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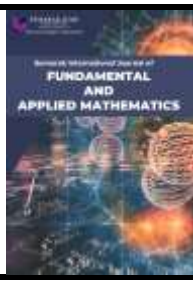

Numerical Analysis of The NSP Epidemic Model for Campus Drinking Dynamics

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ABSTRACT

Significant public health risks have been posed by the drinking habits of college students for several generations. In order to tackle this issue thoroughly, the analysis of the NSP (Non-Drinkers, Social Drinkers, Problem Drinkers) epidemic model is conducted to understand which factors influence the dynamics of drinking behaviour in campus. The impact of different numerical methods for solving the model is evaluated, and recommendations for approaches to reduce the misuse of alcohol are mathematically examined. The model is solved using three techniques: the Euler, Runge-Kutta 4th order (RK-4), and the Non-standard Finite Difference (NSFD). The results provided by the NSFD scheme are the most important, as it maintains essential properties of the NSP model, such as the positivity of solutions and the stability of equilibrium, which are not preserved when the Euler and RK-4 methods are used. The basic reproductive numbers serve as the main result on which the further extension of the outbreak can be investigated. The effective reproduction numbers R_0^S and R_0^P are derived to determine the stability of equilibrium points. Moreover, the NSFD method is shown to preserve positivity for all time step sizes, making it suitable for epidemic modelling. Finally, numerical simulations are considered to investigate the effectiveness of the NSFD method. These results imply that the NSFD scheme is more appropriate for characterizing the dynamics of campus drinking and could provide valuable information for the prevention of alcohol related issues among students.

1. Introduction

In pre-historic times, French chemists discovered the products of natural fermentation, which were quickly followed by the intention to make wines and beers from starchy and sugary vegetation. In the early days, alcohol was regarded as food, a medical drug, and a euphoriant, as well as being used in spiritual symbolism and society. Since the days of Plymouth Rock, alcoholic beverages have been consumed in the USA. In fact, wine and beer were staples on the ships carrying settlers to the

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New World. In colonial times, milk and water were scarce and prone to pollution or waste, while coffee and tea were expensive [1]. The settlers turned to alternatives such as whisky and beer, and less frequently, to liquors like brandy and gin. In 1790, per capita consumption of pure whisky, or absolute whisky, was just under six gallons a year. Natural liquor composes only a small percentage of alcoholic drinks. For instance, if a drink contains ten percent alcohol by volume, one would have to consume ten gallons of it to drink one gallon of pure liquor [2]. Despite the fact that most of the colonists consumed alcohol frequently, sturdy community limitations restrained any tendency towards extreme levels. Drunken conduct was handled by emphasizing harmony and balance, instead of enforcing punishment. Alcohol consumption persisted without much controversy or conflict, as beer and other refined spirits became valuable commercial supplies [3]. When Congress imposed customs taxes on the agriculturists who manufactured alcohol in the 1790s, they refused to pay the tax. Their opposition is referred to as the Whiskey Rebellion, a protest movement of farmers who perceived the tax as an undue burden on their financial activities. A recent survey in the United States reveals that nearly 90 percent of college students drink alcohol at least once [4].

The college students who consume alcohol faced many consequences, like those students who drink may have average results. College is a time of huge change for teenagers across the United States of America and around the world. With newfound freedom comes the chance to explore an entirely new territory of possibilities associated with crossing the bridge from childhood into the trenches of adulthood [5]. These can very well be the defining moments in a person's life. Abusive and immature alcohol consumption among college students is a widespread public health issue, exacting a significant toll on the mental and social lives of college students on campuses all over the USA. Drinking alcohol in college has become a custom that students often see as an essential part of their higher education experience. Several college students arrive on campus with established drinking habits, and the college environment worsens the problem [6].

When a person consumes alcohol, nearly 20 percent of the alcohol is absorbed in the stomach, and approximately 80 percent is absorbed in the small intestine. The rate at which alcohol is absorbed depends on several factors:

i) The biological gender of the drinker: Alcohol is absorbed differently in men and women due to differences in physiology.
ii) The concentration of alcohol in the beverage: The higher the concentration, the faster the absorption.
iii) The type of alcohol: Carbonated drinks tend to accelerate the absorption of alcohol.
iv) The presence of food in the stomach: Food in the stomach slows down alcohol absorption, depending on whether the stomach is full or empty [7].

Whilst we examine males and females of the same height, weight, and build, males generally tend to have more muscle mass and lower fat content than females. This is because muscle volume contains more water compared to fat tissue. As a result, a specific quantity of alcohol can be diluted more effectively in males than in females. Consequently, the blood-alcohol concentration (BAC) from the same amount of alcohol is higher in women than in men, and women also feel the effects of that amount of alcohol faster than men [8].

As a general rule of thumb, an average man or woman can metabolize approximately 0.5 ounces (15 ml) of alcohol per hour. Therefore, it would take about 1 hour to eliminate the alcohol from a 12-ounce (355 ml) can of beer. The BAC increases when the body absorbs alcohol faster than it can eliminate it [9]. Hence, because the body can only metabolize approximately one serving of alcohol

per hour, consuming multiple drinks in one hour will significantly increase your BAC compared to consuming one drink over a period of one hour or more.

The breakdown, or metabolism, of ethanol occurs in the liver. An enzyme in the liver called alcohol dehydrogenase (ADH) strips electrons from ethanol to produce acetaldehyde [10]. Another enzyme, known as aldehyde dehydrogenase, converts acetaldehyde, in the presence of oxygen, into acetic acid the primary component of vinegar. The molecular structure of acetic acid is represented as CH_3COOH. When ethanol reacts with acetic acid, electrons and protons are also produced. Acetic acid can be used to form fatty acids or further broken down into water and carbon dioxide [11].

As stated by the National Cancer Institute:" According to scientists, drinking may cause various kinds of cancer. Research shows that when a man consumes a lot of beer, especially over time, there is an increased possibility of developing beer-related cancer [12]. Also, those who do not drink more than once a day but later consume large amounts of alcohol simultaneously have an increased risk of other cancers. Approximately 3.5 percent of cancer deaths in the USA occurred in 2009 due to alcohol consumption. About 19,500 people died as a result of alcohol intake." Drinking has a significant effect on memory. Firstly, drinking interrupts the ability to form long-term memories, causing less disturbance in recalling earlier-formed long-term memories or the ability to retain new data in short-term memory for several seconds or more [13]. At low levels, the effects of alcohol are usually subtle, although they are visible under controlled conditions. As the dose of alcohol increases, so does the extent of memory impairment. Youngsters who consume alcohol at least once a month are twice as likely to commit an illegal act compared to those who abstain [14]. More than one-third of teens who consume alcohol at least once a week have committed violent crimes, including theft or assaulting. Youngsters who engage in such behaviour often develop a criminal record that can negatively impact their future [15]. This could destroy their opportunities for the rest of their lives. Due to a criminal record, a person may not qualify for certain jobs, and some crimes may bar them from traveling abroad.

The assumptions made by Hethcote [16,17] in 1976 form the foundation for this model. In this paper, we compare the Forward Euler, RK-4, and NSFD methods. The NSFD method proves to be more efficient and reliable than the Forward Euler and RK-4 methods, as it demonstrates convergence even at very small step sizes [18].

This paper is organized into seven sections. Section 1 covers the literature review and background. Section 2 focuses on the formulation of the model. In Section 3, we present the equilibrium states, stability, and threshold analysis. Section 4 provides results about reproductive number, while section 5 insights into numerical modelling and the convergence analysis of NSFD. Section 6 showcases the graphical comparison of Euler, RK4, and NSFD methods. Finally, Section 7 concludes the paper with a summary of key findings.

2. Model Formulation

We took model from [3] where concentrate on college-drinking and divide total students into three compartments: Non-drinkers (N), Social drinkers (S) and Problem drinkers (P) (see Figure 1). The model is governed by following nonlinear differential equation.

The total population is represented by N and is sub-divided into three sub-populations Non-drinkers (N), Social drinkers (S) and Problem drinkers (P).

$$N(t) + S(t) + P(t) = 1$$

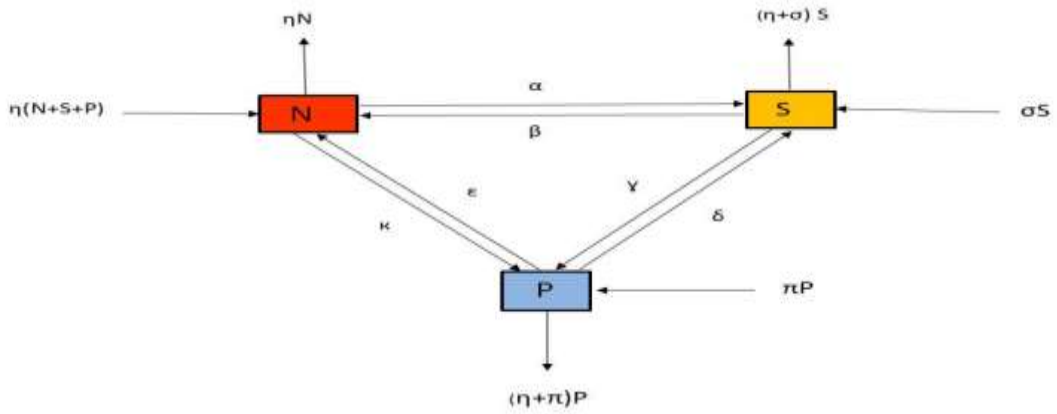

Fig. 1. NSP model

Variable of compartmental model are N: Susceptible, S: Infected and P represent the Infected.

$$\frac{dN}{dt} = \eta - \eta N - \alpha NS - \kappa NP + \beta S + \epsilon P \quad (1)$$

$$\frac{dS}{dt} = \sigma S - (\eta + \sigma)S + \alpha NS - \beta S - \gamma SP + \delta P \quad (2)$$

$$\frac{dP}{dt} = \pi P - (\eta + \pi)P + \gamma SP + \kappa NP - \delta P - \epsilon P \quad (3)$$

Changing from N to S, N to P, and S to P are modeled by using terms αNS, κNP and γSP respectively. And conversion from S to N, P to N and P to S consider recovery process through βS, ϵP and δP respectively.

3. Disease free Equilibrium

$$\pi P - (\eta + \pi)P + \gamma SP + \kappa NP - \delta P - \epsilon P = 0 \quad (4)$$

$$\sigma S - (\eta + \sigma)S + \alpha NS - \beta S - \gamma SP + \delta P = 0 \quad (5)$$

$$\eta - \eta N - \alpha NS - \kappa NP + \beta S + \epsilon P = 0 \quad (6)$$

From above equation we get

$$\varepsilon_0 = (\mathrm{N}, \mathrm{S}, \mathrm{P}) = (1, 0, 0)$$

3.1 Drinking Free Equilibrium

The equilibrium considered by deficiency of the problem drinkers P = 0, eigenvalues at problem DFE $\varepsilon_0 = \left(\frac{\eta+\beta}{\alpha}, 1 - \frac{\eta+\beta}{\alpha}, 0\right)$. Differentiating f, g and h with respect to compartmental model parameters N, S and P.

$$J = \begin{bmatrix} f_N & f_S & f_P \\ g_N & g_S & g_P \\ h_N & h_S & h_P \end{bmatrix} \tag{7}$$

$$J = \begin{bmatrix} -\eta - \alpha S - kP & -\alpha N + \beta & -kN + \epsilon \\ \alpha S & -\eta + \alpha N - \beta - \gamma P & -\gamma S + \delta \\ kP & \gamma P & -\eta + \gamma S + kN - \delta - \epsilon \end{bmatrix} \tag{8}$$

$$J\left(\frac{\eta+\beta}{\alpha}, 1 - \frac{\eta+\beta}{\alpha}, 0\right) = \begin{pmatrix} -\alpha + \beta & -\eta & -\kappa\left(\frac{\eta+\beta}{\alpha}\right) + \epsilon \\ \alpha - \eta + \beta & 0 & -\gamma\left(\frac{\alpha-\eta-\beta}{\alpha}\right) + \delta \\ 0 & 0 & -\eta + \gamma\left(\frac{\alpha-\eta-\beta}{\alpha}\right) + \kappa\left(\frac{\eta+\beta}{\alpha}\right) - \delta - \epsilon \end{pmatrix} \tag{9}$$

$\lambda_1 = -\eta$, $\lambda_2 = \alpha - \eta - \beta$ and $\lambda_3 = \frac{-\eta\alpha+\alpha\gamma-\eta\gamma-\beta\gamma+\eta\kappa+\beta\kappa-\alpha\delta-\alpha\epsilon}{\alpha}$ are eigenvalues at problem DFE.

4. Reproductive Number

In disease transmission research, cutting edge grid is a technique used to determine the multiplication number needed for a compartmental model of unchecked disease propagation. Jacobin Network estimates F and V require a disease-free balance ε_0. Then there is a permanent state structure.

$$\frac{dF_k}{dX_l}(\varepsilon_0) = \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix}$$

$$\frac{dV_k}{dX_l}(\varepsilon_0) = \begin{pmatrix} F & 0 \\ L_3 & L_4 \end{pmatrix}$$

Here F and V are m × m matrices. We used the next generation matrix technique to calculate the reproductive number.

$$\frac{dS}{dt} = \alpha NS - \eta S - \beta S - \gamma SP + \delta P$$

$$\frac{dP}{dt} = \kappa NP - \eta P + \gamma SP - \delta P - \epsilon P$$

Next Generation Method:

$$\frac{dx}{dt} = f(x, y) - v(x, y)$$

$F = \begin{bmatrix} \alpha NS \\ \kappa NP \end{bmatrix}$, V= $\begin{bmatrix} \eta S + \beta S + \gamma SP - \delta P \\ \eta P - \gamma SP + \delta P + \epsilon P \end{bmatrix}$.

Hence, at the disease-free equilibrium (DFE) points, the transmissions matrix T and V are

$$F = \begin{bmatrix} NS \\ NP \end{bmatrix} \tag{10}$$

$$V = \begin{bmatrix} \eta + \beta + \gamma P \\ \eta + \delta + \epsilon - \gamma S \end{bmatrix} \tag{11}$$

F represents the changes of new infections and V transitions approaching equilibrium. So, the fundamental reproductive number has the highest eigenvalue. Largest eigenvalue of Jacobian matrix

$$R_0^S = FV^{-1} = \frac{\alpha}{\eta+\beta} \tag{12}$$

$$R_0^P = FV^{-1} = \frac{k}{\eta+\delta+\epsilon} \tag{13}$$

4.1 Endemic Equilibrium

$$\eta - \eta N - \alpha NS - \kappa NP + \beta S + \epsilon P = 0$$

$$\pi P - (\eta + \pi)P + \gamma SP + \kappa NP - \delta P - \epsilon P = 0$$

$$\sigma S - (\eta + \sigma)S + \alpha NS - \beta S - \gamma SP + \delta P = 0$$

From above equation we get

$$N = \frac{\eta-\gamma S+\delta+\epsilon}{\mathrm{k}}$$

$$P = \frac{\alpha\gamma S^2+(\eta k+\beta k-\alpha\eta-\alpha\delta-\alpha\epsilon)S}{\mathrm{k}(\delta-\gamma \mathrm{S})}$$

$$S^2 + bs + c = 0,$$

here,

$$b = \frac{\kappa\gamma + \alpha\delta + \varepsilon\gamma + \eta\alpha - \delta\kappa + \alpha\varepsilon - \beta\kappa + \eta\gamma + 2\gamma\delta - \eta\kappa}{\kappa\gamma - \gamma^2 - \alpha\gamma}$$

and

$$c = \frac{-\delta\varepsilon - \eta\delta - \delta^2 + \delta\kappa}{\kappa\gamma - \gamma^2 - \alpha\gamma}$$

4.2 Local Stability

The trivial equilibrium points above represent drinking-free environment. So, analysis of local stability of trivial equilibrium prepares us to determine conditions under which drinking environment can be established. This found out from the eigenvalues of Jacobian matrix.

$$\frac{dN}{dt} = f = \eta - \eta N - \alpha NS - \kappa NP + \beta S + \epsilon P$$

$$\frac{dS}{dt} = \mathrm{g} = \sigma \mathrm{S} - (\eta + \sigma)\mathrm{S} + \alpha \mathrm{NS} - \beta \mathrm{S} - \gamma \mathrm{SP} + \delta \mathrm{P}$$

$$\frac{dP}{dt} = \mathrm{h} = \pi \mathrm{P} - (\eta + \pi)\mathrm{P} + \gamma \mathrm{SP} + \kappa \mathrm{NP} - \delta \mathrm{P} - \epsilon \mathrm{P}$$

Differentiating f, g and h with respect to compartmental model parameters N, S and P.

$$J = \begin{bmatrix} f_N & f_S & f_P \\ g_N & g_S & g_P \\ h_N & h_S & h_P \end{bmatrix} \tag{14}$$

$$J = \begin{bmatrix} -\eta - \alpha \mathrm{S} - \mathrm{kP} & -\alpha N + \beta & -kN + \epsilon \\ \alpha S & -\eta + \alpha \mathrm{N} - \beta - \gamma \mathrm{P} & -\gamma S + \delta \\ kP & \gamma P & -\eta + \gamma \mathrm{S} + \mathrm{kN} - \delta - \epsilon \end{bmatrix} \tag{15}$$

By drinking free point

$$J(1,0,0) = \begin{bmatrix} -\eta & -\alpha + \beta & -k + \epsilon \\ 0 & -\eta + \alpha - \beta & \delta \\ 0 & 0 & -\eta + \mathrm{k} - \delta - \epsilon \end{bmatrix} \tag{16}$$

By Characteristic Equation det (J − λI) = 0

$\lambda_1 = -\eta$, $\lambda_2 = \alpha - \eta - \beta$ and $\lambda_3 = k - \eta - \delta - \epsilon$

are eigenvalues at drinking free equilibrium. Table 1 shows the parametric values.

Table 1
Parametric values

Parameters	Values	Reference
α	0.4	[3]
δ	0.2	[3]
β	0.2	DFE(Fitted), EE [3]
γ	0.4	[3]
ϵ	0.2	[3]
η	0.25	[3]
κ	0.15	[3]
σ	0.25	[3]
π	0.1	[3]

5. Numerical Modelling of SEIR on Vaccine

$$\frac{dN}{dt} = \eta - \eta N - \alpha NS - \kappa NP + \beta S + \epsilon P$$

$$\frac{dS}{dt} = \sigma S - (\eta + \sigma)S + \alpha NS - \beta S - \gamma SP + \delta P$$

$$\frac{dP}{dt} = \pi P - (\eta + \pi)P + \gamma SP + \kappa NP - \delta P - \epsilon P$$

After simplification,

$$\frac{dN}{dt} = \eta - \eta N - \alpha NS - \kappa NP + \beta S + \epsilon P$$

$$\frac{dS}{dt} = -\eta S + \alpha NS - \beta S - \gamma SP + \delta P$$

$$\frac{dP}{dt} = -\eta P + \gamma SP + \kappa NP - \delta P - \epsilon P$$

The NSP model is represented using ordinary differential equations. We will now tackle the problem by numerical modelling. The Forward Euler scheme will be used first, then the Fourth Order Runge-Kutta scheme, and lastly the NSFD method.

5.1 Forward Euler's Scheme

$$N^{n+1} = N^n + h(\eta - \eta N^n - \alpha N^n S^n - \kappa N^n P^n + \beta S^n + \epsilon P^n) \quad (17)$$

$$S^{n+1} = S^n + h(-\eta S^n + \alpha N^n S^n - \gamma S^n P^n - \beta S^n + \delta P^n) \quad (18)$$

$$P^{n+1} = P^n + h(-\eta P^n + \gamma P^n S^n + \kappa N^n P^n - \delta P^n - \epsilon P^n) \quad (19)$$

5.2 Fourth Order Runge-Kutta Scheme

NSP system developed according to RK4-method.

$$K_1 = \mathrm{h}(\eta - \eta N^n - \alpha N^n S^n - \kappa N^n P^n + \beta S^n + \epsilon P^n),$$

$$m_1 = \mathrm{h}(-\eta S^n + \alpha N^n S^n - \gamma S^n P^n - \beta S^n + \delta P^n),$$

$$n_1 = \mathrm{h}(-\eta P^n + \kappa N^n P^n + \gamma S^n P^n - \delta P^n - \epsilon P^n).$$

$$k_2 = \mathrm{h}(\eta - \eta(N^n + \tfrac{K_1}{2}) - \alpha(N^n + \tfrac{K_1}{2})(S^n + \tfrac{m_1}{2}) - \kappa(N^n + \tfrac{K_1}{2})(P^n + \tfrac{n_1}{2}) + \beta(S^n + \tfrac{m_1}{2}) + \epsilon(P^n + \tfrac{n_1}{2})),$$

$$m_2 = h(-\eta(S^n + \tfrac{m_1}{2}) + \alpha(N^n + \tfrac{K_1}{2})(S^n + \tfrac{m_1}{2}) - \beta(S^n + \tfrac{m_1}{2}) - \gamma(S^n + \tfrac{m_1}{2})(P^n + \tfrac{n_1}{2}) + \delta(P^n + \tfrac{n_1}{2})),$$

$$n_2 = h(-\eta(P^n + \tfrac{n_1}{2}) + \gamma(S^n + \tfrac{m_1}{2})(P^n + \tfrac{n_1}{2}) + \kappa(N^n + \tfrac{K_1}{2})(P^n + \tfrac{n_1}{2}) - \delta(P^n + \tfrac{n_1}{2}) - \epsilon(P^n + \tfrac{n_1}{2})).$$

$$k_3 = h(\eta - \eta(N^n + \tfrac{K_2}{2}) - \alpha(N^n + \tfrac{K_2}{2})(S^n + \tfrac{m_2}{2}) - \kappa(N^n + \tfrac{K_2}{2})(P^n + \tfrac{n_2}{2}) + \beta(S^n + \tfrac{m_2}{2}) + \epsilon(P^n + \tfrac{n_2}{2})),$$

$m_3 = h(-\eta(S^n + \frac{m_2}{2}) + \alpha(N^n + \frac{K_2}{2})(S^n + \frac{m_2}{2}) - \beta(S^n + \frac{m_2}{2}) - \gamma(S^n + \frac{m_2}{2})(P^n + \frac{n_2}{2}) + \delta(P^n + \frac{n_2}{2}))$,

$n_3 = h(-\eta(P^n + \frac{n_2}{2}) + \gamma(S^n + \frac{m_2}{2})(P^n + \frac{n_2}{2}) + \kappa(N^n + \frac{K_2}{2})(P^n + \frac{n_2}{2}) - \delta(P^n + \frac{n_2}{2}) - \epsilon(P^n + \frac{n_2}{2}))$.

$k_4 = h(\eta - \eta(N^n + K_3) - \alpha(N^n + K_3)(S^n + m_3) - \kappa(N^n + K_3)(P^n + n_3) + \beta(S^n + m_3) + \epsilon(P^n + n_3))$,

$m_4 = h(-\eta(S^n + m_3) + \alpha(N^n + K_3)(S^n + m_3) - \beta(S^n + m_3) - \gamma(S^n + m_3)(P^n + n_3) + \delta(P^n + n_3))$,

$n_4 = h(-\eta(P^n + n_3) + \gamma(S^n + m_3)(P^n + n_3) + \kappa(N^n + K_3)(P^n + n_3) - \delta(P^n + n_3) - \epsilon(P^n + n_3))$.

$$N_{n+1} = N_n + \frac{1}{6}(k_1 + 2k_2 + 2k_3 + k_4) \quad (20)$$

$$S_{n+1} = S_n + \frac{1}{6}(m_1 + 2m_2 + 2m_3 + m_4) \quad (21)$$

$$P_{n+1} = P_n + \frac{1}{6}(n_1 + 2n_2 + 2n_3 + n_4) \quad (22)$$

5.3 Non-Standard Finite Difference (NSFD) Scheme

$$N^{n+1} = \frac{N^n + h\eta + h\beta S^{n`} + h\epsilon P^n}{(1 + \mathrm{h}\eta + \mathrm{h}\alpha S^n + \mathrm{h}\kappa P^n)} \quad (23)$$

$$S^{n+1} = \frac{S^n + h\alpha N^n S^n + h\delta P^n}{(1 + \mathrm{h}\eta + \mathrm{h}\beta + \mathrm{h}\gamma P^n)} \quad (24)$$

$$P^{n+1} = \frac{P^n + h\gamma S^n P^n + h\kappa N^n P^n}{(1 + \mathrm{h}\eta + \mathrm{h}\delta + \mathrm{h}\epsilon)} \quad (25)$$

5.4 Stability Analysis of NSFD scheme

The stability of the [NSFD] schemes of the NSP model at disease-free equilibrium point (DFE).

$$\mathrm{F} = \frac{N + h\eta + h\beta S + h\epsilon P}{1 + \mathrm{h}\eta + \mathrm{h}\alpha \mathrm{S} + \mathrm{h}\kappa \mathrm{P}} \quad (26)$$

$$G = \frac{S + h\alpha NS + h\delta P}{1 + \mathrm{h}\eta + \mathrm{h}\beta + \mathrm{h}\gamma \mathrm{P}} \quad (27)$$

$$H = \frac{P + h\gamma SP + h\kappa NP}{1 + \mathrm{h}\eta + \mathrm{h}\delta + \mathrm{h}\epsilon} \quad (28)$$

Now J will be

$$J = \begin{bmatrix} F_N & F_S & F_P \\ G_N & G_S & G_P \\ H_N & H_S & H_P \end{bmatrix}$$

Now, the Jacobian matrix of equation

$$J = \begin{pmatrix} \frac{1}{1+h\eta} & \frac{h\beta - h\alpha}{1+h\eta} & \frac{h\eta - h\kappa}{1+h\eta} \\ 0 & \frac{1+h\alpha}{1+h\eta+h\beta} & \frac{h\delta}{1+h\eta+h\beta} \\ 0 & 0 & \frac{1+h\kappa}{1+h\eta+h\delta+h\epsilon} \end{pmatrix} \tag{29}$$

From above Jacobian matrix we obtain the eigenvalue.

$\lambda_1 = \frac{1}{1+h\eta}$, $\lambda_2 = \frac{1+h\alpha}{1+h\eta+h\beta}$ and $\lambda_3 = \frac{1+h\kappa}{1+h\eta+h\delta+h\epsilon}$.

So, this means that $\lambda_1 < 1,\ \lambda_2 < 1, \lambda_3 < 1,$ since all eigenvalues are at drinking free equilibrium points are less than one. Therefore, numerical scheme will converge to DFE if $R_0^S < 1$, $R_0^P < 1$. Hence DFE is stable.

6. Graphical Analysis of EULER, RK4 and NSFD Graphs at Different Points

The Euler and RK4 methods can produce divergent solutions at small step sizes due to the explicit nature of these methods which can accumulate errors in stiff or nonlinear systems. In contrast, the Non-Standard Finite Difference (NSFD) method is designed to preserve the qualitative properties of the system, such as positivity and boundedness, leading to convergent solutions even with small step sizes. This makes NSFD more suitable for handling complex epidemiological models, ensuring numerical stability.

The Euler and RK4 methods can produce divergent solutions at small step sizes due to the explicit nature of these methods as shown in Figures 2-4 which can accumulate errors in stiff or nonlinear systems. In contrast, the Non- Standard Finite Difference (NSFD) method is designed to preserve the qualitative properties of the system [19] as described in Figures 5-8 such as positivity and boundedness, leading to convergent solutions even with each step sizes. This makes NSFD more suitable for handling complex epidemiological models, ensuring numerical stability.

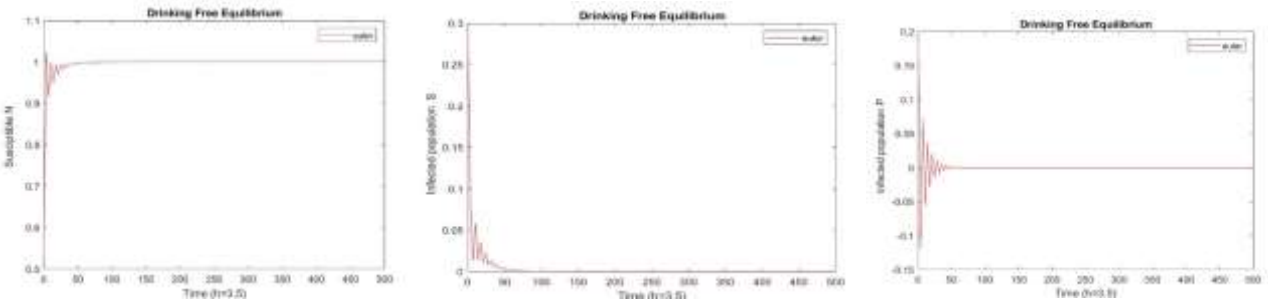

Fig. 2. Euler graphs at h = 3.5

Fig. 3. Behavior of Euler at h = 4.7

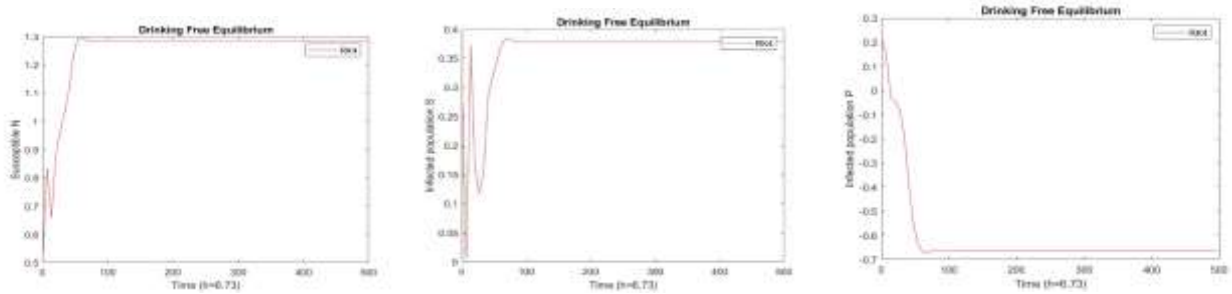

Fig. 4. Fourth Order Runge-Kutta at h = 6.73

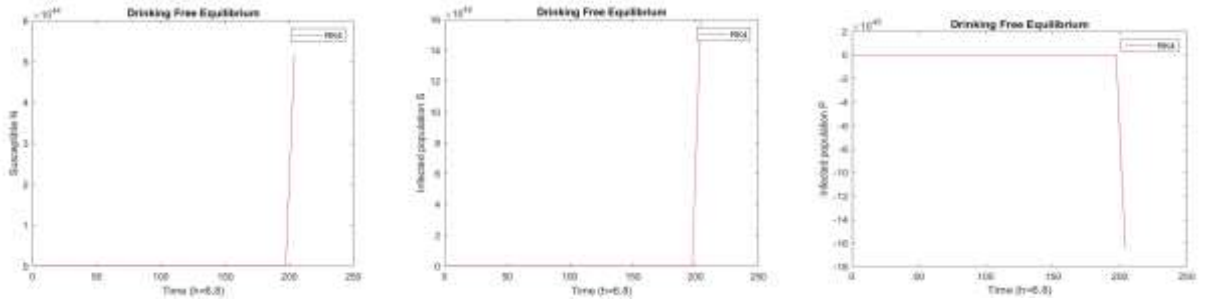

Fig. 5. Fourth Order Runge-Kutta at h = 6.8

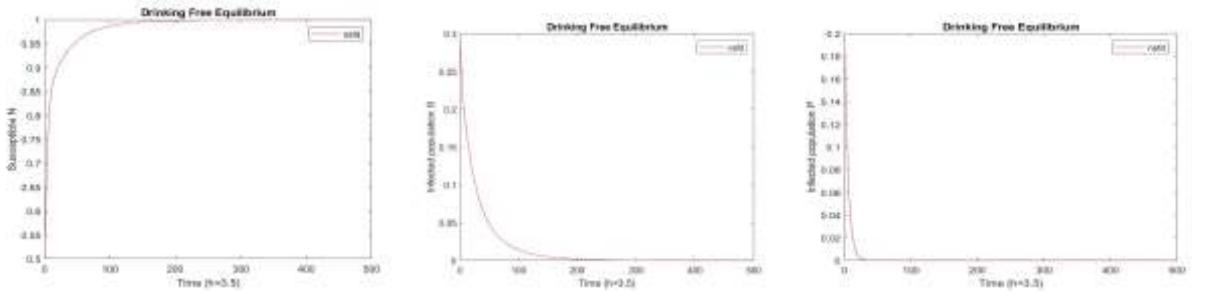

Fig. 6. Non-Standard Finite Difference at h = 3.5

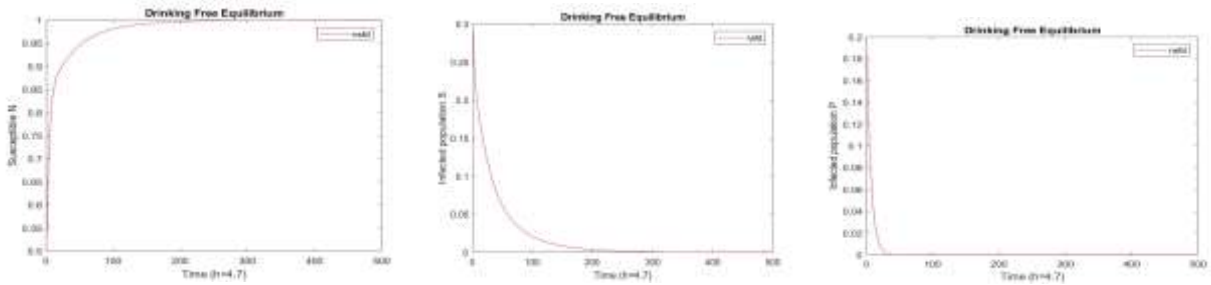

Fig. 7. Non-Standard Finite Difference at h = 4.7

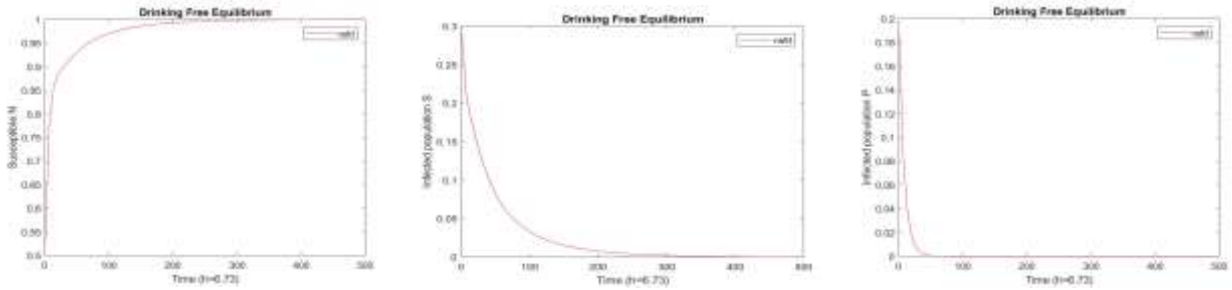

Fig. 8. Non-Standard Finite Difference at h = 6.73

7. Conclusions

In this research work, an accurate and reliable numerical solution of campus drinking epidemic model is provided with the help of nonstandard finite difference method. The proposed method preserves all important properties possessed by campus drinking epidemic model which shows the efficacy of this method. The comparison of proposed method is made with forward Euler method and RK-4 method. It is concluded from the simulations that both techniques are failed to give accurate solution even at very small step sizes. Moreover, equilibrium points of model are worked out and it is noted that the system has two steady states, one is disease free and other is endemic equilibrium. Stability at both the steady states is investigated. The role of basic reproduction numbers R_0^S and R_0^P are evaluated. It is mention able that the equilibrium point is locally asymptotically stable, when R_0^S < 1 and unstable when R_0^S > 1.

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