\upsilon}{1+\eta I}\right) I}{1 + h(\mu + \gamma)} \end{aligned}\right\}$$

Then

$$\left.\begin{aligned} L &= \frac{S + h\Lambda + h\gamma R}{1 + h\mu + \frac{h\beta I}{1+\alpha I}} \\ \frac{\partial L}{\partial S} &= \frac{1}{1 + h\mu + \frac{h\beta I}{1+\alpha I}} \\ \frac{\partial L}{\partial E} &= 0 \\ \frac{\partial L}{\partial I} &= -\frac{h\beta(S + h\Lambda + h\gamma R)}{(1+\alpha I)^2\left(1 + h\mu + \frac{h\beta I}{1+\alpha I}\right)^2} \\ \frac{\partial L}{\partial R} &= \frac{h\gamma}{1 + h\mu + \frac{h\beta I}{1+\alpha I}} \end{aligned}\right\}$$

Also,

$$\left.\begin{aligned} P &= \frac{E + \frac{h\beta SI}{1+\alpha I}}{1 + h(\mu + \rho)} \\ \frac{\partial P}{\partial S} &= \frac{h\beta I}{(1+\alpha I)\left(1 + h(\mu + \rho)\right)} \\ \frac{\partial P}{\partial E} &= \frac{1}{1 + h(\mu + \rho)} \\ \frac{\partial P}{\partial I} &= \frac{h\beta S}{(1+\alpha I)^2\left(1 + h(\mu + \rho)\right)} \\ \frac{\partial P}{\partial R} &= 0 \end{aligned}\right\}$$

Similarly

$$Q = \frac{I + h\rho E}{1 + h(\mu + \zeta + \omega) + \frac{\upsilon h}{1 + \eta I}}$$

$$\left.\begin{aligned} \frac{\partial Q}{\partial S} &= 0 \\ \frac{\partial Q}{\partial E} &= \frac{h\rho}{1 + h(\mu + \zeta + \omega) + \frac{\upsilon h}{1 + \eta I}} \\ \frac{\partial Q}{\partial I} &= \frac{1}{1 + h(\mu + \zeta + \omega) + \frac{\upsilon h}{1 + \eta I}}\left[1 + \frac{h\upsilon}{(1 + \eta I)^2\left(1 + h(\mu + \zeta + \omega) + \frac{\upsilon h}{1 + \eta I}\right)}\right][I - h\rho E] \\ \frac{\partial Q}{\partial R} &= 0 \end{aligned}\right\}$$

Finally

$$T = \frac{R + h\left(\omega + \frac{\upsilon}{1 + \eta I}\right) I}{1 + h(\mu + \gamma)}$$

$$\left.\begin{aligned} \frac{\partial T}{\partial S} &= 0 \\ \frac{\partial T}{\partial E} &= 0 \\ \frac{\partial T}{\partial I} &= \frac{h\omega + \frac{h\upsilon}{1 + \eta I}}{1 + h(\mu + \gamma)} \\ \frac{\partial T}{\partial R} &= \frac{1}{1 + h(\mu + \gamma)} \end{aligned}\right\}$$

$$J = \begin{bmatrix} \frac{\partial L}{\partial S} & \frac{\partial L}{\partial E} & \frac{\partial L}{\partial I} & \frac{\partial L}{\partial R} \\ \frac{\partial P}{\partial S} & \frac{\partial P}{\partial E} & \frac{\partial I}{\partial I} & \frac{\partial P}{\partial R} \\ \frac{\partial Q}{\partial S} & \frac{\partial Q}{\partial E} & \frac{\partial Q}{\partial I} & \frac{\partial Q}{\partial R} \\ \frac{\partial T}{\partial S} & \frac{\partial T}{\partial E} & \frac{\partial T}{\partial I} & \frac{\partial T}{\partial R} \end{bmatrix} \tag{17}$$

The eigenvalues of the matrix at dental caries free equilibrium $\mathrm{T}^0 = (S^0, E^0, I^0, R^0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$ gives

$$J(\mathrm{T}^0) = \begin{bmatrix} \frac{1}{1+h\mu} & 0 & -\frac{h\beta\left(\frac{\Lambda}{\mu}+\Lambda h\right)}{(1+h\mu)^2} & \frac{h\gamma}{1+h\mu} \\ 0 & \frac{1}{1+h(\mu+\rho)} & \frac{h\beta\Lambda}{\mu(1+h(\mu+\rho))} & 0 \\ 0 & \frac{h\rho}{1+h(\mu+\zeta+\omega+\nu)} & \frac{1}{1+h(\mu+\zeta+\omega+\nu)} & 0 \\ 0 & 0 & \frac{h(\omega+\nu)}{1+h(\mu+\gamma)} & \frac{1}{1+h(\mu+\gamma)} \end{bmatrix}$$

It is clear from the above matrix that the two eigenvalues are $\frac{1}{1+h\mu}, \frac{1}{1+h(\mu+\gamma)}$ which are less than unity. The remaining two eigenvalues can be found from the matrix below

$$M(\mathrm{T}^0) = \begin{bmatrix} \frac{1}{1+h(\mu+\rho)} & \frac{h\beta\Lambda}{\mu(1+h(\mu+\rho))} \\ \frac{h\rho}{1+h(\mu+\zeta+\omega+\upsilon)} & \frac{1}{1+h(\mu+\zeta+\omega+\upsilon)} \end{bmatrix}$$

The characteristic determinant is given as $M(\mathrm{T}^0) - \lambda I = 0$ and we obtain

$$\begin{vmatrix} \frac{1}{1+h(\mu+\rho)} - \lambda & \frac{h\beta\Lambda}{\mu(1+h(\mu+\rho))} \\ \frac{h\rho}{1+h(\mu+\zeta+\omega+\upsilon)} & \frac{1}{1+h(\mu+\zeta+\omega+\upsilon)} - \lambda \end{vmatrix} = 0$$

$$\left(\frac{1}{1+h(\mu+\rho)} - \lambda\right)\left(\frac{1}{1+h(\mu+\zeta+\omega+\upsilon)} - \lambda\right) - \left(\frac{h\beta\Lambda}{\mu(1+h(\mu+\rho))}\right)\left(\frac{h\rho}{1+h(\mu+\zeta+\omega+\upsilon)}\right) = 0$$

$$\lambda^2 - \frac{\lambda}{1+h(\mu+\rho)} - \frac{\lambda}{1+h(\mu+\zeta+\omega+\upsilon)} + \left(\frac{1}{1+h(\mu+\rho)}\right)\left(\frac{1}{1+h(\mu+\zeta+\omega+\upsilon)}\right) - \frac{h^2\beta\rho\Lambda}{\mu(1+h(\mu+\rho))(1+h(\mu+\zeta+\omega+\upsilon))} = 0$$

$$\lambda^2 - \left[\frac{2+h(\mu+\rho)+h(\mu+\zeta+\omega+\upsilon)}{(1+h(\mu+\rho))(1+h(\mu+\zeta+\omega+\upsilon))}\right]\lambda + \frac{\mu - h^2\beta\rho\Lambda}{\mu(1+h(\mu+\rho))(1+h(\mu+\zeta+\omega+\upsilon))} = 0 \quad (18)$$

Equation (18) can be rewritten as

$$\lambda^2 - \frac{A}{B}\lambda + \frac{C}{\mu B} = 0 \qquad (19)$$

where

$$A = 2 + h(\mu + \rho) + h(\mu + \zeta + \omega + \upsilon);$$
$$B = \big(1 + h(\mu + \rho)\big)\big(1 + h(\mu + \zeta + \omega + \upsilon)\big) \; and \; C = \mu - h^2\beta\rho\Lambda.$$

It is clear that the absolute of the eigenvalues is less than unity as demonstrated by the spectral radius of the Jacobian matrix $M(\mathrm{T}^0)$ for each time step "h" such that $0 < h < 1000$ shown in figure 2 and we may observe from this figure that the spectral radius remains less than unity for each time step. By implication, it means that all the eigenvalues of the Jacobian matrix at dental caries free equilibrium will be within a unit disc and this assures that the proposed NSFD scheme is convergent for each of the selected time step.

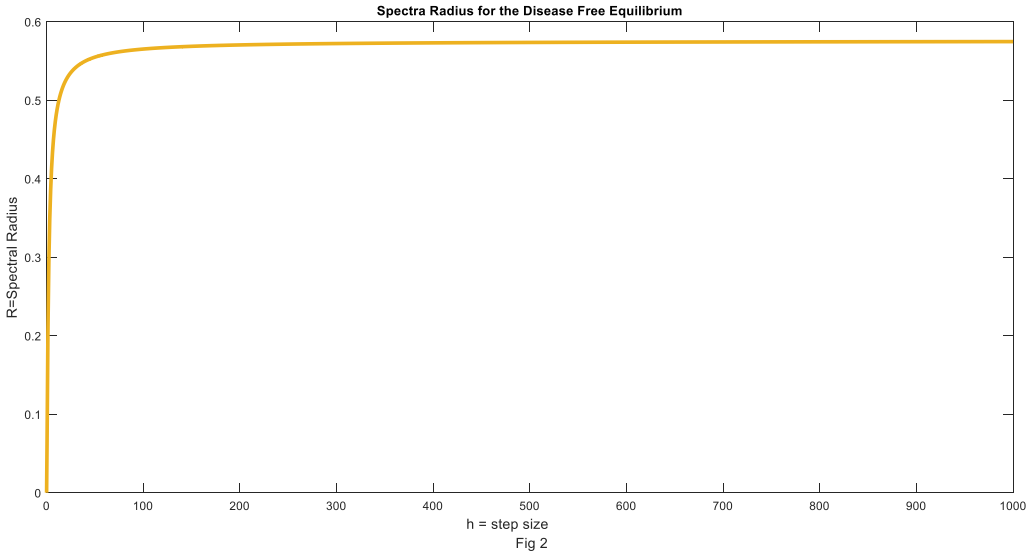

Figure 2: The graph of Spectral radius for the Dental Caries (decay) free equilibrium

Also, the eigenvalues of the Jacobian matrix $J(\mathrm{T}^*)$ at dental caries endemic equilibrium $\mathrm{T}^* = (\mathrm{S}^*, \mathrm{E}^*, \mathrm{I}^*, \mathrm{R}^*)$ will be given as

$$J(\mathrm{T}^*) = \begin{bmatrix} J_{11} & J_{12} & J_{13} & J_{14} \\ J_{21} & J_{22} & J_{23} & J_{24} \\ J_{31} & J_{32} & J_{33} & J_{34} \\ J_{41} & J_{42} & J_{43} & J_{44} \end{bmatrix}$$

$$J_{11} = \frac{1 + \alpha I^*}{(1 + \alpha I^*)(1 + h\mu) + h\beta I^*}; \; J_{12} = 0; \; J_{13} = -\frac{h\beta(S^* + h\Lambda + \mathrm{h}\gamma R^*)}{(1 + \alpha I^*)^2\left(1 + h\mu + \frac{h\beta I^*}{1 + \alpha I^*}\right)^2};$$

$$J_{14} = \frac{h\gamma(1+\alpha I^*)}{(1+\alpha I^*)(1+h\mu)+h\beta I^*};\ J_{21} = \frac{h\beta I^*}{(1+\alpha I^*)\big(1+h(\mu+\rho)\big)};\ J_{22} = \frac{1}{1+h(\mu+\rho)};$$
$$J_{23} = \frac{h\beta S^*}{(1+\alpha I^*)^2\big(1+h(\mu+\rho)\big)};\ J_{24} = 0;\ \ J_{31} = 0;\ J_{32} = \frac{h\rho}{1+h(\mu+\zeta+\omega)+\frac{\upsilon h}{1+\eta I^*}}$$
$$J_{33} = \frac{1}{1+h(\mu+\zeta+\omega)+\frac{\upsilon h}{1+\eta I^*}}\left[1+\frac{h\upsilon}{(1+\eta I^*)^2\left(1+h(\mu+\zeta+\omega)+\frac{\upsilon h}{1+\eta I^*}\right)}\right][I^* - h\rho E^*];$$
$$J_{34} = 0;\ J_{41} = 0;\ J_{42} = 0;\ J_{43} = \frac{h\omega(1+\eta I^*)+h\upsilon}{(1+\eta I^*)\big(1+h(\mu+\gamma)\big)};\ J_{44} = \frac{1}{1+h(\mu+\gamma)}$$
$$\Longrightarrow J(\mathrm{T}^*) = \begin{bmatrix} J_{11} & 0 & J_{13} & J_{14} \\ J_{21} & J_{22} & J_{23} & 0 \\ 0 & J_{32} & J_{33} & 0 \\ 0 & 0 & J_{43} & J_{44} \end{bmatrix}$$

Clearly, we observe from $J(\mathrm{T}^*)$ that two eigenvalues are $J_{11} = \frac{1+\alpha I^*}{(1+\alpha I^*)(1+h\mu)+h\beta I^*}$ and $J_{44} = \frac{1}{1+h(\mu+\gamma)}$ which are both less than unity. The remaining two eigenvalues can be found from the matrix

$$M_1(\mathrm{T}^*) = \begin{bmatrix} J_{22} & J_{23} \\ J_{32} & J_{33} \end{bmatrix}.$$

Thus, $M_1(\mathrm{T}^*) - \lambda I = 0$ gives

$$\begin{vmatrix} J_{22}-\lambda^* & J_{23} \\ J_{32} & J_{33}-\lambda^* \end{vmatrix} = 0$$
$$(J_{22}-\lambda^*)(J_{33}-\lambda^*) - J_{23}J_{32} = 0$$
$$\lambda^{*2} - [J_{22}+J_{33}]\lambda^* + J_{22}J_{33} - J_{23}J_{32} = 0,$$

where

$$J_{22}+J_{33} = \left[\left[\frac{1}{1+h(\mu+\rho)}\right] + \left[\frac{(1+\eta I^*)[(1+\eta I^*)+h(\mu+\zeta+\omega)(1+\eta I^*)+2h\upsilon]}{[(1+\eta I^*)+h(\mu+\zeta+\omega)(1+\eta I^*)+h\upsilon]^2}\right][I^* - h\rho E^*]\right]$$
$$J_{22}J_{33} = \left[\frac{1}{1+h(\mu+\rho)}\right]\left[\frac{(1+\eta I^*)[(1+\eta I^*)+h(\mu+\zeta+\omega)(1+\eta I^*)+2h\upsilon]}{[(1+\eta I^*)+h(\mu+\zeta+\omega)(1+\eta I^*)+h\upsilon]^2}\right][I^* - h\rho E^*]$$

and

$$J_{23}J_{32} = \left[\frac{h\beta S^*}{(1+\alpha I^*)^2(1+h(\mu+\rho))}\right]\left[\frac{h\rho(1+\eta I^*)}{(1+h(\mu+\zeta+\omega))(1+\eta I^*)+\upsilon h}\right].$$

Observe that the spectral radius of the Jacobian matrix $M_1(\mathrm{T}^*)$ for each time step "h" such that $0 < h < 1000$ as shown in figure 3 remains less than unity for each time step. This simply indicates that all the eigenvalues of the Jacobian matrix at the dental caries endemic equilibrium will be within a unit disc which assures that the proposed NSFD scheme is convergent for each of the selected time step.

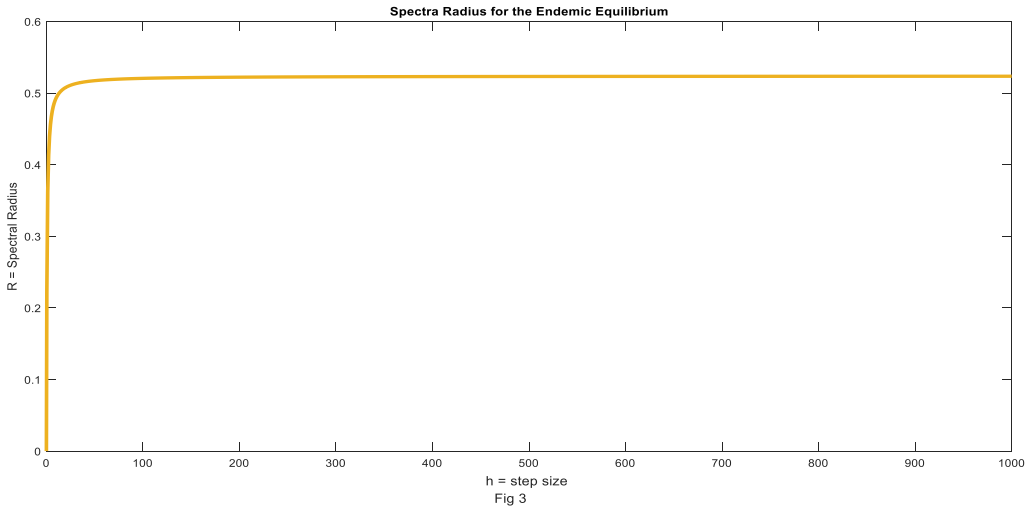

Figure 3: The graph of Spectral radius for the Dental Caries (decay) Endemic equilibrium

5 Numerical Schemes

In this section, we develop three numerical schemes using model system (1) with the help of the parameter values in tables 1 and 2. The numerical schemes that shall be considered in this section are NSFD scheme, R-K 4 and Forward Euler (FD) scheme. Finally, NSFD scheme numerical results will be compared with the other two schemes.

Table 1: Parameter values for the Dental Caries (Decay) Free Equilibrium (DCFE)

Parameter	β	Λ	μ	ζ	ω	α	ρ	η	ν	γ
Value	0.05	0.7	0.13	0.05	0.50	0.0002	0.8	0.000001	0.5	0.000001

Table 2: Parameter values for the Dental Caries (Decay) Endemic Equilibrium (DCFE)

Parameter	β	Λ	μ	ζ	ω	α	ρ	η	ν	γ
Value	0.5	0.7	0.13	0.005	0.30	0.00002	0.7	0.0000001	0.5	0.000001

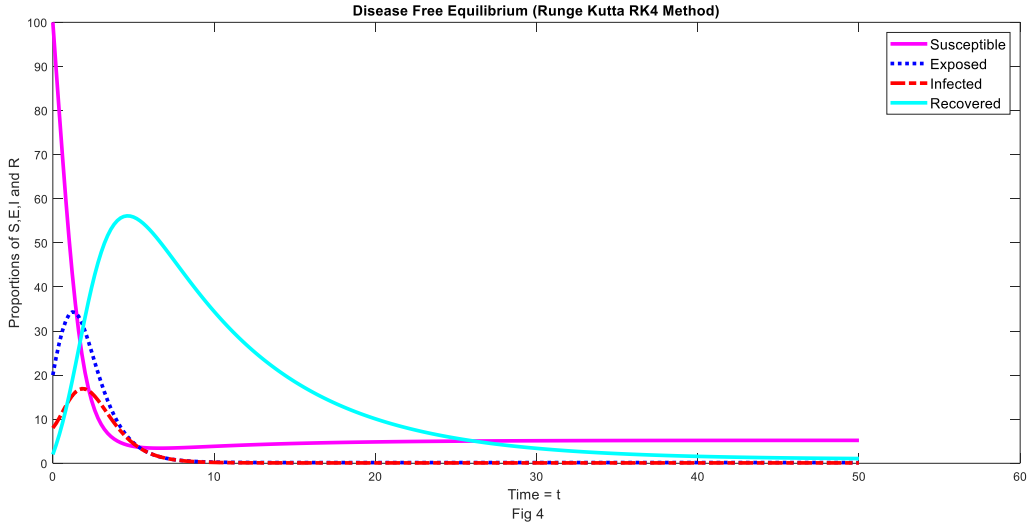

Figure 4: Graph of RungeKutta RK-4 Method for the Dental Caries (Decay) Free Equilibrium with step size $h = 0.2$.

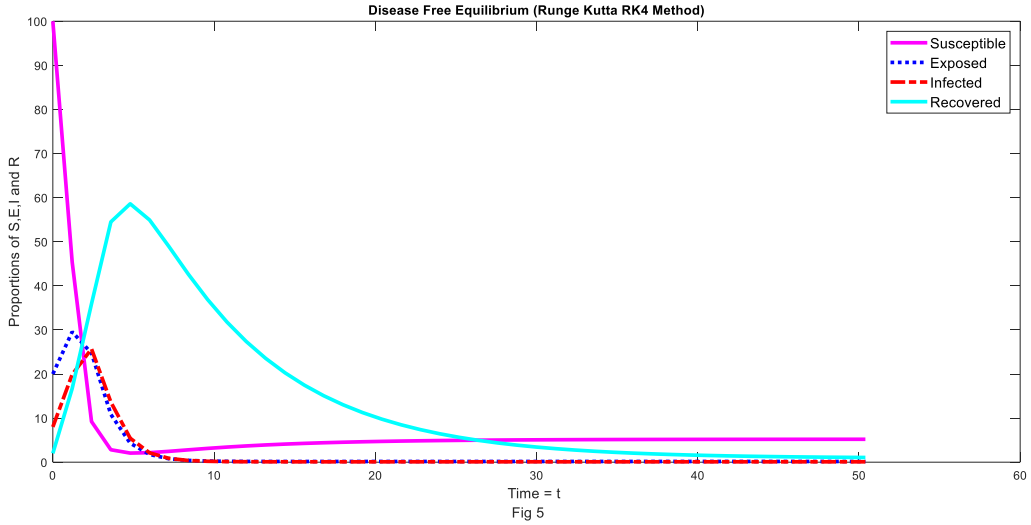

Figure 5: Graph of RungeKutta RK-4 Method for the Dental Caries (Decay) Free Equilibrium with step size $h = 1.2$.

Figures 4 and 5 shows the convergence of the Runge-Kutta fourth order method RK-4 for the dental caries (decay) free equilibrium at $h = 0.2$ *and* $h = 1.2$ respectively from which we observe the behavior of RK-4 scheme for various values of time step.

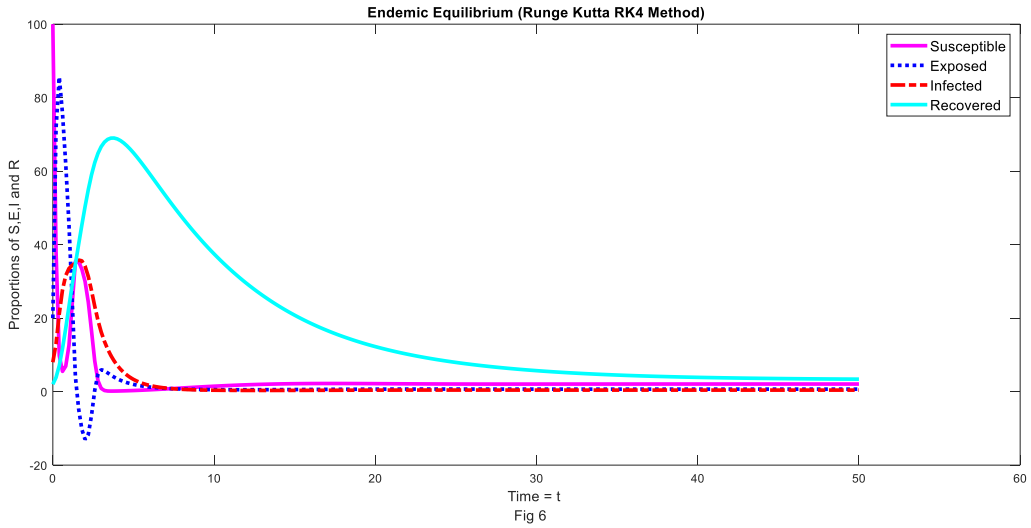

Figure 6: Graph of RungeKutta RK-4 Method for the Dental Caries (Decay) Endemic Equilibrium with step size $h = 0.2$.

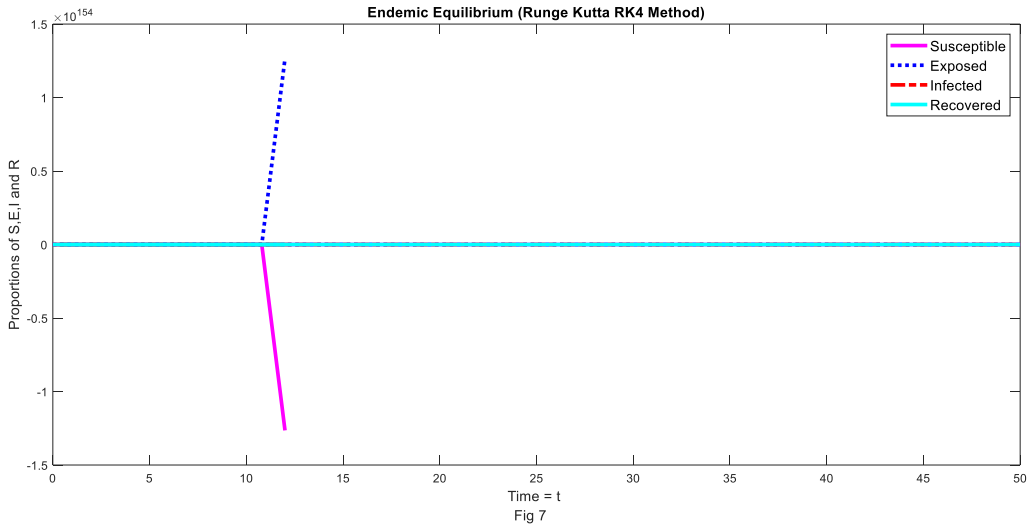

Figure 7: Graph of RungeKutta RK-4 Method for the Dental Caries (Decay) Endemic Equilibrium with step size $h = 1.2$.

Figures 6 and 7 showed that RK-4 scheme loss the positivity property and produce negative values for the compartments at dental caries endemic equilibrium with $h = 0.2$ *and* $h = 1.2$ respectively. Negative values of the compartments are worthless and they are due to drawback of RK-4 scheme. It shows the non- physical behavior of the compartments which is not consistence with the continuous dynamical system. Actually, figure 7 show the overflow of RK-4 scheme at $h = 1.2$.

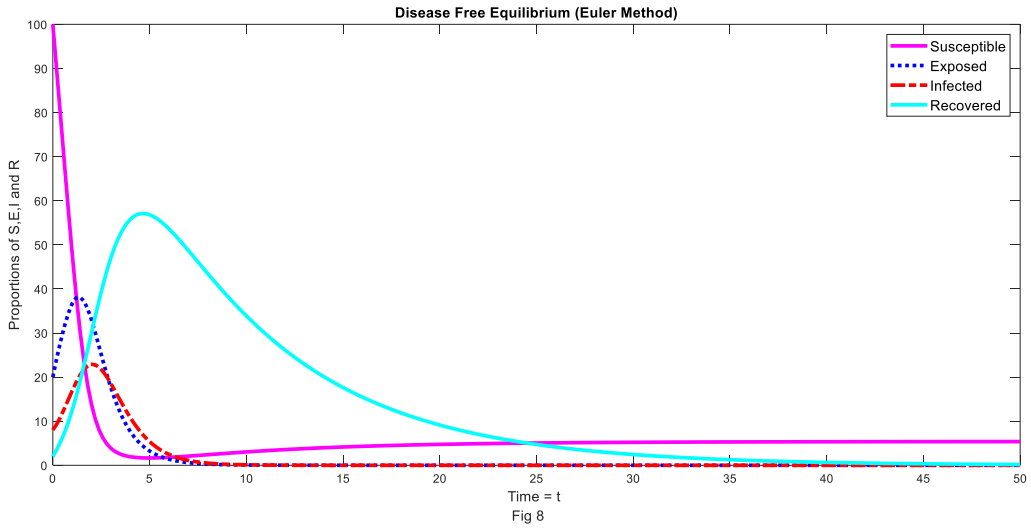

Figure 8: Graph of Forward Euler (FD) Scheme for the Dental Caries (Decay) Free Equilibrium with step size $h = 0.2$.

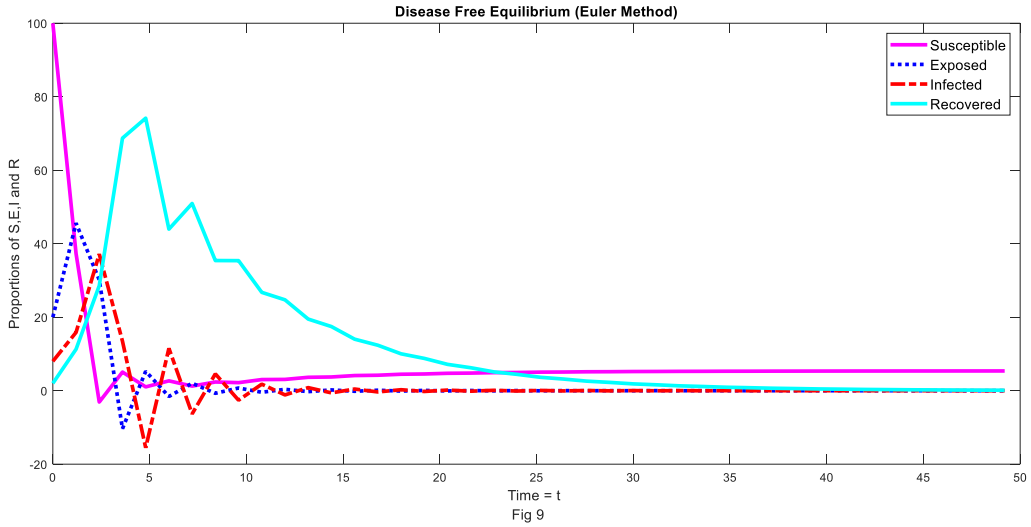

Figure 9: Graph of Forward Euler (FD) Scheme for the Dental Caries (Decay) Free Equilibrium with step size $h = 1.2$.

Figures 8 and 9 showed the convergence of Forward Euler (FD) method for the dental caries (decay) free equilibrium at $h = 0.2$ and $h = 1.2$ respectively. We observe that the scheme lose the positivity property and produce negative values of the compartments at $h = 1.2$. Therefore, we should note that the negative values are due to the drawback of the Forward Euler (FD) scheme. Also, figure 9 shows the overflow of the Forward Euler (FD) scheme.

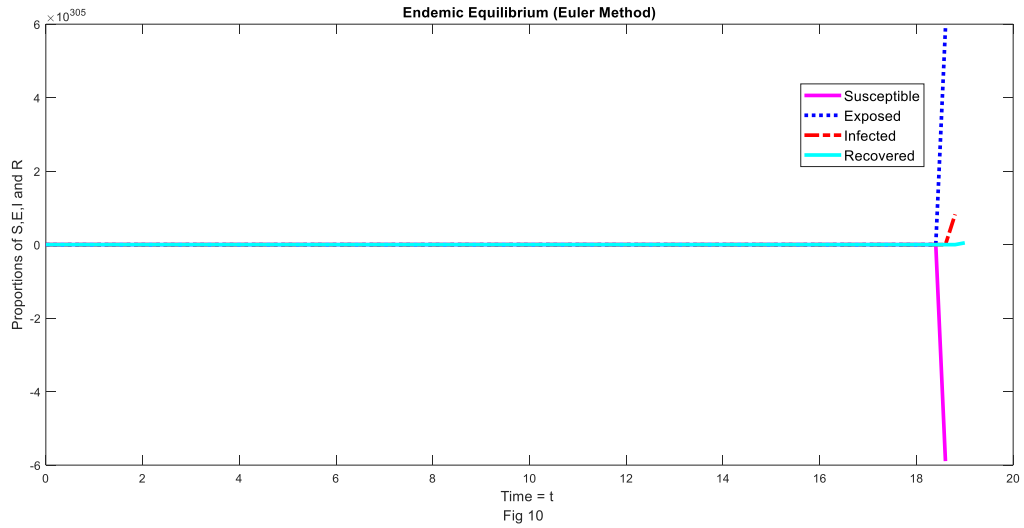

Figure 10: Graph of Forward Euler (FD) Scheme for the Dental Caries (Decay) Endemic Equilibrium with step size $h = 0.2$.

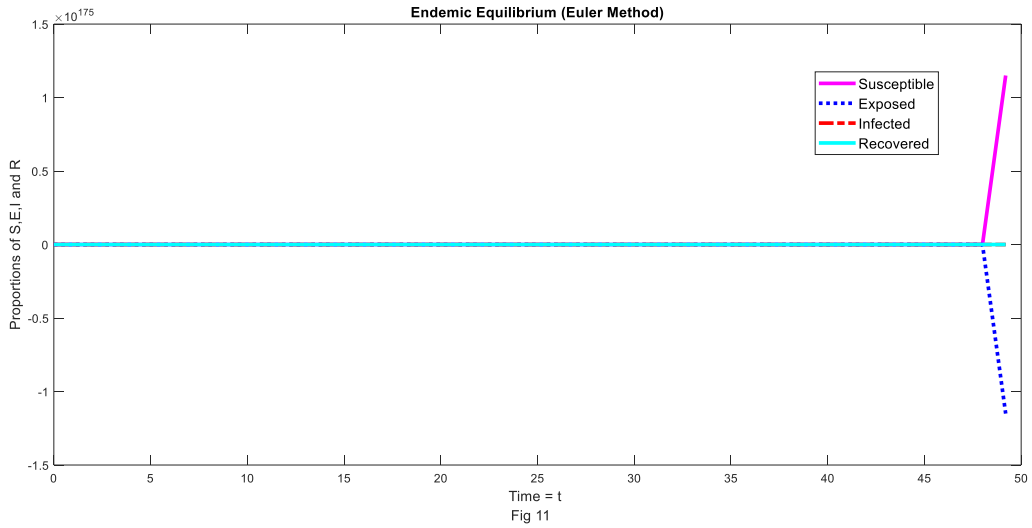

Figure 11: Graph of Forward Euler (FD) Scheme for the Dental Caries (Decay) Endemic Equilibrium with step size $h = 1.2$.

Figures 10 and 11 represents the behavior of both the susceptible, exposed, infected and recovered individuals using Forward Euler (FD) scheme for the dental caries endemic equilibrium at $h = 0.2$ *and* $h = 1.2$ respectively. We shall observe that the scheme at the endemic equilibrium produce negative value for all the compartments. This is worthless and is due to the drawback of the Forward Euler (FD) scheme.

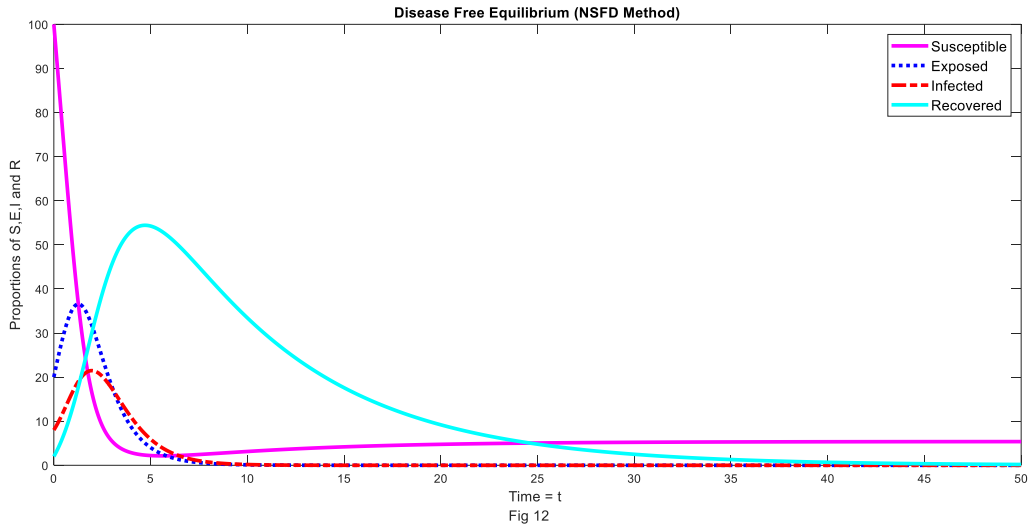

Figure 12: Graph of Non Standard Finite Difference (NSFD) Scheme for the Dental Caries (Decay) Free Equilibrium with step size $h = 0.2$.

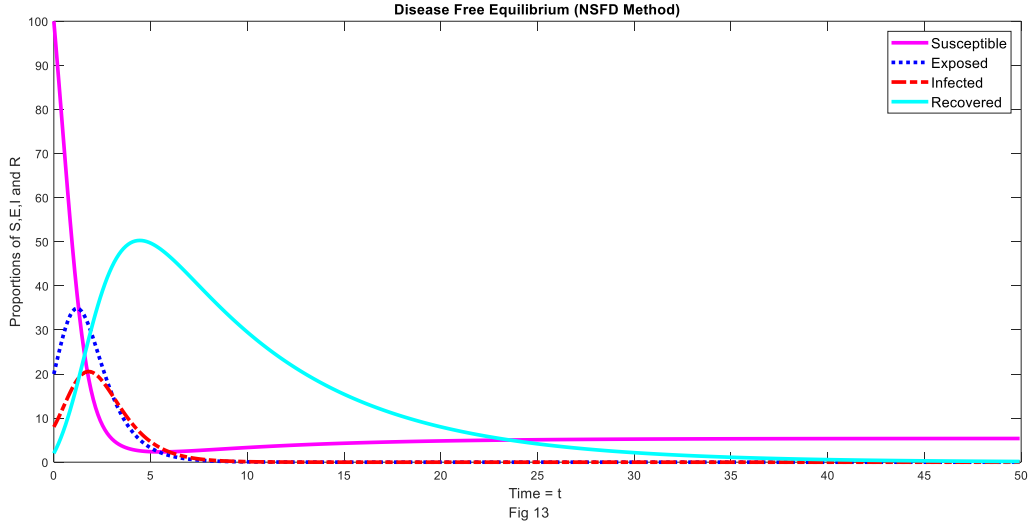

Figure 13: Graph of Non Standard Finite Difference (NSFD) Scheme for the Dental Caries (Decay) Free Equilibrium with step size $h = 1.2$.

Figures 12 and 13 showed the graphs of susceptible, exposed, infected and recovered populations using non-standard finite difference (NSFD) scheme with $h = 0.2$ *and* $h = 1.2$ respectively at the dental caries free equilibrium. These graphs of the proposed NSFD scheme preserves positivity property and converges to the true steady state of the continuous model for each values of the time step h.

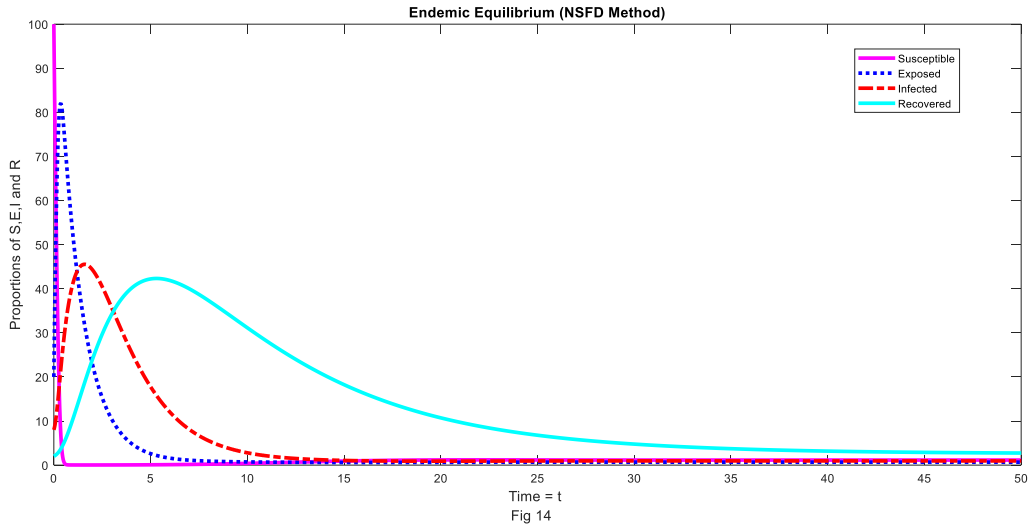

Figure 14: Graph of Non Standard Finite Difference (NSFD) Scheme for the Dental Caries (Decay) Endemic Equilibrium with step size $h = 0.2$.

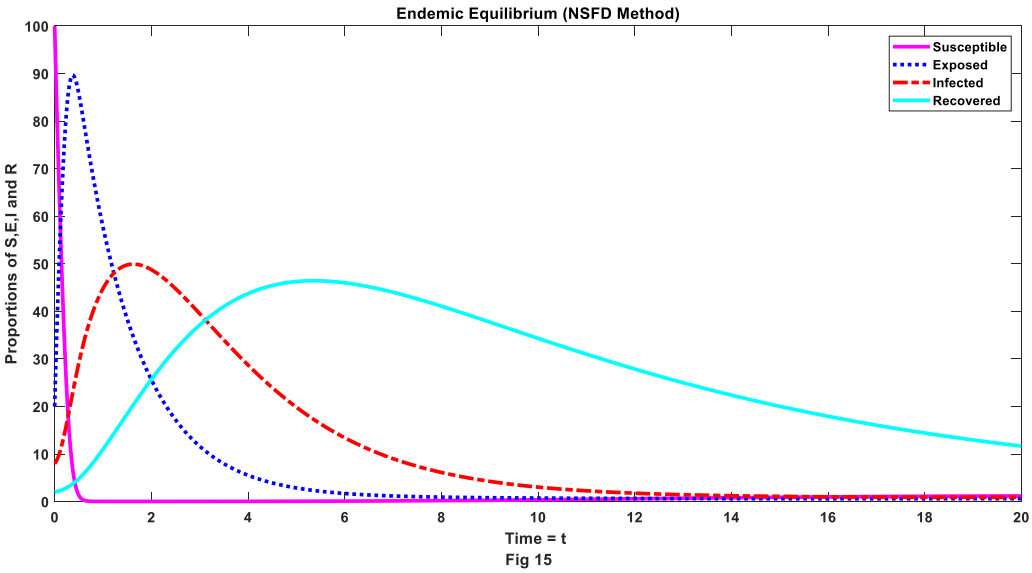

Figure 15: Graph of Non Standard Finite Difference (NSFD) Scheme for the Dental Caries (Decay) Endemic Equilibrium with step size $h = 1.2$.

Figure 14 and 15 showed the dental caries endemic equilibrium for the different compartments of the model system (1) using NSFD scheme at $h = 0.2\ and\ h = 1.2$ respectively. The graphs indicated that NSFD scheme is unconditionally convergent for all time steps and preserves positivity property.

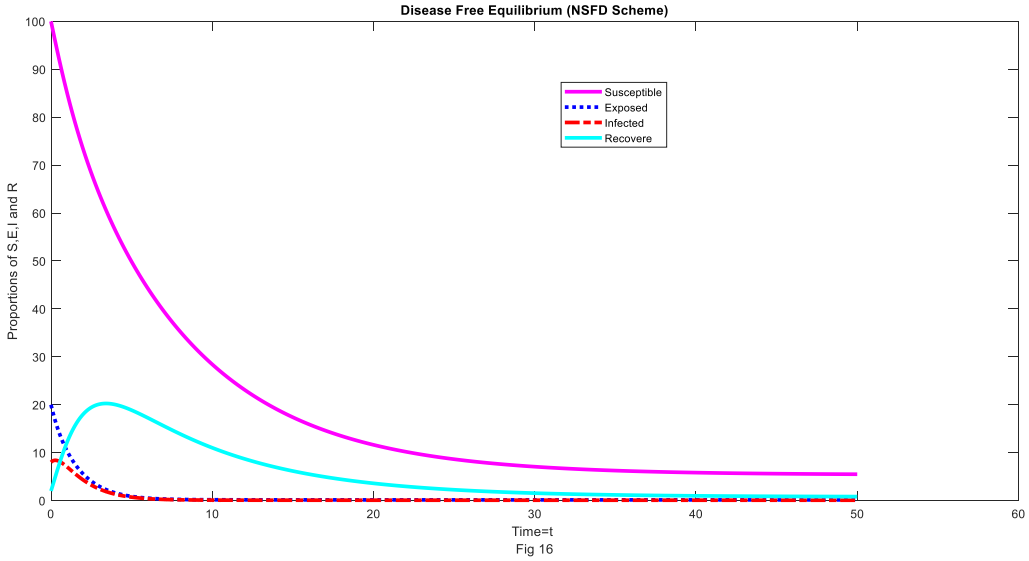

Figure 16: Graph of Non Standard Finite Difference (NSFD) Scheme for the Dental Caries (Decay) infection with step size $h = 0.2, \eta = 0.001$ *and* $\nu = 0.9$.

Figure 16 displayed the effect of maximum medical resources, saturated factors that measure the effect of the infected being delayed for treatment and saturated factor that measures the inhibitory effects, that is, prevention from being exposed. It is observed that with, with adequate prevention from being exposed, maximum medical resources and treatment not delayed, both the exposed and infected individuals dynamics will automatically reduce in the population thereby controlling dental caries (decay) infection in the population.

6 Conclusion

Many researchers applied many numerical techniques to solve many mathematical models. To analyze the dental caries (decay) SEIR epidemic model, we used three different numerical schemes such as; Runge Kutta RK-4 method, Forward Euler (FD) scheme and Non-Standard Finite Difference (NSFD) scheme. In this paper we are interested in the in the numerical solution dental caries infection using SEIR epidemic model with saturation incidence rate and saturated treatment function. In this research work, we developed Runge Kutta RK-4 method, Forward Euler (FD) scheme and Non Standard Finite Difference (NSFD) scheme for our SEIR epidemic model.

The basic reproduction number and stability analysis for dental caries (decay) free equilibrium and endemic equilibrium points are evaluated. In the three numerical schemes applied in this work, the Non-standard finite difference scheme was found to preserve the essential properties such as positivity, unconditional convergence, and stability. We analyze the convergence of the three numerical schemes of the epidemic model of dental caries (decay) and found from the result that NSFD scheme converges unconditionally for each step size, while Runge-Kutta RK-4 method and Forward Euler (FD) scheme fail to converge at the endemic equilibrium even with the small step size $h = 0.2$. The result also showed that only NSFD scheme preserve positivity property where as Runge Kutta RK-4 method and Forward Euler (FD) scheme fail preserve positivity property.

The effect of maximum medical resources and saturated factors that measure the effect of delaying the treatment of infected dental caries individuals were discussed graphically in figure 16. It was found that if medical resources are maximized and treatment of dental caries infected individuals not delayed, the exposed and infected individuals in the population will be decreased thereby reducing the effective contact rate. In this regard, dental caries (decay) is effectively and efficiently controlled in the population.

From the numerical analysis we conclude that the non-standard finite difference (NSFD) scheme performed better than Runge Kutta RK-4 method and Forward Euler (FD) scheme when considered especially in obtaining solution curves that are consistent with analytic solution. Also, that if maximum medical resources are made available with timely treatment of the infected dental caries individuals, dental caries infection will be eradicated in the population.

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