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A Modified Differential Transform Method (MDTM) of Solving Mathematical Model for Sorghum Pest Management

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Abstract

Pest management is essential to prevent devastating impact on economy, food security, social life, health and biodiversity. Insect pest control in stored grains such as sorghum has relied heavily on synthetic insecticides due to their instant effectiveness and ease of application. In this paper, Numerical scheme known as Modified Differential Transform Method (MDTM) was employed to solve mathematical model of sorghum pest management, and the results obtained was compared favorably with that of classical forth order Runge-Kutta scheme (RK45). In addition, biological interpretation of the results were presented, good strategies and proper implementation provide good results.

Keywords: Pest control; sorghum pest; mathematical model; Numerical scheme; Biological interpretation.

Introduction

Pest insects are responsible for substantial damages on crops all over the world. Their presence can account for high production losses having repercussions on trading and exports as well on the sustainability of small farmers whose incomes entirely rely on their production. Exotic pests can be particularly harmful as they can exhibit high invading potential due to the lack of natural enemies and their capacity to adapt to wide range of hosts and/or climate conditions. Therefore, pest management is essential to prevent devastating impact on economy, food security, social life, health and biodiversity.

Differential Transform Method (DTM) is a well-known semi analytical numerical method used by different researchers to solve linear and nonlinear partial

differential equation. It was first applied by Zhou (1986) during on electrical circuits [(Pekeret *et al.* (2011); Akinboro *et al.* (2014); Adio (2015); Onwubuoya *et al.* (2018)], it has since then grown over a period of time. Chen and Ho (1999) developed DTM for partial differential equation with two independent variables. Ayaz (2004) extended the two dimensional DTM into three dimensional DTM and used it to solve linear and nonlinear partial differential equation. Patel and Dhadiya (2022) considered the applications of DTM to real life problem. Patmaik *et al.* (2023) looked at the application of DTM for fluid and heat transfer. Despite numerous application of DTM in solving real life phenomena, it does not exhibit good approximation for larger domain. In order to improve the accuracy of differential transform method (DTM), it is usually modified either through the application of Pade approximation; Yousif *et al.* (2017), Laplace-Pade Resummation; Abdelhafez (2016), or multi step approach; Alawneh (2013), Zeb *et al.* (2014), Devi, (2021).

Pade approximants are extensively used to overcome the shortcomings of DTM, it increases the domain of convergence of truncated power series, Bararnia *et al.* (2012). It has been applied to improve the accuracy of truncated power obtained by power series method (PSM), Vazquez-Leal and Guerrero (2014), Vazquez-Leal *et al.* (2014), Homotopy perturbation method (HPM), Bararnia *et al.* (2012), Adomian decomposition method (ADM) Wang *et al.* (2011), and Differential transform method (DTM), Esmaeel *et al.* (2010) among others. Pade approximants are widely used in computer calculations due to the fact that it often gives better approximation of the function than truncating its power series, Peker *et al.* (2011). The Pade approximation of a function $f(t)$ of order (N/M) is defined by Mohamed and Torky (2013), Vazquez-Leal and Guerrero (2014).

This paper presents modified differential transform method (MDTM); an hybrid scheme for improving DTM truncated series solutions in convergence rate, which contain DTM, Laplace transform and Padé's approximant which sometimes referred to as Laplace-Pade differential transform method (LPDTM) for analysis on the sorghum production model. The Padé's approximant shall be used to truncate the series solution emerging from DTM and the results of the schemes will be compared with classical Fourth Order Runge-Kutta Scheme (RK45).

Literature Review

One of the most important cereal grain crops worldwide is sorghum, *Sorghum bicolor* L. Moench (*Poaceae*). The grain is used for food, preparation of beverages and biofuel, while the stalks are used for animal feed, fuel, and fence construction in some rural areas. Over 90% of sorghum grain is for food, being a staple diet in parts of Asia and Africa. In the United States, sorghum is mainly used for ethanol production and as livestock feed. The increase in use as a food crop in high-income countries is based on studies showing the anti-inflammatory and cholesterol-lowering properties of the grain and its byproducts.

Many researcher had worked on improving the accuracy of differential transform method (DTM), Marwan *et al.* (2012) proposed a combined form of the Laplace

transform method (LTM) with the differential transform method (DTM) to solve non-homogeneous linear partial differential equations (PDEs). The combined method is capable of handling non-homogeneous linear partial differential equations with variable coefficient. Laplace transform was used to overcome the deficiency that is caused by unsatisfied boundary conditions in using differential transform method. Illustrative examples were examined to support the proposed analysis. It was established that the exact solution were obtained even with just the two first terms of the Laplace differential transform method (LDTM) solutions, which indicates that the proposed method (LDTM) need less computational work compared with the standard differential transform method (DTM) and it can be applied for other non-linear partial differential equations (PDEs).

Hooman and Hossein (2013) stated that differential transform method, although being powerful yet suffers from a drawback which is the lack of systematic methodology for derivation of the differential transform for non-linear expressions, it was shown that this defect can be overcome with the help of the Adomian Polynomials perfectly. In addition, the transformed analogues of some frequent nonlinearities were presented. A simple general routine involving the Adomian polynomials was proposed to overcome the defect.

Brahim *et al.* (2014) applied the application of the differential transform method (DTM) to the model of pollution for a system of three lakes interconnected by channels. Three input models (periodic, exponentially decaying and linear) were solved to show that DTM can provide analytic solutions of pollution model in convergent series form. Post treatment of the power series solutions with the laplace-Paderesummation method was presented as a useful strategy to extend the domain of convergence of the approximate solutions.

Akinyemi *et al.* (2017) considered the analytic solution of a time fractional enzymes kinetic model, using differential transform method and pade approximant called DTM-Pade. It was established that the proposed method avoids large computational work, is reliable and efficient and it was concluded that the method is effective, powerful and accurate to solve a time fractional enzymes kinetic model. DTM-Pade not only work for system of non-linear differential equation with fractional derivatives but also for integers when compared with existing methods.

Onwubuoya *et al.* (2018) extended the application of a modified differential transform method by providing an approximate analytic solutions to a model that describes the spread of computer virus over a network. Hybrid technique that combines differential transform method with a post treatment of laplace-Paderesummation method was proposed. The accuracy of the solutions generated by the proposed method was established when compared with those of Runge-Kutta fourth order method. The proposed method (LPDTM) improves the solution obtained by DTM by increasing its domain of convergence and it requires no discretization, perturbation or linearization and can be used to solve other non-linear problems in science and engineering.

Harir *et al.* (2020) presented a study to analyze and find a solution for the model of non-linear ordinary differential equations (ODEs) describing the so-called corona virus

(COVID-19), a very deadly virus. The mathematical method based on four non-linear ODEs was presented, and its corresponding numerical results were studied by applying the variational iteration method and differential transform method. It was conclude that these two methods are effective in providing analytic form solutions for such problems and the two method are in excellent agreement.

Moosavi-Noori and Nasir (2020) employed an hybrid technique for improving the differential transform method (DTM), the modified differential transform method (MDTM) which is a combination of differential transform method, Laplace transform and the Pade approximant. It was employed for the first time to ascertain exact solution of linear and non-linear pantograph type of differential and volterraintegro differential equations (DEs and VIDEs) with proportional delay. It solves the problem directly without requiring large computational work, perturbation or linearization and it enlarges the domain of convergence and leads to exact solutions effectively.

Yadav and Methi (2024), considered new numerical technique to obtain numerical solution of a system of fractional delay differential equations.

MODEL FORMULATION

Seed-Germination-Cropping-Harvest (SGCH) Model of Sorghum

A deterministic dynamical and Vector-Host model is considered for optimality. The host is seeds/plant while vector is the insect population. The seed/plant population is further classified into Seed $S_c(t)$, Germination $G(t)$, Crop $C(t)$ and Harvest (H), while the insect population is categorized into two: the beneficial insect population, $P_v(t)$, which could there after become harmful to sorghum seeds and pest population $I_v(t)$ are right from time, harmful to sorghum. The model shows different growth stages from seedling to the harvest time as can be seen in the diagram,

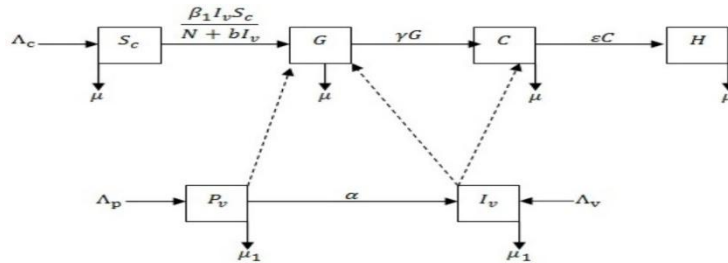


Figure 1: Sorghum Production Flow Diagram

Λ_c is the quantity of replenishing seed for plantation, $\frac{\beta_1 I_v S_v}{N + b I_v}$ is the force of infection on seed by pest and it is in mathematical epidemiology referred to as saturated incidence function. γ and μ are the removal rate from germination class where γ is the proportion that produces crop and μ denote natural bad seeds/seedlings failed to develop. ϵC is the quantity of matured crop that farmers will be able to harvest.

Associated Assumption of the Model

The model assumptions are as follow:

- i. The growth and development stages of sorghum was used to formulate the model (i.e. from seedling development, panicle initiation, and reproduction stage).
- ii. A vector-Host mathematical modelling of epidemiology is used to modeled the four stages of development of sorghum and the stages are examined in an homogeneous form (i.e vectors can attack every stages of the sorghum).
- iii. The Model equations are considered as a prey-predator population, where prey is the seed and predator is the pest.
- iv. In the absent of pest, there exist no germination, this means, no matter how good and fertile the land is, it must contain pest otherwise, there will be no germination.
- v. The seeds are systemic (i.e the seeds are all in good condition and preserved with chemical).
- vi. Some pest have negative economic importance on sorghum while some have both positive and negative economic importance.

Model Equations

In view of the aforementioned assumptions and from the model diagram in Figure 1, the following first order nonlinear system of autonomous ordinary differential equations were derived.

$$\begin{aligned}
 \frac{dS_c}{dt} &= \Lambda_c - \mu S_c - \frac{\beta_1 I_v S_c}{N + b I_v} \\
 \frac{dG}{dt} &= \frac{\beta_1 I_v S_c}{N + b I_v} - (\gamma + \mu) G \\
 \frac{dC}{dt} &= \gamma G - (\varepsilon + \mu) C \\
 \frac{dH}{dt} &= \varepsilon C - \mu H \\
 \frac{dP_v}{dt} &= \Lambda_p - \mu_1 P_v - \alpha P_v \\
 \frac{dI_v}{dt} &= \Lambda_v + \alpha P_v - \mu_1 I_v
 \end{aligned} \quad (1)$$

Table 1: Description of Parameters and Variables. The values are hypothetical

Parameter/ variables	Description	Value
Λ_c	Proportion of seed replenishing	50
β_1	Probability of successful harm on sorghum seed.	[0.1]
N	Total number of seed	80
γ	Percentage of germinating seed producing crop.	0.6
δ	Fraction of crop which successfully harvest.	0.7
μ	Amount of natural bad seed/natural loss on seeds.	0.1
Λ_p	Relative recruitment rate for insect with both negative and positive impact.	15
Λ_v	Relative recruitment rate for insect with negative impact.	50
bI_v	Proportion of pest interacting/having contact with seeds.	5.1421
μ_1	Natural death rate for insects.	0.1
α	Progressive of destructive insects.	0.02
S_c	Sorghum Seeds	50
G	Germination Stage	35
C	Cropping	30
H	Harvest	20
P_v	Beneficial Pest	7.5
I_v	Pest	10.285

Numerical Implications

Differential Transform Method and Variational Iteration Method are efficient and reliable for solving system of Ordinary Differential Equation (ODE) thus, it was employed to investigate numerical solution of the proposed model.

Modified Differential Transform Method (MDTM)

Consider the general concept of Differential Transform Method (DTM), an arbitrary function $f(x)$ can be expanded in Taylor series about a point $x=0$ as

$$f(x) = \sum_{k=0}^{\infty} \frac{x^k}{k!} \left[\frac{d^k f}{dx^k} \right]_{x=0} \quad (2)$$

The differential transformation of $f(x)$ is defined as

$$F(x) = \frac{1}{k!} \left[\frac{d^k f}{dx^k} \right]_{x=0} \quad (3)$$

Then the inverse differential transformation is

$$f(x) = \sum_{k=0}^{\infty} x^k F(x) \quad (4)$$

The fundamental mathematical operations performed by differential transform method are listed in Table 2

Table 2: Fundamental Mathematical Operators of DTM. Amit *et al.* (2017)

Original Function	Transformed Function
$y(x) = g(x) \pm h(x)$	$Y(k) = G(k) \pm H(k)$
$y(x) = \alpha g(x)$	$Y(k) = \alpha G(k)$
$y(x) = \frac{dg(x)}{dx}$	$Y(k) = (k+1)G(k+1)$
$y(x) = \frac{d^2 g(x)}{dx^2}$	$Y(k) = (k+1)(k+2)G(k+2)$
$y(x) = \frac{d^m g(x)}{dx^m}$	$Y(k) = (k+1)(k+2) \dots (k+m)G(k+m)$
$y(x) = 1$	$Y(k) = \delta(k)$
$y(x) = x$	$Y(k) = \delta(k-1)$
$y(x) = g(x)h(x)$	$Y(k) = \sum_{m=0}^k H(m)G(k-m)$
$y(x) = e^{(\lambda x)}$	$Y(k) = \frac{\lambda^k}{k!}$
$y(x) = (1+x)^m$	$Y(k) = \frac{m(m-1) \dots (m-k+1)}{k!}$
$y(x) = x^m$	$Y(k) = \delta(k-m); \delta(k-m) = \begin{cases} 1 & k = a \\ 0 & \text{else} \end{cases}$
$y(x) = \sin(\alpha x + \beta)$	$Y(k) = \frac{\alpha^k}{k!} \sin\left(\frac{\pi}{2}k + \beta\right)$
$y(x) = \cos(\alpha x + \beta)$	$Y(k) = \frac{\alpha^k}{k!} \cos\left(\frac{\pi}{2}k + \beta\right)$

$y(x) = \tan(\alpha x + \beta)$	$Y(k) = \sum_{i=0}^k \frac{(k-i)!}{i!} \alpha^{2i-k} \frac{\sin\left(\frac{\pi}{2}i + \beta\right)}{\cos\left(\frac{\pi}{2}(k-i) + \beta\right)}$
$y(x) = \sin h(ax)$	$Y(k) = \frac{1}{2k!} (a^k - (-a^k))$
$y(x) = \cos h(ax)$	$Y(k) = \frac{1}{2k!} (a^k + (-a^k))$
$y(x) = \tan h(ax)$	$Y(k) = \sum_{i=0}^k \frac{(k-i)!}{i!} \frac{\frac{1}{2k!} (a^k - (-a^k))}{\frac{1}{2k!} (a^{k-i} + (-a^{k-i}))}$

Application of Differential-Laplace Transform Methods (DLTM)

Using the fundamental operators in Table 2 on (1), the following transform and recurrence relation were obtained as:

$$\begin{aligned}
 S_c(k+1) &= \frac{1}{k+1} \left[\Lambda_c - \beta_1 \sum_{m=0}^k I_v(m) S_c(k-m) - \mu S_c(k) \right] \\
 G(k+1) &= \frac{1}{k+1} \left[\beta_1 \sum_{m=0}^k I_v(m) S_c(k-m) - \gamma G(k) - \mu G(k) \right] \\
 C(k+1) &= \frac{1}{k+1} [\gamma G(k) - (\mu + \delta) C(k)] \\
 H(k+1) &= \frac{1}{k+1} [\delta C(k) - \mu H(k)] \\
 P_v(k+1) &= \frac{1}{k+1} [\Lambda_v - \mu_1 P_v(k) - \alpha P_v(k)] \\
 I_v(k+1) &= \frac{1}{k+1} [\Lambda_v + \alpha P_v(k) - \mu_1 I_v(k)]
 \end{aligned} \tag{5}$$

Implementing this on Maple 18, the following series solutions were obtained

$$\begin{aligned}
 S_c(t) &= 50 - 314.9750t + 995.2037560t^2 - 1766.201940t^3 + 1653.690632t^4 - 149.0587902t^5 \\
 &\quad - 1645.471419t^6 + 1948.932183t^7 - 518.3100158t^8 - 992.9859863t^9 + 1128.152579t^{10} + \dots \\
 &\quad - 3.684623416 * 10^{(-179)} t^{298} + 6.794110348 * 10^{(-179)} t^{299} - 4.923895153 * 10^{(-180)} t^{300} \\
 G(t) &= 35 + 335.4750t - 1096.871256t^2 + 1988.965107t^3 - 1957.604478t^4 + 390.0496046t^5
 \end{aligned}$$

$$\begin{aligned}
 &+1602.449945t^6 - 2085.670443t^7 + 676.4445272t^8 + 946.1326344t^9 - 1184.452003t^{10} + \dots \\
 &+3.141922473*10^{(-179)}t^{298} - 6.800233706*10^{(-179)}t^{299} + 5.059920240*10^{(-180)}t^{300} \\
 C(t) = &30 - 3.0t + 101.8425000t^2 - 246.5322512t^3 + 347.6512162t^4 - 290.5367320t^5 \\
 &+77.74319140t^6 + 128.4679163t^7 - 169.2720749t^8 + 60.14270847t^9 + 51.95654138t^{10} \\
 &+5.47707589610^{(-180)}t^{298} + 4.839422783*10^{(-182)}t^{299} - 1.361337254*10^{(-181)}t^{300} \\
 H(t) = &20 + 19.0t - 2.00t^2 + 23.82991667*t^3 - 43.7388919*t^4 + 49.5459481*t^5 - 34.721718*t^6 \\
 &+8.270343681*t^7 + 11.13756338*t^8 - 13.28935652*t^9 + 4.342883158*t^{10} + \dots \\
 &-5.006648050*10^{(-182)}t^{298} + 1.283933035*10^{(-182)}t^{299} + 1.086400881*10^{(-184)}t^{300} \\
 P_v(t) = &7.5 + 15.600t + 0.62400t^2 + 0.01664t^3 + 0.0003328t^4 + 0.0000053248t^5 + 7.099733*10^{-8}t^6 \\
 &+8.11398095*10^{-10}t^7 + 8.11398095*10^{-12}t^8 + 7.2124275*10^{-14}t^9 + 5.7699420*10^{-16}t^{10} + \dots \\
 &+7.548143282*10^{-935}t^{298} + 2.019570109*10^{-938}t^{299} + 5.385520290*10^{-942}t^{300} \\
 I_v(t) = &10.285 + 8.8215t - .5970750t^2 + 0.1574250e^{-1}t^3 - 0.4767625e^{-1}t^4 + 0.8204050e^{-1}t^5 \\
 &-1.544835*10^{-7}t^6 + 2.0040576*10^{-9}t^7 - 2.707922*10^{-11}t^8 + 2.82849*10^{-13}t^9 - 2.9727396*10^{-15}t^{10} + \dots \\
 &-3.093433462*10^{(-906)}t^{298} + 1.034593131*10^{(-909)}t^{299} - 3.448643770*10^{(-913)}t^{300} \quad (6)
 \end{aligned}$$

The Laplace transform result obtained from (6) is

$$\begin{aligned}
 S_c(s) &= \frac{50.0000}{s^1} - \frac{314.9750000}{s^2} + \frac{(1990.407512)}{s^3} + \dots - \frac{1.506995101*10^{435}}{s^{301}} \\
 G(s) &= \frac{35.0000}{s^1} + \frac{335.4750000}{s^2} - \frac{2193.742512}{s^3} + \dots + \frac{1.548626601*10^{435}}{s^{301}} \\
 C(s) &= \frac{30.0000}{s^1} - \frac{3.0000}{s^2} + \frac{203.6850000}{s^3} + \dots - \frac{4.166474932*10^{433}}{s^{301}} \\
 H(s) &= \frac{20.0000}{s^1} + \frac{19.0000}{s^2} - \frac{4.0000}{s^3} + \dots + \frac{3.325011509*10^{430}}{s^{301}} \\
 P(s) &= \frac{7.5000000000}{s^1} + \frac{15.600000000}{s^2} + \frac{1.248000000}{s^3} + \dots + \frac{1.648278942*10^{-327}}{s^{301}} \\
 I_v(s) &= \frac{10.285000000}{s^1} + \frac{8.821500000}{s^2} - \frac{1.194150000}{s^3} + \dots - \frac{1.055483333*10^{-298}}{s^{301}} \quad (7)
 \end{aligned}$$

After simplification and truncation of the series using Padé's approximant, the following result was obtained

$$\begin{aligned}
 S_1(t) &= \frac{49.99999997t + 2003.805t^2 + 4552.6810t^3 + 6399.79072t^4 + 6096476.808t^5 + 38261404.29t^6 + 152994027.5t^7 + 320482311.0t^8 + 263240752.3t^9 - 78768467.99t^{10}}{0.9999999996 + 46.3759886t + 1162.87114t^2 + 18490.90561t^3 + 201156.5162t^4 + 1506334.755t^5 + 7577639.594t^6 + 23635410.60t^7 + 37567234.22t^8 + 12790199.14t^9 - 9401166.385t^{10}} \\
 G(t) &= \frac{35.000000001t + 1465.872963t^2 + 25725.40083t^3 + 246488.8028t^4 + 1259178.500t^5 + 2764997.584t^6}{1.0 + 32.29708465t + 488.1222332t^2 + 4047.248195t^3 + 18108.52317t^4 + 34688.69966t^5 + 1628.016909t^6} \\
 C(t) &= \frac{30.0t + 869.8251356t^2 + 1291.641906t^3 + 108864.5232t^4 + 541505.4835t^5 + 1418037.187t^6 + 1518140.618t^7}{1.0 + 29.09417118t + 426.667219t^2 + 3523.255739t^3 + 16662.06060t^4 + 39120.86117t^5 + 28389.70961t^6 - 1133.949569t^7} \\
 H(t) &= \frac{20.0t + 574.5137372t^2 + 8941.472453t^3 + 81452.26454t^4 + 460185.2374t^5 + 1497565.154t^6 + 2368544.944t^7 + 1061137.580t^8}{1.0 + 27.77568686t + 420.8867202t^2 + 3671.177002t^3 + 19459.74005t^4 + 55277.40908t^5 + 58644.39186t^6 - 30513.56173t^7 + 21324.61700t^8} \\
 P_1(t) &= \frac{7.499999998t + 14.91479057t^2 - 0.04412042005t^3 + 0.2491928798t^4 - 0.004783156049t^5 + 0.004008577886t^6 - 0.00006173523562t^7 + 0.00004063474436t^8}{0.9999999998 - 0.0913612566t + 0.0177486911t^2 - 0.001801047122t^3 + 0.0003062827227t^4 - 0.00002706806286t^5 + 0.000003146596863t^6 - 0.0000002167186366t^7 + 3.673211370 \times 10^{-10}t^8} \\
 I_1(t) &= \frac{10.28500000t + 10.58123529t^2 + 0.2661062775t^3 - 0.1454086545t^4 + 0.01639390571t^5 - 0.002025305734t^6}{0.9999999999 + 0.1710972568t - 0.004771820450t^2 + 0.0006365336655t^3 + 0.00003517386523t^4 - 0.00001472949626t^5 + 1.131017540 \times 10^{-11}t^6}
 \end{aligned}$$

After some transformation and simplification, the following is inverse laplace transform for the solution of Seedling, Germination, Cropping, and Harvesting stage, and Anthropoids and pest population respectively

$$\begin{aligned}
 S(t) = & (-0.0007790138620 - 0.0006688807779i)e^{(-6.428706237 - 11.38530499i)t} \\
 & - (0.0007790138620 - 0.0006688807779i)e^{(-6.428706237 + 11.38530499i)t} \\
 & - (0.08856214519 + 0.1549728456i)e^{(-5.908311766 - 7.939380619i)t} \\
 & - (0.08856214519 - 0.1549728456i)e^{(-5.908311766 + 7.939380619i)t} \\
 & - (0.04223945613 + 4.256055054i)e^{(-5.447273251 - 4.857366564i)t} \\
 & - (0.04223945613 - 4.256055054i)e^{(-5.447273251 + 4.857366564i)t} \\
 & + (20.45481147 - 17.26934252i)e^{(-5.004052899 - 1.707638721i)t} \\
 & + (20.45481147 + 17.26934252i)e^{(-5.004052899 + 1.707638721i)t} \\
 & + 8.252983486e^{-1.131992598t} + 1.100554806e^{0.3330820275t}
 \end{aligned} \tag{9}$$

$$\begin{aligned}
 G(t) = & (-0.1633751212 + 0.2800847317i)e^{(-6.686724911 - 7.561075094i)t} \\
 & - (0.1633751212 + 0.2800847317i)e^{(-6.686724911 + 7.561075094i)t} \\
 & - (7.659373591 - 5.793353260i)e^{(-6.364839720 - 3.675838041i)t} \\
 & - (7.659373591 + 5.793353260i)e^{(-6.364839720 + 3.675838041i)t} \\
 & - 31.38887439e^{-6.145826920t} + 82.03437182e^{-0.04812846857t}
 \end{aligned} \tag{10}$$

$$\begin{aligned}
 C(t) = & (-0.01235647871 - 0.001871508503i)e^{(-6.058503727 - 8.072812778i)t} \\
 & - (0.01235647871 - 0.001871508503i)e^{(-6.058503727 + 8.072812778i)t} \\
 & - (0.5283260020 + 0.7653000535i)e^{(-5.422257003 - 4.134583387i)t} \\
 & - (0.5283260020 - 0.7653000535i)e^{(-5.422257003 + 4.134583387i)t} \\
 & + 8.084649834e^{-4.876154704t} - 27.04186248e^{-1.294422748t} + 50.03857760e^{0.03792773236t}
 \end{aligned} \tag{11}$$

$$\begin{aligned}
 H(t) = & (-0.0001062056501 - 0.0004042597026i)e^{(-5.567746903 - 8.699991085i)t} \\
 & - (0.0001062056501 - 0.0004042597026i)e^{(-5.567746903 + 8.699991085i)t} \\
 & + (0.03691361152 - 0.04617396200i)e^{(-4.758579709 - 4.989632812i)t} \\
 & + (0.03691361152 + 0.04617396200i)e^{(-4.758579709 + 4.989632812i)t}
 \end{aligned} \tag{12}$$

$$\begin{aligned}
 & + (0.8185995598 + 1.373979082i)e^{(-3.825054031 - 1.651567759i)t} \\
 & + (0.8185995598 - 1.373979082i)e^{(-3.825054031 + 1.651567759i)t} \\
 & + (9.144593033 + 20.11635162i)e^{(0.2635372121 - 0.4156320599i)t} \\
 & + (9.144593033 - 20.11635162i)e^{(0.2635372121 + 0.4156320599i)t} \\
 P(t) = & (0.00000007828955565 - 0.000000004434661733i)e^{(-0.07770523221 - 0.09725722005i)t} \\
 & + (0.00000007828955565 + 0.000000004434661733i)e^{(-0.07770523221 + 0.09725722005i)t} \\
 & - 187.5000088e^{0.000000001694921820t} + (0.0000003031474883 - 0.0000002184211241i)x \\
 & e^{(0.002428145464 - 0.1121806160i)t} + (0.0000003031474883 + 0.0000002184211241i)x \\
 & e^{(0.002428145464 + 0.1121806160i)t} + 195.0000079e^{0.07999999906t} + (0.00000006453649078 \\
 & - 0.0000004572566614i)e^{(0.08095771469 - 0.08561509527i)t} \\
 & + (0.00000006453649078 + 0.0000004572566614i)e^{(0.08095771469 + 0.08561509527i)t} \\
 I_v(t) = & -0.00000002889710896e^{-0.1973339734t} - 105.5483325e^{-0.1000000003t} \\
 & + 137.4999995e^{0.0000000007678589424t} + (0.000000007154189030 - 0.0000001238507454i)x \\
 & e^{(0.02311835815 - 0.09378619796i)t} + (0.000000007154189030 + 0.0000001238507454i)x \\
 & e^{(0.02311835815 + 0.09378619796i)t} - 21.66666704e^{0.07999999979t}
 \end{aligned} \tag{13}$$

$$\begin{aligned}
 & + (0.000000007154189030 - 0.0000001238507454i)x \\
 & e^{(0.02311835815 - 0.09378619796i)t} + (0.000000007154189030 + 0.0000001238507454i)x \\
 & e^{(0.02311835815 + 0.09378619796i)t} - 21.66666704e^{0.07999999979t}
 \end{aligned} \tag{14}$$

Numerical simulation was performed and the results of MDTM are plotted in comparison with the classical fourth order Runge-Kuta, the following were achieved using data presented in Table 3.

Table 3: Numerical Solution obtained by Runge-Kutta Forth Order}

$[t, C(t), I_v(t), G(t), H(t), P_v(t), S_c(t)]$						
0.0	10.0	10.285	35.0	20.0	7.5	50.0
1.0	36.191454212 9022010	19.822910823 5110694	78.352445964 6860976	34.226213873 2162171	21.47999224 08770989	2.8674800053 5067726
2.0	47.872383789 8386435	29.158973853 1856746	75.351001880 5088890	59.643624226 9047341	34.51485060 86040143	1.9216501677 2405894
3.0	52.182913311 2573729	38.285231154 1689119	73.581498288 6683708	87.563626185 4915716	46.66847199 40221981	1.4569472521 2111198
4.0	53.587230479 0154814	47.195316441 2388659	72.599099500 9479860	114.56087265 8183740	58.00043346 32628300	1.1795796381 3178179
5.0	53.925613422 9545552	55.884296086 6284457	72.062615084 4050244	139.50227751 7961772	68.56628430 45951433	0.9951899853 15175774
6.0	53.918141129 6454343	64.348523382 8654231	71.770169158 4464498	162.15540535 0592645	78.41781833 11014720	0.8638044651 15942666
7.0	53.827597656 9081394	72.585505042 2833301	71.609764297 0011350	182.61177262 1785008	87.60332777 40090597	0.7655234646 13955484
8.0	53.738815460 6516295	80.593778989 6623826	71.520716909 8317479	201.05950627 1288512	96.16784001 12638201	0.6893101730 98751755
9.0	53.671994830 4011751	88.372802573 9197989	71.470431461 5030540	217.69970876 1811138	104.1533382 91788970	0.6285459461 54955404
10.0	53.626598215 0580811	95.922850389 2545916	71.441418228 5692902	232.71937981 0704766	111.5989675 37420236	0.5790188946 63807404
11.0	53.597216607 4198526	103.24492095 6018362	71.424254677 0075108	246.28524108 1913114	118.5412262 31357825	0.5379204099 00292884
12.0	53.578624628 9280754	110.34065156 7148171	71.413822097 2215834	258.54447627 6963906	125.0141453 33773570	0.5033054106 89940169
13.0	53.566946821 3401458	117.21224065 7572295	71.407305191 1932168	269.62722362 1210362	131.0494551 01617127	0.4737840481 62691540
14.0	53.559594776 8235945	123.86237710 1855827	71.403129824 3170767	279.64910753 9090835	136.6767406 30372564	0.4483367422 51477183
15.0	53.554926902 7432118	130.29417588 9764460	71.400397522 8708612	288.71336535 3526854	141.9235868 80230090	0.4261985637 14940915
16.0	53.551929016 7076250	136.51111967 0623003	71.398582732 6773972	296.91254441 9359392	146.8157138 97593980	0.4067844033 14657206
17.0	53.549981307 0220441	142.51700569 5567718	71.397369354 1093155	304.32985350 9998486	151.3771028 94778290	0.3896390685 67149138
18.0	53.548705006 1568368	148.31589772 2246802	71.396561447 1481871	311.04025845 3980130	155.6301138 05939999	0.3744031274 02965830
19.0	53.547866742 6063097	153.91208247 9386243	71.396033722 3760320	317.11139214 4190006	159.5955948 95499772	0.3607889901 11196362
20.0	53.547320593 2738949	159.31003031 9150570	71.395703586 4570031	322.60432795 5218082	163.2929849 56359248	0.3485638189 52239646

Discussion of Result

Three (3) numerical schemes (i.e. Runge-Kutta, DTM, and Modified DTM also known as Laplace-DTM) were employed to analyze the dynamics of proposed mathematical model of sorghum. DTM which is a semi-analytic scheme was employed to solve the model equations, and findings revealed that the method can only be accurate for small computation interval. The choice for the use of semi-analytical method was due to the fact that sorghum farming is a real life project and most real life problems do not have exact solution. Thus, to improve on the result of DTM, prompted the consideration for another scheme; Laplace-DTM.

The results presented in Tables 4-7 and Figures 2 to 12 show that DTM lost consistency compare to the remaining two methods. For instance, the initial point in Table 4 for $t=0$ to $t=10$, swiftly alternation is observed with wide gap. Also, from Table 5, at $t=3$, the germination dynamics changes direction from positive magnitude to a large negative value and within two variation in time t , returned to largely positive value. To obtain result with negative value for a real life model, in an ideal situation is not realistic and also contradicted the assumption of formulation of the model for which all the state variables assumed non-negative. The results from all the Tables indicates that Laplace-DTM indeed was used to improve the results of DTM. Also, the result shows that RK45 performs excellently when compare with MDTM.

Table 4: Numerical Scheme comparison (i.e. Runge-Kutta forth order-RK45, DTM and Laplace-DTM) for Susceptible Seeds to Seedlings ($S_c(t)$)

T	RK45	DTM	Laplace-DTM
0	50.0	50.0	50.0
1	2.86748000535067726	3.061433818	3.048547900
2	1.92165016772405894	1.500000	1.950415041
3	1.45694725212111198	1.0785204×10^9	1.252627308
4	1.17957963813178179	$4.999999994 \times 10^{23}$	0.8044835840
5	0.995189985315175774	$-3.114481516 \times 10^{46}$	0.5166691110
6	0.863804465115942666	$-1.904084581 \times 10^{70}$	0.3318240114
7	0.765523464613955484	$-2.423897391 \times 10^{90}$	0.2131096524
8	0.689310173098751755	$-6.247805993 \times 10^{107}$	0.1368669005
9	0.628545946154955404	$-1.413780268 \times 10^{123}$	0.08790098529
10	0.579018894663807404	$-7.644249546 \times 10^{136}$	$0.5645326370e^{-1}$

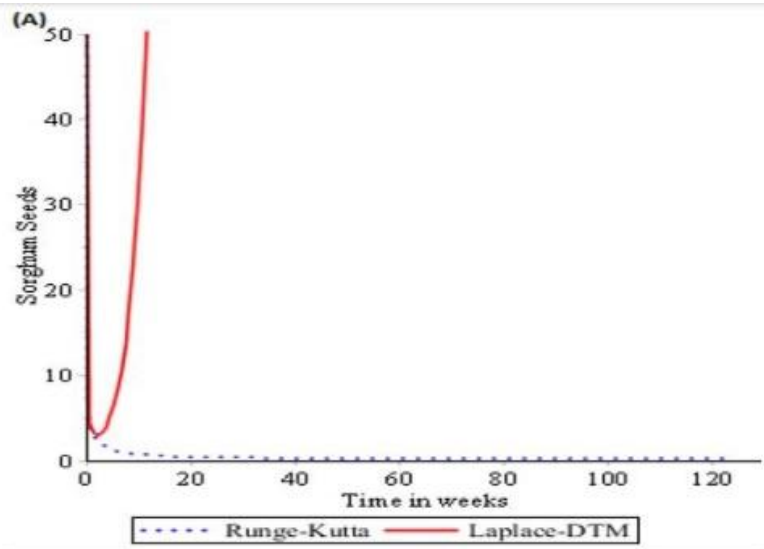


Figure 2: Comparison of Runge-Kutta forth order-RK45, DTM and Laplace-DTM for Suscep-
tible Seeds to Seedlings

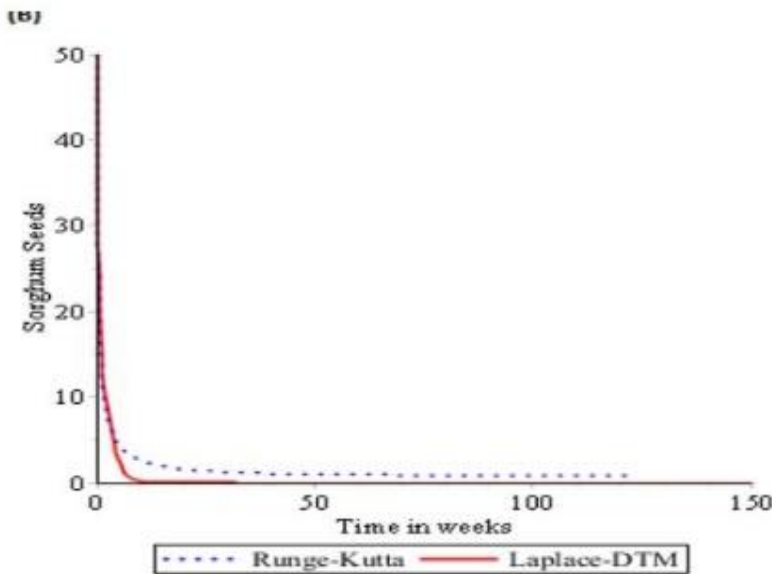


Figure 3: Comparison of Runge-Kutta forth order-RK45, DTM and Laplace-DTM for Suscep-
tible Seeds to Seedlings

Table 5: Numerical Scheme comparison (i.e. Runge-Kutta forth order-RK45, DTM and Laplace-DTM) for Germination stage ($G(t)$)

T	RK45	DTM	Laplace-DTM
0	35.000000	35.0000000	35.00000002
1	78.3524459646860976	78.22193800	78.15589146
2	75.3510018805088890	74.13120000	74.17805355
3	73.5814982886683708	-2.88686×10^9	70.39831159
4	72.5990995009479860	-1.695804×10^{22}	66.81116553
5	72.0626150844050244	3.1144460×10^{46}	63.40680249
6	71.7701691584464498	$1.902462135 \times 10^{70}$	60.17590878
7	71.6097642970011350	$2.420042558 \times 10^{90}$	57.10964526
8	71.5207169098317479	$6.233785030 \times 10^{107}$	54.19962320
9	71.4704314615030540	$1.409783318 \times 10^{123}$	51.43788133
10	71.4414182285692902	$7.618639699 \times 10^{136}$	48.81686401

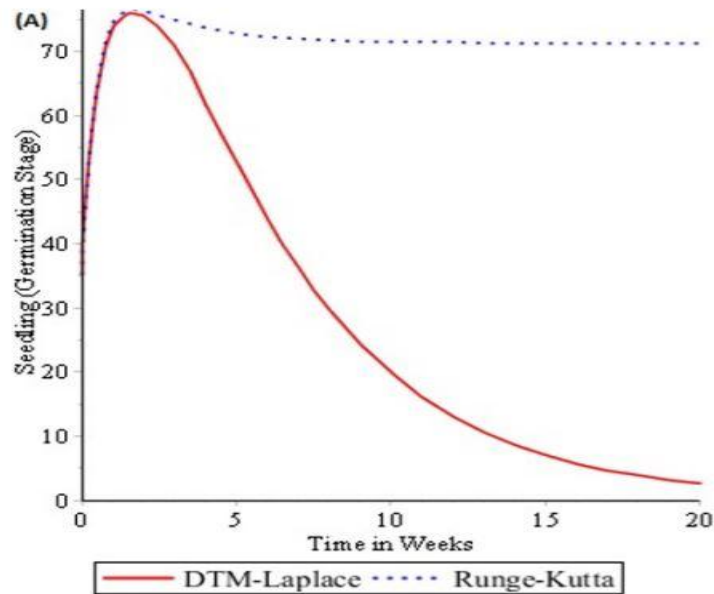


Figure 4: Comparison of Runge-Kutta forth order-RK45, DTM and Laplace-DTM for Susceptible Seeds to Seedlings

Table 6: Numerical Scheme comparison (i.e. Runge-Kutta forth order-RK45, DTM and Laplace-DTM) for Cropping ($C(t)$)

T	RK45	DTM	Laplace-DTM
0.0	10.000000000	30.00000000	30.00000001
1.0	36.1914542129022010	45.1309114	45.13529775
2.0	47.8723837898386435	51.8631600	52.08209820
3.0	52.1829133112573729	1.00000000×10^8	54.90137977
4.0	53.5872304790154814	$-2.299987195 \times 10^{21}$	56.30065867
5.0	53.9256134229545552	5.1743979×10^{42}	57.21526596
6.0	53.9181411296454343	$1.926962062 \times 10^{67}$	57.96775778
7.0	53.8275976569081394	$4.254746849 \times 10^{87}$	58.66940791
8.0	53.7388154606516295	$1.507975530 \times 10^{105}$	59.35864141
9.0	53.6719948304011751	$4.242183068 \times 10^{120}$	60.04876614
10.0	53.6265982150580811	$2.696315926 \times 10^{134}$	60.74443051

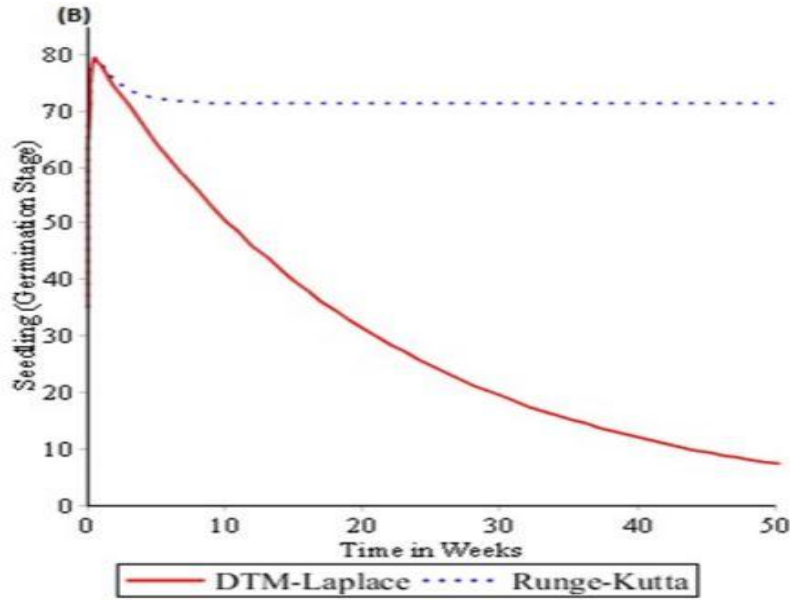


Figure 5: Comparison of Runge-Kutta forth order-RK45, DTM and Laplace-DTM for Susceptible Seeds to Seedlings

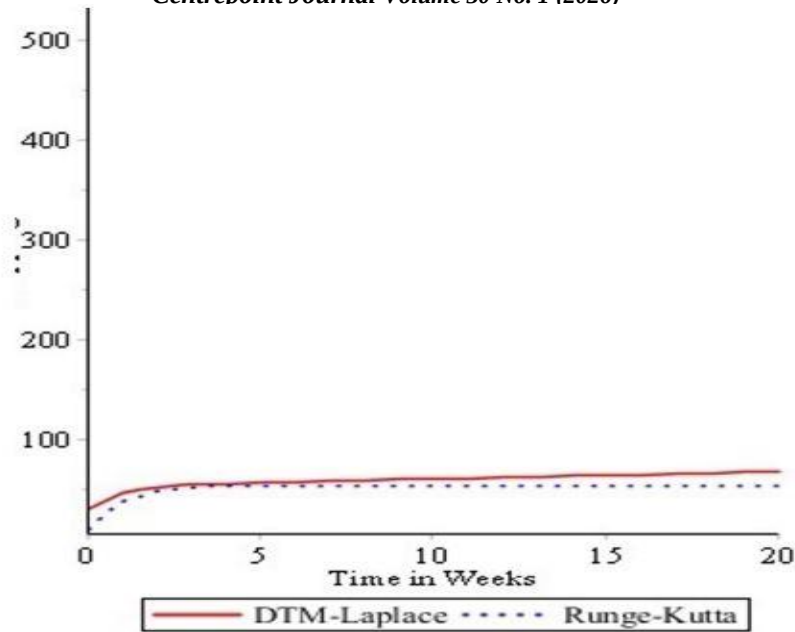


Figure 6: Comparison of Runge-Kutta forth order-RK45, DTM and Laplace-DTM for Suscep-
tible Seeds to Seedlings

Table 7: Numerical Scheme comparison (i.e. Runge-Kutta forth order-RK45, DTM and Laplace-DTM) for Harvest ($H(t)$)

T	RK45	DTM	Laplace-DTM
0	20.0000000	20.0000000	19.99999998
1	34.2262138732162171	43.32007088	43.32006380
2	59.6436242269047341	71.92300000	71.90556988
3	87.5636261854915716	2.20000000×10^6	100.0489202
4	114.560872658183740	$-8.54040000 \times 10^{18}$	124.5429486
5	139.502277517961772	$-4.801534680 \times 10^{42}$	142.9224632
6	162.155405350592645	$-3.045152066 \times 10^{66}$	153.0783656
7	182.611772621785008	$-3.999119537 \times 10^{86}$	153.2997681
8	201.059506271288512	$-1.058790333 \times 10^{104}$	142.4090549
9	217.699708761811138	$-2.452307159 \times 10^{119}$	119.9012815
10	232.719379810704766	$-1.353311766 \times 10^{133}$	86.05733828

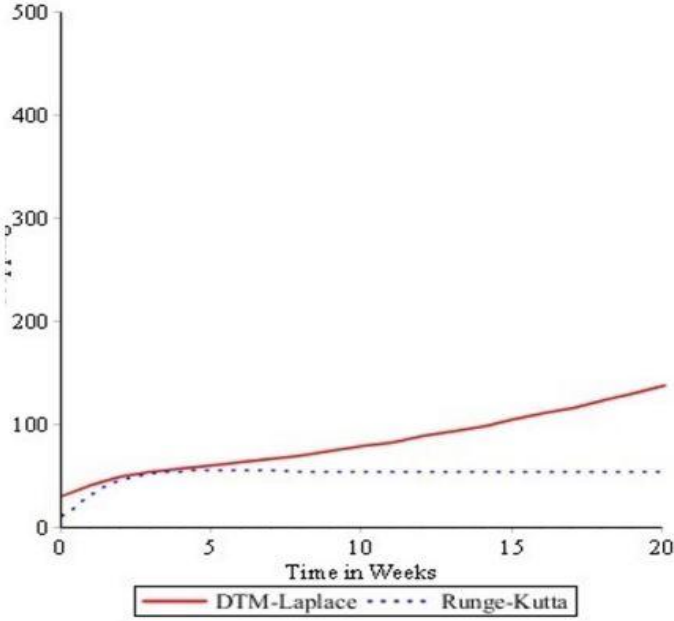


Figure 7: Comparison of Runge-Kutta forth order-RK45, DTM and Laplace-DTM for Suscep-
tible Seeds to Seedlings

Table 8: Numerical Scheme comparison (i.e. Runge-Kutta forth order-RK45, DTM and Laplace-DTM) for Beneficial Anthropodes ($P_v(t)$)

T	RK45	DTM	Laplace-DTM
0	7.50000000	7.50000000	7.500000030
1	21.4799922408770989	23.74097819	23.74097816
2	34.5148506086040143	41.33461983	41.33461976
3	46.6684719940221981	60.39358431	60.39358436
4	58.0004334632628300	81.03991405	81.03991404
5	68.5662843045951433	103.4058160	103.4058161
6	78.4178183311014720	127.6345083	127.6345084
7	87.6033277740090597	153.8811376	153.8811376
8	96.1678400112638201	182.3137713	182.3137714
9	104.153338291788970	213.1144760	213.1144761
10	111.598967537420236	246.4804810	246.4804810

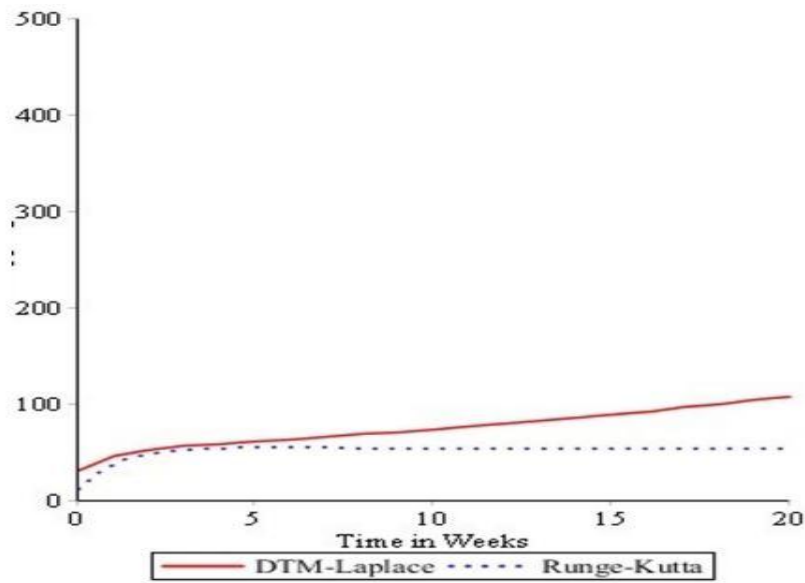


Figure 8: Comparison of Runge-Kutta forth order-RK45, DTM and Laplace-DTM for Susceptible Seeds to Seedlings

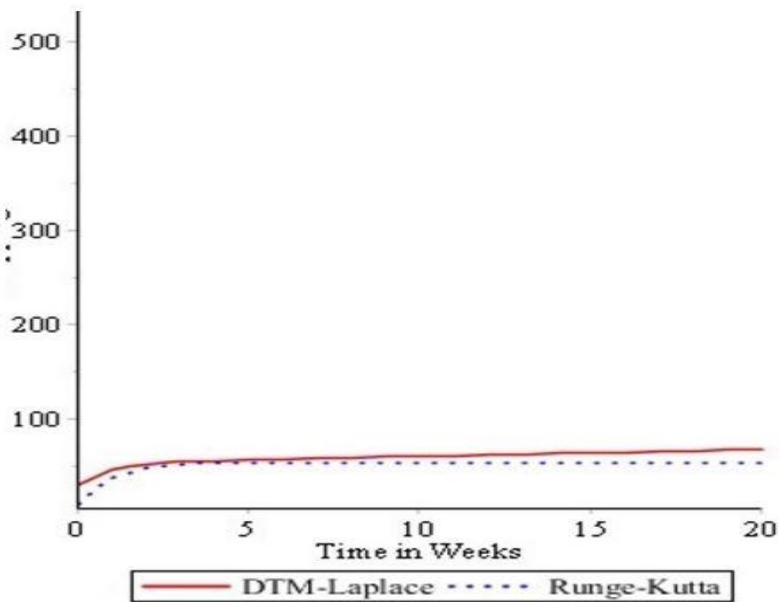


Figure 9: Comparison of Runge-Kutta forth order-RK45, DTM and Laplace-DTM for Susceptible Seeds to Seedlings

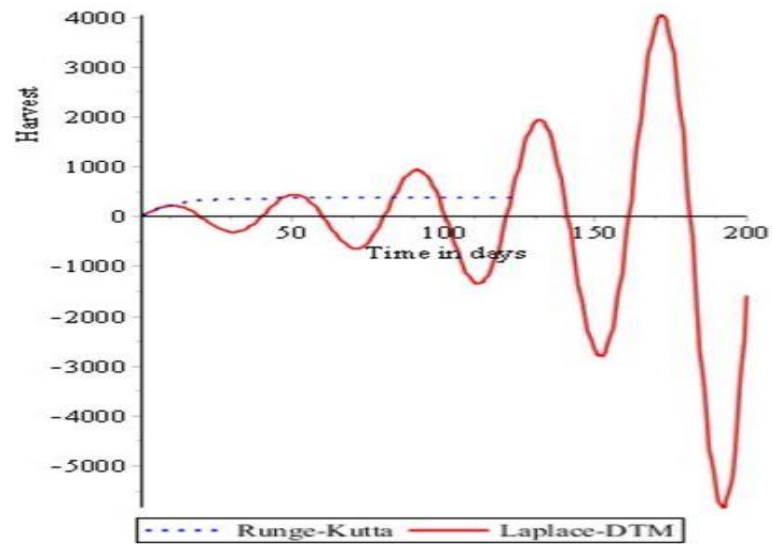


Figure 10: Comparison of Runge-Kutta forth order-RK45, DTM and Laplace-DTM for Susceptible Seeds to Seedlings

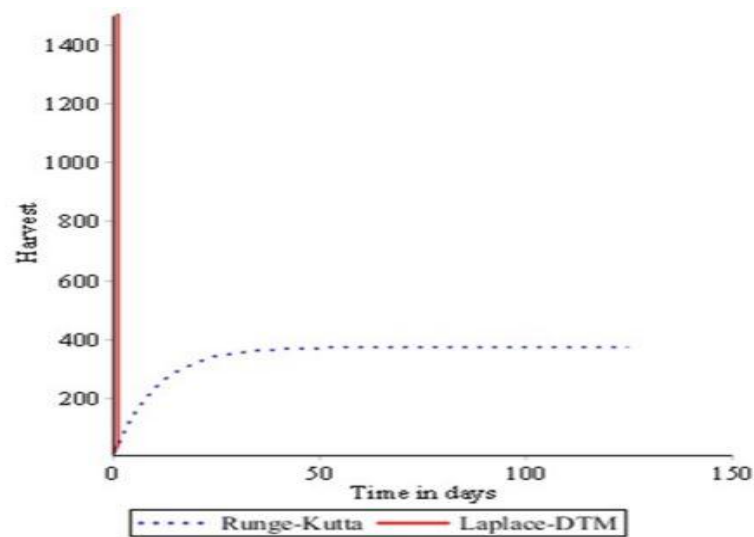


Figure 11: Comparison of Runge-Kutta forth order-RK45, DTM and Laplace-DTM for Susceptible Seeds to Seedlings

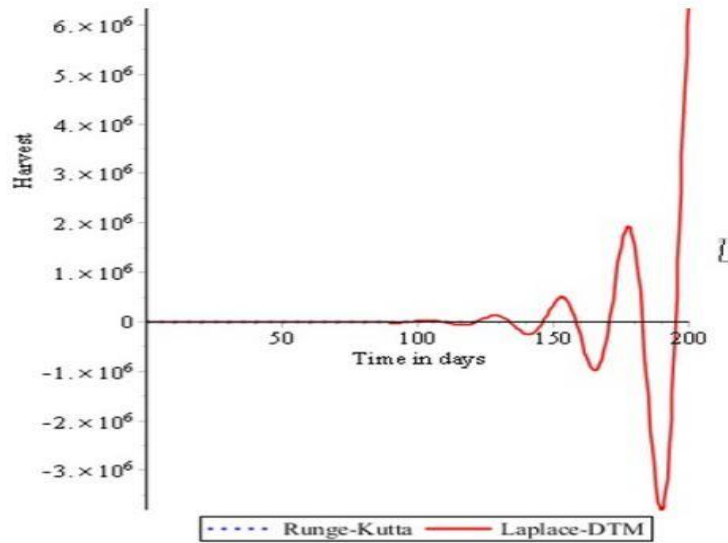


Figure 12: Comparison of Runge-Kutta forth order-RK45, DTM and Laplace-DTM for Susceptible Seeds to Seedlings

Table 9: Numerical Scheme comparison (i.e. Runge-Kutta forth order-RK45, DTM and Laplace-DTM) for Pest ($I_v(t)$)

t	RK45	DTM	Laplace-DTM
0	10.285000000	10.28500000	10.28499998
1	19.8229108235110694	18.52469879	18.52469876
2	29.1589738531856746	25.65826469	25.65826473
3	38.2852311541689119	31.76413991	31.76413985
4	47.1953164412388659	36.91106812	36.91106809
5	55.8842960866284457	41.15883131	41.15883125
6	64.3485233828654231	44.55890112	44.55890117
7	72.5855050422833301	47.15501132	47.15501128
8	80.5937789896623826	48.98365766	48.98365761
9	88.3728025739197989	50.07453047	50.07453052
10	95.9228503892545916	50.45088470	50.45088472

Conclusion

The study employed the Modified Differential Transform Method to solve the proposed mathematical model of sorghum pest management and the results obtained were consistent, more effective and accurate when compared with classical Runge-Kutta scheme (fourth Order) which is recognized to be accurate. However, due to the reputation possessed by classical Runge-Kutta scheme (fourth Order), it is still the best numerical scheme. The biological and botanical are good strategies for pest management control on sorghum farming. Hence, the ability for farmers to effectively identify and manage the controls determine optimum productivity to be harvest.

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Mahgoub Homotopy Analysis Method for the Solutions of Nonlinear Ordinary and Partial Differential Equations

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Abstract

Solving nonlinear problems involving both partial and ordinary differential equations is always more involving and mostly impossible through the well-established traditional analytical methods. We therefore proposed an analytical method that is derived through exploiting Mahgoub transform that is capable of solving variable coefficient linear differential equations. But this transform being unable to solve nonlinear problem, informed the integration of Homotopy analysis method to handle the nonlinear part. Thus, MahgoubHomotopy Analysis Method (MHAM) that is proposed reduces the volume of computation as well as producing the exact solution of the problem considered. Mahgoub of all the terms in the given equation is taken, and most importantly the differential coefficient. The Mahgoub terms independent of nonlinear term constitute the initial approximation, while the recurrence relation is developed by using the nonlinear terms. The two important deformation equation in the HAM are therefore obtained. The final solution is arrived at via taking the inverse Mahgoub of terms. Few questions on both ordinary and partial differential equations are used to validate the proposed method. The coding and general computations are carried out via Mathematica 13.3.

Keywords: Traditional methods, Mahgoub transform, Homotopy analysis method.

Introduction

Differential equations, such as ordinary, partial, and fractional differential equations, are used to mathematically model many physical, engineering, and biological processes. While integer-order derivatives are local in nature, fractional differential equations (FDEs), on the other hand, are defined by expanding the standard derivative to an arbitrary order, allowing for the inclusion of "memory effects" and "hereditary properties." (Podlubny, 1998).

Many real-world processes, such as heat transfer, fluid dynamics, viscoelastic materials, signal processing, and anomalous diffusion, are described by nonlinear fractional differential or integral equations. According to (Kilbas, Srivastava, and Trujillo, 2006), fractional differential equations are more effective than classical models because their power-law memory structure allows them to better capture the behavior of complex materials and processes. Furthermore, (Hilfer, 2000) extensively documented the transition from local classical mechanics to non-local fractional dynamics, demonstrating that fractional derivatives are essential for modeling time-dependent processes with long-range memory. Due to the complexity and nonlinearity of these models, obtaining exact solutions is often difficult or impossible. In recent years, researchers have developed various analytical, semi-analytical, and hybrid methods to obtain approximate solutions with acceptable accuracy.

Among these approaches are the application of integral transform to different forms of Differential Equation (DE) have been explored by different scientists, among them is the Laplace transform introduced by Pierre-Simon Laplace in the late eighteenth century, which is one of the oldest and most widely used transforms for solving linear differential equations with constant coefficients (Deakin, 1981). Fourier transform, introduced by Joseph Fourier in 1822 in *Théorie analytique de la chaleur*, has been extensively used for solving partial differential equations (PDEs) arising in heat conduction and wave propagation problems (Grattan-Guinness, 2005). The Sumudu transform, introduced by the Sri Lankan mathematician Watugala in the early 1990s, has been widely used to solve various ordinary differential equations (ODEs), PDEs, and integro-differential equations arising from problems in science and engineering, particularly in fields such as control systems, fluid mechanics, and mathematical physics (Watugala, 1993).

Several new integral transforms have been introduced in the literature to address these shortcomings. These include the Elzaki transform proposed by (Elzaki, 2011), the Shehu transform, and the Mahgoub transform. Out of the listed integral transforms employed, the Mahgoub transform in this work has drawn attention due to its wider range of applications, particularly when applied to differential equations with variable coefficients (Mahgoub, 2016).

In addition to the integral transform method approach, several semi-analytical methods have been developed to solve nonlinear differential and fractional differential equations without discretization. Prominent among these are Adomian decomposition method (ADM), the Variational Iteration Method (VIM) was introduced by (He, 1999). Despite its simplicity, VIM relies greatly on the proper selection of Lagrange multipliers, which is often problem specific. The Homotopy Analysis Method (HAM), introduced by Liao in 1992, is a powerful semi-analytical method that is independent of small or large parameters. The method introduces a convergence-control parameter h , which allows explicit control of the series convergence (Liao, 1992). HAM has been widely applied to nonlinear and fractional differential equations (Turq, 2020), and its flexibility in choosing the linear operator and initial approximation enhances both convergence and solution efficiency.

In the recent past, hybrid methods have been proposed to further enhance the accuracy and convergence. Integral transforms are integrated into semi-analytical methods, notable examples among these are the Laplace Adomian Decomposition Method (LADM) (Khan, Shah, Kumam, Baleanu, and Arif, 2020), the Shehu Transform Homotopy Analysis Method (STHAM) (Yisa and Baruwa, 2023), and the Laplace Homotopy Analysis Method (Vahidi, 2016). These methods reduce computational complexity and give greater convergence control compared to standalone methods.

In this work, the Mahgoub transform integrated with the Homotopy Analysis Method (HAM) is developed and proven to provide superior performance in solving variable-coefficient, strongly nonlinear, and fractional-order problems.

Brief Preliminaries of Mahgoub's Transform Method

This section will briefly review the definition and fundamental properties of the Mahgoub Transform.

Definition of Mahgoub Transform

The Mahgoub transform defined on the set of continuous function and of exponential order. We consider functions in the set B defined by:

$$M[f(\zeta)] = Q(\delta) = \delta \int_0^{\infty} f(\zeta) e^{-\delta\zeta} d\zeta, \quad \zeta \geq 0, \quad \xi_1 \leq \delta \leq \xi_2 .$$

The inverse Mahgoub Transform denoted by $M^{-1}[\cdot]$ is

$$f(\zeta) = M^{-1}[Q(\delta)] = \frac{1}{2\pi i} \int_{k-i\infty}^{k+i\infty} e^{\delta\zeta} \frac{Q(\delta)}{\delta} d\delta ,$$

where δ is the Mahgoub transform variable, i is the imaginary unit, and k is a real constant.

Mahgoub transform of some mathematical functions

Function $f(\zeta)$	Mahgoub Transform $M[f(\zeta)] = Q(\delta)$
1	1
ζ^n	$\frac{n!}{\delta^n}$
$e^{a\zeta}$	$\frac{\delta}{\delta - a}$
$\sin(a\zeta)$	$\frac{a\delta}{\delta^2 + a^2}$
$\cos(a\zeta)$	$\frac{\delta^2}{\delta^2 + a^2}$

Mahgoub Transform of Derivative

Let $f(\zeta)$ be a function for which the derivative is to be determined using the Mahgoub transform. Applying the Mahgoub transform, we obtain

- i. $M[f'(\zeta)] = \delta Q(\delta) - \delta f(0)$
- ii. $M[f''(\zeta)] = \delta^2 Q(\delta) - \delta f'(0) - \delta^2 f(0)$
- iii. $M[f'''(\zeta)] = \delta^3 Q(\delta) - \delta f''(0) - \delta^2 f'(0) - \delta^3 f(0)$

The n th order derivative is given as

$$M[f^{(n)}(\zeta)] = \delta^n Q(\delta) - \sum_{k=0}^{n-1} \delta^{n-k} f^{(k)}(0)$$

On the Applications of Mahgoub Transform to IVPs

In this section, we demonstrate the effectiveness of the Mahgoub transform by solving number of differential equations.

Example 1

Consider the inhomogeneous constant IVP

$$y''(\zeta) - 3y'(\zeta) + 2y(\zeta) = 4e^{3\zeta}, \quad y(0) = -3, y'(0) = 5.$$

Solution

$$y''(\zeta) - 3y'(\zeta) + 2y(\zeta) = 4e^{3\zeta}$$

By taking the Mahgoub transform of both sides, to get

$$(\delta^2 - 3\delta + 2)Q(\delta) - \delta y'(0) - \delta^2 y(0) + 3\delta y(0) = \frac{4\delta}{\delta - 3}$$

Using the initial conditions, we have

$$\begin{aligned} (\delta^2 - 3\delta + 2)Q(\delta) - 5\delta + 3\delta^2 - 9\delta &= \frac{4\delta}{\delta - 3} \\ Q(\delta) &= \delta \left(\frac{-38 + 23\delta - 3\delta^2}{(\delta - 1)(\delta - 2)(\delta - 3)} \right) \\ Q(\delta) &= \frac{4\delta}{\delta - 2} + \frac{2\delta}{\delta - 3} - \frac{9\delta}{\delta - 1} \end{aligned}$$

Taking the inverse Mahgoub transform, we have

$$y(\zeta) = 4e^{2\zeta} + 2e^{3\zeta} - 9e^{\zeta}.$$

Brief Review of Homotopy Analysis Method (HAM)

This section will briefly review some fundamental properties of HAM

The Homotopy Derivatives

The homotopy-derivatives operator D_k is defined as

$$D_k[\varphi(\eta; \rho)] = \frac{1}{k!} \frac{\partial^k \varphi(\eta; \rho)}{\partial \rho} \Big|_{\rho=0}, \quad k \geq 0,$$

where $\rho \in [0,1]$ is the embedding parameter.

If

$$\varphi(\eta; \rho) = \sum_{k=0}^{\infty} \varphi_k \rho^k,$$

then

$$D_k[\varphi(\eta; \rho)] = \varphi_k.$$

The Zeroth and k th Order Deformation Equation

Let

$$N[u(x)] = 0$$

be the nonlinear problem. The zeroth order deformation equation is defined as

$$(1 - \rho)L[\varphi(\eta; \rho) - \varphi_0(\eta)] = \rho h N[\varphi(\eta; \rho)],$$

with the properties

$$\varphi(\eta; 0) = \varphi_0(\eta), \quad \varphi(\eta; 1) = \varphi(\eta),$$

where L is a linear operator, N is a nonlinear operator, $\varphi_0(\eta)$ is the initial approximation, h is the convergence-control parameter, and $\rho \in [0,1]$ is the embedding parameter.

Apply the k th order homotopy-derivative D_k to the zeroth-order equation gives k th order deformation equation

$$L[\varphi_k(\eta; \rho) - \chi_k \varphi_{k-1}(\eta; \rho)] = h D_{k-1} [N[\varphi(\eta; \rho)]],$$

where

$$\chi_k = \begin{cases} 0, & k \leq 1, \\ 1, & k > 0. \end{cases}$$

Problems Considered

The class of the problems considered in this work are as listed below.

Integer Order Nonlinear Ordinary Differential Equation

$$Ly(\eta) + N[y(\eta)] + Hy(\eta) = f(\eta), \quad (2.1)$$

where L denotes a linear differential operator, $N[y(\eta)]$ is the nonlinear term, $Hy(\eta)$ is the remaining linear term, and $f(\eta)$ is the given inhomogeneous source term.

Subject to initial conditions $y(\eta_0) = y_0, \quad y'(\eta_0) = y_1, \dots, y^{(n-1)}(\eta_0) = y_{n-1}$.

Integer Order Nonlinear Partial Differential Equation

$$Ly(\eta, \xi) + N[y(\eta, \xi)] + Hy(\eta, \xi) = f(\eta, \xi), \quad (2.2)$$

where L denotes a linear differential operator, $N[y(\eta, \xi)]$ is the nonlinear term, $Hy(\eta, \xi)$ is the remaining linear term, and $f(\eta, \xi)$ is a given nonhomogeneous source term, subject to the initial conditions

$$y(\eta, 0) = y_0(\eta), \quad y'(\eta, 0) = y_1(\eta), \dots, y^{(n-1)}(\eta, 0) = y_{n-1}(\eta).$$

The class of problems above is solved by employing the proposed method tagged MahgoubHomotopyAnalysis Method (MHAM).

Methodology

This section outlines the algorithm of our proposed methods for solving the class of problems listed above.

Proposed Algorithm for the Solution of Nonlinear ODEs

To solve these using MHAM, we first apply the Mahgoub transform of both sides of (2.1)

$$M[Ly(\eta)] + M[N[y(\eta)]] + M[Hy(\eta)] = M[f(\eta)]. \quad (3.1)$$

But

$$M[Ly(\eta)] = \delta^n Q(\delta) - \sum_{i=0}^{n-1} \delta^{n-i} y^{(i)}(0). \quad (3.2)$$

Putting (3.2) in (3.1), we have

$$\delta^n Q(\delta) - \sum_{i=0}^{n-1} \delta^{n-i} y^{(i)}(0) + M[N[y(\eta)]] + M[Hy(\eta)] - M[f(\eta)] = 0. \quad (3.3)$$

Dividing(3.3) through by δ^n , we have

$$Q(\delta) - \frac{1}{\delta^n} \sum_{i=0}^{n-1} \delta^{n-i} y^{(i)}(0) + \frac{1}{\delta^n} [M[N[y(\eta)]] + M[Hy(\eta)] - M[f(\eta)]] = 0. \quad (3.4)$$

From (3.4), the resulting general nonlinear operator is defined as

$$N[y(\eta; \rho)] = Q(\delta) - \frac{1}{\delta^n} \sum_{i=0}^{n-1} \delta^{n-i} y^{(i)}(0) + \frac{1}{\delta^n} [M[N[y(\eta)]] + M[Hy(\eta)] - M[f(\eta)]] \quad (3.5)$$

The result in (3.5) shall be used in the k th order deformation equation

$$L[Q_k(\delta; \rho) - \chi_k Q(\delta; \rho)] = h D_{k-1} [N[y(\eta; \rho)]] \quad (3.6)$$

where ρ is the embedding parameter, and h is the convergence control parameter. The linear operator is defined as

$$L[Q_k(\delta; \rho) = Q_k(\delta),$$

thus

$$\begin{aligned} Q_k(\delta) - \chi_k Q_{k-1}(\delta) \\ = h D_{k-1} \left[Q(\delta) - \frac{1}{\delta^n} \sum_{i=0}^{n-1} \delta^{n-i} y^{(i)}(0) \right. \\ \left. + \frac{1}{\delta^n} [M[N[y(\eta)]] + M[Hy(\eta)] - M[f(\eta)]] \right] \end{aligned} \quad (3.7)$$

with the application of Homotopy Derivative D_{k-1} , (3.7) becomes

$$\begin{aligned} Q_k(\delta) - \chi_k Q_{k-1}(\delta) \\ = h \left[Q_{k-1}(\delta) - (1 - \bar{\chi}_{k-1}) \frac{1}{\delta^n} \left[\sum_{i=0}^{n-1} \delta^{n-i} y^{(i)}(0) + M[f(\eta)] \right] \right. \\ \left. + \frac{1}{\delta^n} [M[D_{k-1}[N[y(\eta)]]] + M[D_{k-1}[Hy(\eta)]]] \right] \end{aligned} \quad (3.8)$$

Taking $h = -1$, (3.8) becomes

$$\begin{aligned} Q_k(\delta) = -(1 - \chi_k) Q_{k-1}(\delta) + (1 - \bar{\chi}_{k-1}) \frac{1}{\delta^n} \left[\sum_{i=0}^{n-1} \delta^{n-i} y^{(i)}(0) + M[f(\eta)] \right] \\ - \frac{1}{\delta^n} [M[D_{k-1}[N[y(\eta)]]] + M[D_{k-1}[Hy(\eta)]]], \end{aligned} \quad (3.9)$$

where

$$\begin{aligned} \chi_k &= \begin{cases} 0, & k \leq 1, \\ 1, & k > 0 \end{cases} \\ \bar{\chi}_{k-1} &= \begin{cases} 0, & k-1 < 1, \\ 1, & k-1 \geq 1. \end{cases} \end{aligned}$$

But

$$y_k(\eta) = M^{-1}[Q(\delta)].$$

The series solution is obtained from (3.9) and the initial approximation $y_0(\eta)$ is derived from the Taylor series expansion using the initial condition prescribed.

Proposed Algorithm for the Solution of Nonlinear PDE

Let us consider the Integer order partial differential equation (PDE) in (2.2)

In order to solve these using MHAM, we first apply the Mahgoub transform of both sides of (2.2)

$$M[Ly(\eta, \xi)] + M[N[y(\eta, \xi)]] + M[Hy(\eta, \xi)] = M[f(\eta, \xi)] \quad (3.10)$$

but

$$M[Ly(\eta, \xi)] = \delta^n Q(\eta, \delta) - \sum_{i=0}^{n-1} \delta^{n-i} y_{\xi}^{(i)}(\eta, 0) \quad (3.11)$$

Putting (3.11) in (3.10), we have

$$\begin{aligned} \delta^n Q(\eta, \delta) - \sum_{i=0}^{n-1} \delta^{n-i} y_{\xi}^{(i)}(\eta, 0) + M[N[y(\eta, \xi)]] + M[Hy(\eta, \xi)] - M[f(\eta, \xi)] \\ = 0 \end{aligned} \quad (3.12)$$

Dividing (3.12) through by δ^n , we have

$$Q(\eta, \delta) - \frac{1}{\delta^n} \sum_{i=0}^{n-1} \delta^{n-i} y_{\xi}^{(i)}(\eta, 0) + \frac{1}{\delta^n} [M[N[y(\eta, \xi)]] + M[Hy(\eta, \xi)] - M[f(\eta, \xi)]] = 0 \quad (3.13)$$

From (3.13), the resulting general nonlinear operator is defined as

$$\begin{aligned} N[y(\eta, \delta; \rho)] = Q(\eta, \delta) - \frac{1}{\delta^n} \sum_{i=0}^{n-1} \delta^{n-i} y_{\xi}^{(i)}(\eta, 0) \\ + \frac{1}{\delta^n} [M[N[y(\eta, \xi)]] + M[Hy(\eta, \xi)] - M[f(\eta, \xi)]] \end{aligned} \quad (3.14)$$

The result above shall be implemented in k th order deformation equation

$$L[Q_k(\eta, \delta; \rho) - \chi_k Q_{k-1}(\eta, \delta; \rho)] = h D_{k-1} [N[y(\eta, \delta; \rho)]], \quad (3.15)$$

where ρ is the embedding parameter, and h is the convergence control parameter

The linear operator is obtained as

$$L[Q_k(\eta, \delta; \rho) = Q_k(\eta, \delta),$$

thus

$$\begin{aligned}
 Q_k(\eta, \delta) - \chi_k Q_{k-1}(\eta, \delta) &= h D_{k-1} \left[Q(\eta, \delta) - \frac{1}{\delta^n} \sum_{i=0}^{n-1} \delta^{n-i} y_\xi^{(i)}(0) \right. \\
 &\quad \left. + \frac{1}{\delta^n} [M[N[y(\eta, \xi)]] + M[Hy(\eta, \xi)] \right. \\
 &\quad \left. - M[f(\eta, \xi)] \right] \quad (3.16)
 \end{aligned}$$

With the application of Homotopy Derivative D_{k-1} , (3.16) becomes

$$\begin{aligned}
 Q_k(\eta, \delta) - \chi_k Q_{k-1}(\eta, \delta) &= h \left[Q_{k-1}(\eta, \delta) - (1 - \bar{\chi}_{k-1}) \frac{1}{\delta^n} \left[\sum_{i=0}^{n-1} \delta^{n-i} y_\xi^{(i)}(0) + M[f(\eta, \xi)] \right] \right. \\
 &\quad \left. + \frac{1}{\delta^n} [M[D_{k-1}[N[y(\eta, \xi)]]] + M[D_{k-1}[Hy(\eta, \xi)]] \right] \quad (3.17)
 \end{aligned}$$

Taking $h = -1$, (3.17) becomes

$$\begin{aligned}
 Q_k(\eta, \delta) &= -(1 - \chi_k) Q_{k-1}(\eta, \delta) + (1 - \bar{\chi}_{k-1}) \frac{1}{\delta^n} \left[\sum_{i=0}^{n-1} \delta^{n-i} y_\xi^{(i)}(0) + M[f(\eta, \xi)] \right] \\
 &\quad - \frac{1}{\delta^n} [M[D_{k-1}[N[y(\eta, \xi)]]] + M[D_{k-1}[Hy(\eta, \xi)]]], \quad (3.18)
 \end{aligned}$$

where

$$\chi_k = \begin{cases} 0, & k \leq 1, \\ 1, & k > 0 \end{cases}, \quad \bar{\chi}_{k-1} = \begin{cases} 0, & k - 1 < 1, \\ 1, & k - 1 \geq 1. \end{cases}$$

But

$$y_k(\eta, \xi) = M^{-1}[Q(\eta, \xi)].$$

The series solution is obtained from (3.18) and the initial approximation $y_0(\eta, \xi)$ is obtained from the Taylor series expansion of two variables using the initial condition prescribed.

On the Application of MHAM to ODE and PDE

Here, we solved examples employing the two algorithms explored in sections (3.1) and (3.2).

Examples on Ordinary Differential Equations

Problem 1 (Yisa and Baruwa, 2023) Consider the nonlinear homogeneous IVP

$$y''(\eta) + 2y(\eta)y'(\eta) = 0, \quad y(0) = 0, y'(0) = 1.$$

Solution

$$y''(\eta) + 2y(\eta)y'(\eta) = 0$$

We take the Mahgoub transform of both sides, to get

$$M[y''(\eta)] + 2M[y(\eta)y'(\eta)] = M[0] \\ \delta^2 Q(\delta) - \delta y'(0) - \delta^2 y(0) + 2M[y(\eta)y'(\eta)] = 0$$

Dividing through by δ^2 , we get

$$Q(\delta) - \frac{1}{\delta} y'(0) - y(0) + \frac{2}{\delta^2} M[y(\eta)y'(\eta)] = 0$$

Using the initial condition, we get

$$Q(\delta) - \frac{1}{\delta} + \frac{2}{\delta^2} M[y(\eta)y'(\eta)] = 0$$

The linear and nonlinear operators are obtained as

$$L[Q(\delta; \rho)] = Q(\delta)$$

$$N[y(\delta; \rho)] = Q(\delta) - \frac{1}{\delta} + \frac{2}{\delta^2} M[y(\eta)y'(\eta)]$$

Using the k th order deformation equation

$$L[Q_k(\delta; \rho) - \chi_k Q_{k-1}(\delta; \rho)] = h D_{k-1} [N[y(\delta; \rho)]] \\ Q_k(\delta) - \chi_k Q_{k-1}(\delta) = h D_{k-1} \left[Q(\delta) - \frac{1}{\delta} + \frac{2}{\delta^2} M[y(\eta)y'(\eta)] \right] \\ Q_k(\delta) = \chi_k Q_{k-1}(\delta) + h D_{k-1} \left[Q(\delta) - \frac{1}{\delta} + \frac{2}{\delta^2} M[y(\eta)y'(\eta)] \right]$$

With the application of Homotopy Derivative, we have

$$Q_k(\delta) = \chi_k Q_{k-1}(\delta) + h \left[Q_{k-1}(\delta) - (1 - \bar{\chi}_{k-1}) \frac{1}{\delta} + \frac{2}{\delta^2} M \left[\sum_{i=0}^{k-1} y_i \frac{dy_{k-1-i}}{d\eta} \right] \right]$$

Taking $h = -1$,

$$Q_k(\delta) = -(1 - \chi_k) Q_{k-1}(\delta) + (1 - \bar{\chi}_{k-1}) \frac{1}{\delta} - \frac{2}{\delta^2} M \left[\sum_{i=0}^{k-1} y_i \frac{dy_{k-1-i}}{d\eta} \right]$$

When $k = 1$:

$$Q_1(\delta) = -(1 - \chi_1)Q_0(\delta) + (1 - \bar{x}_0)\frac{1}{\delta} - \frac{2}{\delta^2}M[y_0y_0']$$

The initial approximation

$$y_0(\eta) = y(0) + \eta y'(0) + \frac{\eta^2}{2!}y''(0) + \frac{\eta^3}{3!}y'''(0) + \dots$$

We have, $y_0(\eta) = \eta$, $H_0(v) = \frac{1}{\delta}$.

$$Q_1(\delta) = -\frac{1}{\delta} + \frac{1}{\delta} - \frac{2}{\delta^2}M[x] = -\frac{2}{\delta^3}$$

Taking the inverse Mahgoub, we have $y_1(\eta) = -\frac{\eta^3}{3}$.

When $k = 2$:

$$Q_2(\delta) = -(1 - \chi_2)Q_1(\delta) + (1 - \bar{x}_1)\frac{1}{\delta} - \frac{2}{\delta^2}M\left[\sum_{i=0}^1 y_i \frac{dy_{1-i}}{d\eta}\right] = -\frac{2}{\delta^2}M[y_0y_1' + y_1y_0']$$

$$Q_2(\delta) = -\frac{2}{\delta^2}M\left[-\eta^3 - \frac{\eta^3}{3}\right]$$

$$Q_2(\delta) = \frac{16}{\delta^5}$$

Taking the inverse Mahgoub, we have

$$y_2(\eta) = \frac{2}{15}\eta^5.$$

When $k = 3$:

$$Q_3(\delta) = -(1 - \chi_3)Q_2(\delta) + (1 - \chi_2)\frac{1}{\delta} - \frac{2}{\delta^2}M\left[\sum_{i=0}^2 y_i \frac{dy_{2-i}}{d\eta}\right]$$

$$Q_3(\delta) = -\frac{2}{\delta^2}M[y_0y_2' + y_2y_0' + y_1y_1']$$

$$Q_3(\delta) = -\frac{2}{\delta^2}M\left[\frac{2}{3}\eta^5 + \frac{2}{15}\eta^5 + \frac{1}{5}\eta^5\right]$$

$$Q_3(\delta) = -\frac{2}{\delta^2}M[\eta^5]$$

$$H_3(v) = -\frac{240}{\delta^7}.$$

Taking the inverse Mahgoub, we have $y_3(\eta) = -\frac{\eta^7}{21}$.

The third- order approximation

$$y(\eta) = \sum_{k=0}^3 y_k(\eta) = \eta - \frac{1}{3}\eta^3 + \frac{2}{15}\eta^5 - \frac{1}{21}\eta^7$$

Problem 2 (Yisa and Baruwa, 2023)

Consider the inhomogeneous nonlinear IVP

$$y''(\eta) - y'(\eta)^2 + y(\eta)^2 = 1, \quad y(0) = 1, \quad y'(0) = 0.$$

Solution

$$y''(\eta) - y'(\eta)^2 + y(\eta)^2 = 1$$

The procedure followed in the solution to Problem 1 leads to

Taking $h = -1$,

$$Q_k(\delta) = -(1 - \chi_k)Q_{k-1}(\delta) + (1 - \bar{\chi}_{k-1})\left(1 + \frac{1}{\delta^2}\right) + \frac{1}{\delta^2}M\left[\sum_{i=0}^{k-1} y'_i y'_{k-1-i}\right] - \frac{1}{\delta^2}M\left[\sum_{i=0}^{k-1} y_i y_{k-1-i}\right]$$

The initial approximation

$$y_0(\eta) = y(0) + \eta y'(0) + \frac{\eta^2}{2!} y''(0) + \frac{\eta^3}{3!} y'''(0) + \dots$$

$$y(\eta) = \sum_{k=0}^3 y_k(\eta)$$

$$y(\eta) = y_0(\eta) + y_1(\eta) + y_2(\eta) + y_3(\eta)$$

$$y(x) = 1 + 0 + 0 + 0$$

$$y(\eta) = 1.$$

Examples on Partial Differential Equations

Problem 1 (Olayiwola, Morufu Oyedunsi and Yunus, Akeem Olarewaju and Alaje, Adedapo Ismaila and Adedeji, Joseph Adeleke, 2025)

Consider the nonlinear, nonlocal Fornberg-Whitham equation

$$\frac{\partial u}{\partial \xi} = \frac{\partial^3 u}{\partial \eta^2 \partial \xi} - \frac{\partial u}{\partial \eta} - u \frac{\partial u}{\partial \eta} + u \frac{\partial^3 u}{\partial \eta^3} + 3 \frac{\partial u}{\partial \eta} \frac{\partial^2 u}{\partial \eta^2}$$

$$\text{Subject to } u(\eta, 0) = \frac{4}{3} e^{\frac{\eta}{2}}.$$

Solution

$$\frac{\partial u}{\partial \xi} = \frac{\partial^3 u}{\partial \eta^2 \partial \xi} - \frac{\partial u}{\partial \eta} - u \frac{\partial u}{\partial \eta} + u \frac{\partial^3 u}{\partial \eta^3} + 3 \frac{\partial u}{\partial \eta} \frac{\partial^2 u}{\partial \eta^2}$$

Take the Mahgoub transform of both sides

$$\delta Q(\eta, \delta) - \delta u(\eta, 0) = M \left[\frac{\partial^3 u}{\partial \eta^2 \partial \xi} \right] - M \left[\frac{\partial u}{\partial \eta} \right] - M \left[u \frac{\partial u}{\partial \eta} \right] + M \left[u \frac{\partial^3 u}{\partial \eta^3} \right] + 3M \left[\frac{\partial u}{\partial \eta} \frac{\partial^2 u}{\partial \eta^2} \right]$$

$$Q(\eta, \delta) - u(\eta, 0) = \frac{1}{\delta} M \left[\frac{\partial^3 u}{\partial \eta^2 \partial \xi} \right] - \frac{1}{\delta} M \left[\frac{\partial u}{\partial \eta} \right] - \frac{1}{\delta} M \left[u \frac{\partial u}{\partial \eta} \right] + \frac{1}{\delta} M \left[u \frac{\partial^3 u}{\partial \eta^3} \right] + \frac{3}{\delta} M \left[\frac{\partial u}{\partial \eta} \frac{\partial^2 u}{\partial \eta^2} \right]$$

Using the initial condition, we have

$$Q(\eta, \delta) - \frac{4}{3} e^{\frac{\eta}{2}} = \frac{1}{\delta} M \left[\frac{\partial^3 u}{\partial \eta^2 \partial \xi} \right] - \frac{1}{\delta} M \left[\frac{\partial u}{\partial \eta} \right] - \frac{1}{\delta} M \left[u \frac{\partial u}{\partial \eta} \right] + \frac{1}{\delta} M \left[u \frac{\partial^3 u}{\partial \eta^3} \right] + \frac{3}{\delta} M \left[\frac{\partial u}{\partial \eta} \frac{\partial^2 u}{\partial \eta^2} \right]$$

$$Q(\eta, \delta) - \frac{4}{3} e^{\frac{\eta}{2}} - \frac{1}{\delta} M \left[\frac{\partial^3 u}{\partial \eta^2 \partial \xi} \right] + \frac{1}{\delta} M \left[\frac{\partial u}{\partial \eta} \right] + \frac{1}{\delta} M \left[u \frac{\partial u}{\partial \eta} \right] - \frac{1}{\delta} M \left[u \frac{\partial^3 u}{\partial \eta^3} \right] - \frac{3}{\delta} M \left[\frac{\partial u}{\partial \eta} \frac{\partial^2 u}{\partial \eta^2} \right] = 0$$

The linear and nonlinear operator is obtained as

$$L[Q(\eta, \delta; \rho)] = Q(\eta, \delta)$$

$$\begin{aligned} N[u(\eta, \delta; \rho)] &= Q(\eta, \delta) - \frac{4}{3} e^{\frac{\eta}{2}} - \frac{1}{\delta} M \left[\frac{\partial^3 u}{\partial \eta^2 \partial \xi} \right] + \frac{1}{\delta} M \left[\frac{\partial u}{\partial \eta} \right] + \frac{1}{\delta} M \left[u \frac{\partial u}{\partial \eta} \right] - \frac{1}{\delta} M \left[u \frac{\partial^3 u}{\partial \eta^3} \right] \\ &\quad - \frac{3}{\delta} M \left[\frac{\partial u}{\partial \eta} \frac{\partial^2 u}{\partial \eta^2} \right] \end{aligned}$$

Using the k th order deformation equation

$$L[Q_k(\eta, \delta; \rho) - \chi_k Q_{k-1}(\eta, \delta; \rho)] = h D_{k-1} [N[u(\eta, \delta; \rho)]]$$

$$\begin{aligned} Q_k(\eta, \delta) - \chi_k Q_{k-1}(\eta, \delta) &= h D_{k-1} \left[Q(\eta, \delta) - \frac{4}{3} e^{\frac{\eta}{2}} - \frac{1}{\delta} M \left[\frac{\partial^3 u}{\partial \eta^2 \partial \xi} \right] + \frac{1}{\delta} M \left[\frac{\partial u}{\partial \eta} \right] + \frac{1}{\delta} M \left[u \frac{\partial u}{\partial \eta} \right] \right. \\ &\quad \left. - \frac{1}{\delta} M \left[u \frac{\partial^3 u}{\partial \eta^3} \right] - \frac{3}{\delta} M \left[\frac{\partial u}{\partial \eta} \frac{\partial^2 u}{\partial \eta^2} \right] \right] \end{aligned}$$

$$\begin{aligned} Q_k(\eta, \delta) &= \chi_k Q_{k-1}(\eta, \delta) \\ &\quad + h D_{k-1} \left[Q(\eta, \delta) - \frac{4}{3} e^{\frac{\eta}{2}} - \frac{1}{\delta} M \left[\frac{\partial^3 u}{\partial \eta^2 \partial \xi} \right] + \frac{1}{\delta} M \left[\frac{\partial u}{\partial \eta} \right] + \frac{1}{\delta} M \left[u \frac{\partial u}{\partial \eta} \right] \right. \\ &\quad \left. - \frac{1}{\delta} M \left[u \frac{\partial^3 u}{\partial \eta^3} \right] - \frac{3}{\delta} M \left[\frac{\partial u}{\partial \eta} \frac{\partial^2 u}{\partial \eta^2} \right] \right] \end{aligned}$$

With the application of Homotopy Derivative, we have

$$Q_k(\eta, \delta) = \chi_k Q_{k-1}(\eta, \delta) + h \left[Q_{k-1}(\eta, \delta) - (1 - \bar{\chi}_{k-1}) \frac{4}{3} e^{\frac{\eta}{2}} - \frac{1}{\delta} M \left[\frac{\partial^3 u_{k-1}}{\partial \eta^2 \partial \xi} \right] + \frac{1}{\delta} M \left[\frac{\partial u_{k-1}}{\partial \eta} \right] + \frac{1}{\delta} M \left[\sum_{i=0}^{k-1} u_i \frac{\partial u_{k-1-i}}{\partial \eta} \right] - \frac{1}{\delta} M \left[\sum_{i=0}^{k-1} u_i \frac{\partial^3 u_{k-1-i}}{\partial \eta^3} \right] - \frac{3}{\delta} M \left[\sum_{i=0}^{k-1} \frac{\partial u_i}{\partial \eta} \frac{\partial^2 u_{k-1-i}}{\partial \eta^2} \right] \right]$$

Taking $h = -1$, we have

$$Q_k(\eta, \delta) = -(1 - \chi_k) Q_{k-1}(\eta, \delta) + (1 - \bar{\chi}_{k-1}) \frac{4}{3} e^{\frac{\eta}{2}} + \frac{1}{\delta} M \left[\frac{\partial^3 u_{k-1}}{\partial \eta^2 \partial \xi} \right] - \frac{1}{\delta} M \left[\frac{\partial u_{k-1}}{\partial \eta} \right] - \frac{1}{\delta} M \left[\sum_{i=0}^{k-1} u_i \frac{\partial u_{k-1-i}}{\partial \eta} \right] + \frac{1}{\delta} M \left[\sum_{i=0}^{k-1} u_i \frac{\partial^3 u_{k-1-i}}{\partial \eta^3} \right] + \frac{3}{\delta} M \left[\sum_{i=0}^{k-1} \frac{\partial u_i}{\partial \eta} \frac{\partial^2 u_{k-1-i}}{\partial \eta^2} \right]$$

When $k = 1$:

$$Q_1(\eta, \delta) = -(1 - \chi_1) Q_0(\eta, \delta) + (1 - \bar{\chi}_0) \frac{4}{3} e^{\frac{\eta}{2}} + \frac{1}{\delta} M \left[\frac{\partial^3 u_0}{\partial \eta^2 \partial \xi} \right] - \frac{1}{\delta} M \left[\frac{\partial u_0}{\partial \eta} \right] - \frac{1}{\delta} M \left[u_0 \frac{\partial u_0}{\partial \eta} \right] + \frac{1}{\delta} M \left[u_0 \frac{\partial^3 u_0}{\partial \eta^3} \right] + \frac{3}{\delta} M \left[\frac{\partial u_0}{\partial \eta} \frac{\partial^2 u_0}{\partial \eta^2} \right]$$

The initial approximation

$$u_0(\eta, \xi) = u(\eta, 0) + \xi \frac{\partial u}{\partial \xi}(\eta, 0) + \frac{\xi^2}{2!} \frac{\partial^2 u}{\partial \xi^2}(\eta, 0) + \dots$$

We have

$$u_0(\eta, \xi) = \frac{4}{3} e^{\frac{\eta}{2}}, \quad Q_0(\eta, \delta) = \frac{4}{3} e^{\frac{\eta}{2}}.$$

$$Q_1(\eta, \delta) = -\frac{4}{3} e^{\frac{\eta}{2}} + \frac{4}{3} e^{\frac{\eta}{2}} + \frac{1}{\delta} M[0] - \frac{1}{\delta} M \left[\frac{2}{3} e^{\frac{\eta}{2}} \right] - \frac{1}{\delta} M \left[\frac{8}{9} e^{\eta} \right] + \frac{1}{\delta} M \left[\frac{2}{9} e^{\eta} \right] + \frac{3}{\delta} M \left[\frac{2}{9} e^{\eta} \right]$$

$$Q_1(\eta, \delta) = -\frac{2}{3\delta} e^{\frac{\eta}{2}}.$$

Taking the inverse Mahgoub, we have

$$u_1(\eta, \xi) = -\frac{2}{3}e^{\frac{\eta}{2}}\xi.$$

When $k = 2$:

$$\begin{aligned} Q_2(\eta, \delta) &= -(1 - \chi_2)Q_1(\eta, \delta) + (1 - \bar{\chi}_1)\frac{4}{3}e^{\frac{\eta}{2}} + \frac{1}{\delta}M\left[\frac{\partial^3 u_1}{\partial \eta^2 \partial \xi}\right] - \frac{1}{\delta}M\left[\frac{\partial u_1}{\partial \eta}\right] \\ &\quad - \frac{1}{\delta}M\left[\sum_{i=0}^1 u_i \frac{\partial u_{1-i}}{\partial \eta}\right] + \frac{1}{\delta}M\left[\sum_{i=0}^1 u_i \frac{\partial^3 u_{1-i}}{\partial \eta^3}\right] + \frac{3}{\delta}M\left[\sum_{i=0}^1 \frac{\partial u_i}{\partial \eta} \frac{\partial^2 u_{1-i}}{\partial \eta^2}\right] \\ Q_2(\eta, \delta) &= \frac{1}{\delta}M\left[-\frac{1}{6}e^{\frac{\eta}{2}}\right] - \frac{1}{\delta}M\left[-\frac{1}{3}e^{\frac{\eta}{2}}\xi\right] - \frac{1}{\delta}M\left[-\frac{8}{9}e^{\eta}\xi\right] + \frac{1}{\delta}M\left[-\frac{2}{9}e^{\eta}\xi\right] \\ &\quad + \frac{3}{\delta}M\left[-\frac{2}{9}e^{\eta}\xi\right] \\ Q_2(\eta, \delta) &= -\frac{1}{6\delta}e^{\frac{\eta}{2}} + \frac{1}{3\delta^2}e^{\frac{\eta}{2}} \end{aligned}$$

Taking the inverse Mahgoub, we have

$$u_2(\eta, \xi) = \frac{1}{6}e^{\frac{\eta}{2}}(\xi^2 - \xi).$$

The second- order approximation

$$\begin{aligned} u(\eta, \xi) &= \sum_{k=0}^2 u_k(\eta, \xi) \\ u(\eta, \xi) &= \frac{4}{3}e^{\frac{\eta}{2}} - \frac{2}{3}e^{\frac{\eta}{2}}\xi + \frac{1}{6}e^{\frac{\eta}{2}}(\xi^2 - \xi). \end{aligned}$$

Discussion of the Results

The results obtained for Problems 1 and 2 on integer-order ordinary differential equations (ODEs), using the MahgoubHomotopy Analysis Method (MHAM), are in complete agreement with the results in the existing literature Yisa & Baruwa (2023), where the standard HAM and STHAM were employed respectively. Also, Problems 1 and 2, which involve integer-order PDEs, were previously solved by Olayiwola *et al.* (2025) using the Laplace Adomian Decomposition Method; notably, the MHAM employed in this work yielded the same results.

Conclusion

Mahgoub Homotopy Analysis Method (MHAM) has been successfully implemented to obtain approximate analytical solutions of some integer order differential equations. By integrating the Mahgoub transform with the Homotopy Analysis Method, we have developed a robust framework that simplifies the process of solving complex functional equations. This proposed method yields good results with less computation. Ultimately, MHAM proves to be a more efficient and reliable tool for solving integer order differential equations.

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A Review: Neuroprotective Properties of Aidan Fruit (*Tetrapleuratetraptera*)

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Abstract

Neurodegenerative disorders such as Alzheimer's disease, Parkinson's disease, stroke, epilepsy, and diabetic neuropathy pose a major global health burden. *Tetrapleuratetraptera*, a perennial tree widely distributed in West and Central Africa, has traditionally been used to manage pain, inflammation, metabolic disorders, and nervous system imbalances. This review synthesizes current evidence on its neuroprotective potential. A systematic search of PubMed, Scopus, Web of Science, Google Scholar, ScienceDirect, and African repositories was conducted, including peer-reviewed English-language studies with manual reference screening. Findings indicate that *T. tetraptera* exerts neuroprotective effects through antioxidant activity, suppression of neuroinflammation, modulation of neurotransmitter systems, inhibition of apoptosis, metal chelation, and reduction of excitotoxicity. Experimental studies report improved neuronal viability, cognitive performance, and biochemical markers of neural integrity. Overall, *T. tetraptera* shows promising neuroprotective potential, warranting further clinical trials, bioactive compound isolation, and standardized formulation development for evidence-based neurological therapy.

Keywords: Aidan fruit, Neuroprotection, Neurodegenerative diseases, Oxidative stress, Phytochemicals

Introduction

Neurological and neurodegenerative disorders constitute a major and growing public health challenge worldwide (Wang et al., 2024). Conditions such as Alzheimer's disease, Parkinson's disease, stroke, epilepsy, and multiple sclerosis significantly impair quality of life and place enormous emotional and economic burdens on individuals, families, and healthcare systems (Harsh et al., 2025; Godswill et al., 2024). Neurological disorders are the leading cause of physical and cognitive disability across the globe, currently affecting approximately 15% of the worldwide population (Van Schependom&D'haeseleer, 2023). Global health estimates indicate that neurological disorders are among the leading causes of disability-adjusted life years (DALYs), with prevalence steadily increasing due to population growth, ageing, urbanisation, and

lifestyle changes (Ding et al., 2022). In low- and middle-income countries, including many parts of sub-Saharan Africa, limited access to specialised neurological care exacerbates disease outcomes, highlighting the urgent need for affordable and effective preventive and therapeutic strategies (Kandel, 2025).

Neurodegenerative disorders are characterised by progressive loss of neuronal structure and function, often resulting in irreversible cognitive decline and motor dysfunction (Przedborski, 2016). Common pathological mechanisms include oxidative stress, neuroinflammation, mitochondrial dysfunction, excitotoxicity, and abnormal protein aggregation (Błaszczuk, 2020). Despite advances in modern medicine, pharmacological treatments are largely symptomatic and do not adequately halt disease progression (Zhang et al., 2024). High costs, limited efficacy, and adverse effects restrict long-term use of synthetic drugs, prompting increasing interest in natural products (Cohen & Weinstein, 2018).

Medicinal plants are rich sources of bioactive compounds such as polyphenols, flavonoids, alkaloids, terpenoids, and saponins, many of which exhibit antioxidant, anti-inflammatory, and neuromodulatory properties (Oladele et al., 2025). These compounds can scavenge reactive oxygen species, modulate neurotransmitter systems, regulate intracellular signalling pathways, and inhibit neuronal cell death (Akter et al., 2022). Unlike single-target synthetic drugs, plant-derived compounds often exert multi-target effects, advantageous in complex disorders such as neurodegenerative diseases (Makhoba et al., 2020).

Aidan fruit (Tetrapleuratetraptera), a perennial plant in the Fabaceae family, shows considerable therapeutic promise (Mensal et al., 2024). Commonly known as Aidan fruit, Aridan, or Prekese, it is widely distributed across West and Central Africa (Imade et al., 2024). The dried fruit is aromatic and used as a spice, but also in traditional medicine to manage inflammation, infections, metabolic disorders, pain, and cardiovascular conditions (Imade et al., 2024). Phytochemical studies indicate that the fruit is rich in flavonoids, phenolic acids, saponins, tannins, alkaloids, and essential oils, which are critical for neuroprotection (Adeyeni et al., 2024). Previous research has shown that *Tetrapleuratetraptera* therapy is beneficial for wound healing, diabetes, hypertension, epilepsy, convulsions, leprosy, mental illness, inflammation, arthritis and rheumatoid pain, and muscle relaxants (Igwe & Akabuiké, 2016). Recent research has also demonstrated that the fruits, bark, and pod extracts have antioxidant properties and the fruit is also used as a vitamin-rich food supplement (Onimisi et al., 2024; Saliu et al., 2021).

Despite its traditional use and promising biological activities, the neuroprotective potential of *T. tetraptera* has not been systematically reviewed. Existing studies are fragmented, focusing on isolated pharmacological properties, and variations in extraction methods, experimental models, and outcomes complicate comparisons. A comprehensive review is therefore warranted to consolidate knowledge and guide future research.

Methodology

Study Design

This study adopted a comprehensive literature review approach to evaluate and synthesise existing scientific evidence on the neuroprotective properties of *Tetrapleuratetraptera* (Aidan fruit). The review focused on experimental, preclinical, and limited clinical studies reporting neuroprotective mechanisms, bioactive compounds, and therapeutic relevance in neurodegenerative conditions.

Data Sources

A systematic search of reputable electronic databases was conducted to identify relevant literature. The databases searched included:

- PubMed/MEDLINE
- Scopus
- Web of Science
- Google Scholar
- ScienceDirect

In addition, African-focused databases and institutional repositories were consulted to capture region-specific studies on *Tetrapleuratetraptera*. Reference lists of eligible articles were manually screened to identify additional relevant publications.

Search Strategy

A structured search strategy was employed using combinations of Medical Subject Headings (MeSH) terms and free-text keywords. Boolean operators (AND, OR) were applied to refine the search. Key search terms included:

- Aidan fruit
- Neuroprotection
- Neurodegenerative diseases
- Oxidative stress
- Phytochemicals

Searches were limited to peer-reviewed articles published in English. No strict publication year restriction was imposed in order to capture both foundational and recent studies.

Inclusion Criteria

Studies were included in the review if they met the following criteria:

1. Original research articles, review papers, or short communications.
2. Studies investigating *Tetrapleuratetraptera* or its extracts, isolates, or bioactive compounds.
3. Studies reporting neuroprotective mechanisms, including antioxidant, anti-inflammatory, anti-apoptotic, or neurotransmitter-modulating effects.

4. In vitro, in vivo (animal), and limited human studies relevant to neurological or neurodegenerative outcomes.
5. Articles published in English and accessible in full text.

Exclusion Criteria

The following studies were excluded:

1. Articles unrelated to neurological or neuroprotective outcomes.
2. Studies focusing solely on non-neurological pharmacological effects (e.g., antimicrobial or cardiovascular effects without neural relevance).
3. Conference abstracts, editorials, letters to the editor, and unpublished theses without sufficient methodological details.
4. Duplicated studies across databases.
5. Articles with insufficient data on study design, outcomes, or relevance to neuroprotection.

Study Selection and Review Procedure

All retrieved articles were exported into a reference management system, where duplicates were identified and removed. The study selection was conducted in three stages: Title screening to exclude clearly irrelevant articles, Abstract screening to assess relevance to neuroprotective properties of *Tetrapleura tetraptera* and Full-text review of eligible articles to confirm inclusion based on predefined criteria.

Botanical and Ethnomedicinal Profile of Aidan Fruit



Figure 1: Aidan fruit tree and fruit (Adesina *et al.*, 2016)

Aidan fruit is a flowering plant belonging to the family Fabaceae (subfamily Mimosoideae). Taxonomically, it is classified as follows: Kingdom *Plantae*; Division *Magnoliophyta*; Class *Magnoliopsida*; Order *Fabales*; Family *Fabaceae*; Genus *Tetrapleura*; Species *T. tetraptera*. The plant is a perennial deciduous tree that can grow up to 20–25 meters in height, with a spreading crown and rough grey-brown bark (Ogbedeagu *et al.*, 2025).

The most distinctive feature of *T. tetraptera* is its fruit, which is a long, dark brown pod characterised by four longitudinal wings, hence the name *tetraptera*, meaning “four-winged.” The pods typically measure 15–25 cm in length and emit a strong, sweet, aromatic fragrance due to the presence of essential oils (Olaniyi et al., 2023). Inside the pod are small, hard seeds embedded in a fibrous pulp. The leaves are bipinnate, while the flowers are small, pale pink to yellowish, and borne in dense spikes. These botanical features not only aid in plant identification but are also relevant to its traditional harvesting and use (Akintola. Et al., 2015)

Geographical Distribution

Tetrapleuratetraptera is indigenous to tropical Africa and is widely distributed across West and Central African regions (Akintola. Et al., 2015). It is commonly found in countries such as Nigeria, Ghana, Cameroon, Côte d’Ivoire, Sierra Leone, Liberia, and parts of Uganda. The plant thrives in lowland rainforests and savannah-forest transition zones, preferring well-drained soils and humid climatic conditions.

In Nigeria, Aidan fruit is particularly popular in the southern regions, where it is known by different local names, including *Aidan* or *Aridan* among the Yoruba, *Uyayak* among the Efik and Ibibio, and *Oshosho* in some Igbo communities. In Ghana, it is commonly referred to as *Prekese*. Its wide geographical distribution and cultural relevance have contributed to its extensive use as both a culinary spice and a medicinal plant. The ease of availability and long shelf-life of the dried fruit further enhance its role in traditional healthcare systems (Olaniyi et al., 2023)..

Traditional Medicinal Uses (with Emphasis on Neurological-Related Uses)

Aidan fruit has a long history of use in African traditional medicine, where it is valued for its broad therapeutic applications. Traditionally, different parts of the plant—especially the fruit and bark—are used in decoctions, infusions, or powdered forms to manage a variety of ailments. These include inflammatory conditions, infections, hypertension, diabetes, postpartum complications, and gastrointestinal disorders (Akintola *et al.*, 2015).

Of particular relevance to neurological health, Aidan fruit is traditionally used for conditions associated with pain, stress, fatigue, and nervous system imbalance (Kuroki et al., 2024). In some communities, decoctions of the fruit are administered for the relief of headaches, body aches, and febrile illnesses, which may involve neuroinflammatory processes. The fruit is also used as a calming agent in traditional postpartum care, where it is believed to promote relaxation, restore strength, and improve overall well-being. Such uses suggest potential sedative, analgesic, or neuro-modulatory effects (Adeniyi et al., 2023).

Additionally, *T. tetraptera* is employed in the management of convulsive disorders and mental agitation in certain ethnomedicinal practices, although these applications are often reported anecdotally and vary between cultures. The plant’s use in managing chronic conditions such as diabetes and hypertension is also indirectly relevant to neurological health, as these metabolic disorders are known risk factors for cognitive

decline, stroke, and neuropathy. While traditional healers may not explicitly classify these conditions as “neurological,” their treatments often target symptoms and underlying factors now recognised in modern medicine as contributors to nervous system dysfunction(El Sayed & Egbuna, 2023).

Nutritional Composition

Beyond its medicinal applications, Aidan fruit is nutritionally rich, supporting its classification as a functional food. Proximate analyses have shown that the fruit contains appreciable amounts of carbohydrates, dietary fibre, and moderate levels of protein, with low fat content. The high fibre content is particularly beneficial for metabolic health, which is closely linked to neurological function(David et al., 2025).

The fruit is also a good source of essential minerals, including calcium, potassium, magnesium, iron, and zinc. These minerals play important roles in nerve conduction, synaptic transmission, and maintenance of neuronal membrane integrity. Furthermore, *T. tetraptera* contains several vitamins, notably vitamin C and some B-complex vitamins, which are essential for antioxidant defence and normal nervous system function (SANDO & UKO-AVIOMOH, 2023).

Importantly, the nutritional value of Aidan fruit is complemented by its rich phytochemical composition, including phenolic compounds and flavonoids that contribute to its antioxidant capacity. The combination of nutritional and bioactive components positions *Tetrapleuratetraptera* as a nutraceutical candidate with potential benefits for brain health (Edem, 2025).

Phytochemical Constituents of Aidan Fruit (*Tetrapleuratetraptera*)

Polyphenols and Flavonoids

Polyphenols and flavonoids constitute one of the most abundant and biologically significant classes of phytochemicals present in Aidan fruit. Quantitative analyses have revealed high total phenolic and flavonoid contents in both aqueous and organic extracts of *T. tetraptera* fruit. Identified phenolic compounds include phenolic acids such as gallic acid and related derivatives, while flavonoids such as quercetin-like and kaempferol-like compounds have been reported.

These compounds are well known for their potent antioxidant properties, which are crucial in protecting neurons from oxidative damage induced by reactive oxygen and nitrogen species. Flavonoids can cross the blood–brain barrier and exert direct effects on neuronal cells by modulating intracellular signalling pathways, enhancing synaptic plasticity, and inhibiting neuroinflammation. In the context of Aidan fruit, the presence of these polyphenolic compounds supports its traditional use in conditions associated with pain, inflammation, and cognitive stress, and suggests a mechanistic basis for neuroprotection (Mensah et al., 2024).

Alkaloids

Alkaloids are another important group of secondary metabolites identified in *Tetrapleuratetraptera*. Although present in relatively moderate concentrations compared

to polyphenols, alkaloids are pharmacologically significant due to their strong biological activity. Alkaloids are known to interact with neurotransmitter systems, including cholinergic, dopaminergic, and serotonergic pathways, which play key roles in cognition, mood regulation, and motor control(Mensah et al., 2024).

The alkaloid content of Aidan fruit may contribute to its observed analgesic and central nervous system–modulating effects in traditional medicine. By influencing neurotransmission and neuronal excitability, these compounds may help attenuate excitotoxicity, a major mechanism of neuronal injury in disorders such as epilepsy, stroke, and neurodegenerative diseases. However, the specific alkaloid structures in *T. tetraptera* remain insufficiently characterised, highlighting the need for further isolation and structural elucidation studies(Mensah et al., 2024).

Saponins and Tannins

Saponins and tannins are widely distributed in Aidan fruit and contribute significantly to its therapeutic profile. Saponins are amphipathic glycosides known for their anti-inflammatory, immunomodulatory, and membrane-stabilising properties. In neurological contexts, saponins have been shown to protect neuronal membranes, inhibit inflammatory mediators, and reduce apoptosis, thereby preserving neuronal integrity(Mensah et al., 2024).

Tannins, on the other hand, are polyphenolic compounds with strong astringent and free radical–scavenging abilities. They can chelate metal ions such as iron and copper, which are implicated in oxidative stress and amyloid aggregation in neurodegenerative diseases. The combined presence of saponins and tannins in Aidan fruit may therefore enhance its ability to counteract oxidative stress, inflammation, and metal-induced neurotoxicity(Mensah et al., 2024).

Essential Oils and Other Bioactive Compounds

The characteristic aromatic fragrance of Aidan fruit is attributed to its essential oil content, which comprises a complex mixture of volatile compounds. These essential oils contain various terpenoids and aromatic compounds that exhibit antimicrobial, anti-inflammatory, and antioxidant activities. Some volatile constituents are also known to possess anxiolytic and sedative properties, suggesting potential benefits for stress-related and mood disorders(Mensah et al., 2024).

In addition to essential oils, *T. tetraptera* contains other bioactive compounds such as glycosides, steroids, and reducing sugars. These compounds may not directly target neuronal pathways but can influence overall metabolic and inflammatory status, which indirectly affects brain health. The synergistic interactions among these diverse phytochemicals likely enhance the overall neuroprotective efficacy of Aidan fruit (Mensah et al., 2024).

Antioxidant Capacity

Oxidative stress is a central mechanism in the pathogenesis of most neurological and neurodegenerative disorders, making antioxidant activity a key criterion for

neuroprotective agents. Numerous in vitro studies have demonstrated that extracts of Aidan fruit exhibit strong antioxidant capacity, as evidenced by their ability to scavenge free radicals, reduce lipid peroxidation, and enhance endogenous antioxidant defences (Korang et al., 2023; Mensah et al., 2024).

The high antioxidant activity of *T. tetraptera* is largely attributed to its polyphenols, flavonoids, and tannins, which can donate hydrogen atoms or electrons to neutralise reactive species. These compounds also upregulate antioxidant enzymes such as superoxide dismutase, catalase, and glutathione peroxidase in experimental models. By reducing oxidative damage to lipids, proteins, and DNA, the phytochemicals in Aidan fruit help preserve neuronal structure and function (Adusi et al., 2019).

Collectively, the diverse phytochemical constituents of Aidan fruit provide a strong biochemical foundation for its neuroprotective properties. The presence of multiple antioxidant and neuro-modulatory compounds supports the hypothesis that *Tetrapleura tetraptera* may offer multi-target protection against the complex pathological processes underlying neurological disorders (Fauzi & Nurfarahin, 2025).

Pathophysiology of Neurodegeneration

Oxidative Stress and Neuronal Damage

Oxidative stress is one of the most prominent and widely implicated mechanisms in neurodegeneration. It arises from an imbalance between the production of reactive oxygen and nitrogen species and the capacity of endogenous antioxidant systems to neutralise them (Houldsworth, 2024; Butterfield & Boyd-Kimball, 2018). Neurons are particularly vulnerable to oxidative damage due to their high oxygen consumption, abundant lipid content, and relatively low regenerative capacity (Singh et al., 2019; Lin & Beal, 2006).

Excessive oxidative stress leads to lipid peroxidation of neuronal membranes, oxidation of proteins, and damage to nucleic acids, resulting in impaired synaptic transmission and neuronal death. In neurodegenerative disorders, oxidative stress may be triggered by mitochondrial dysfunction, neuroinflammation, and metal ion dysregulation. Over time, cumulative oxidative damage compromises neuronal integrity and accelerates disease progression (Olufunmilayo et al., 2023; Butterfield et al., 2013).

Neuroinflammation

Neuroinflammation is another key contributor to neurodegenerative processes. It involves the activation of resident immune cells in the central nervous system, particularly microglia and astrocytes, in response to injury, infection, or abnormal protein accumulation (Heneka et al., 2015). While acute inflammatory responses can be protective, chronic and uncontrolled neuroinflammation is detrimental to neuronal survival (Glass et al., 2010).

Activated glial cells release pro-inflammatory cytokines, chemokines, nitric oxide, and reactive oxygen species, which exacerbate neuronal damage and disrupt synaptic function. Sustained neuroinflammation has been implicated in the progression of

Alzheimer's disease, Parkinson's disease, and multiple sclerosis. Importantly, oxidative stress and neuroinflammation are closely interconnected, forming a self-perpetuating cycle that amplifies neuronal injury (Block et al., 2007; Heneka et al., 2015).

Mitochondrial Dysfunction

Mitochondria play a central role in neuronal health by regulating energy production, calcium homeostasis, and apoptotic signalling. Mitochondrial dysfunction is a hallmark of neurodegenerative diseases and is characterised by impaired oxidative phosphorylation, reduced adenosine triphosphate (ATP) production, and increased generation of reactive oxygen species (Lin & Beal, 2006; Bose & Beal, 2016).

Defective mitochondria compromise neuronal energy supply, making neurons more susceptible to stress and injury. Additionally, mitochondrial damage can trigger intrinsic apoptotic pathways through the release of pro-apoptotic factors, leading to programmed neuronal death. Given the high energy demands of neurons, even mild mitochondrial dysfunction can have profound consequences for brain function and viability (Nicholls & Budd, 2000).

Excitotoxicity

Excitotoxicity refers to neuronal injury caused by excessive stimulation of excitatory neurotransmitter receptors, particularly those activated by glutamate. Under normal physiological conditions, glutamate-mediated signalling is essential for learning, memory, and synaptic plasticity. However, excessive glutamate release or impaired reuptake leads to prolonged activation of glutamate receptors, resulting in abnormal calcium influx into neurons (Dong et al., 2009).

Elevated intracellular calcium levels activate a cascade of destructive enzymes, including proteases, lipases, and endonucleases, which damage cellular structures and promote neuronal death. Excitotoxicity is a major pathological mechanism in acute conditions such as stroke and traumatic brain injury, as well as in chronic neurodegenerative disorders. It is closely linked to oxidative stress and mitochondrial dysfunction, further amplifying neuronal damage (Hardingham & Bading, 2010).

Protein Misfolding and Aggregation

Abnormal protein folding and aggregation are defining features of many neurodegenerative diseases. Misfolded proteins can accumulate within neurons or in extracellular spaces, forming toxic aggregates that interfere with cellular processes. Examples include amyloid-beta plaques and tau tangles in Alzheimer's disease, alpha-synuclein aggregates in Parkinson's disease, and huntingtin protein aggregates in Huntington's disease (Ross & Poirier, 2004; Soto & Pritzkow, 2018).

These protein aggregates disrupt synaptic function, impair axonal transport, and trigger inflammatory and oxidative responses. Moreover, they overwhelm cellular quality control systems such as the ubiquitin-proteasome system and autophagy pathways, leading to further accumulation of toxic proteins. The progressive nature of protein

aggregation contributes to the chronic and irreversible course of neurodegenerative disorders (Taylor et al., 2002; Soto & Pritzkow, 2018).

Mechanisms of Neuroprotective Action of Aidan Fruit

Antioxidant Mechanisms

Antioxidant activity represents one of the most important mechanisms underlying the neuroprotective effects of Aidan fruit. The fruit is rich in polyphenols, flavonoids, tannins, and other antioxidant compounds that can directly scavenge reactive oxygen and nitrogen species (Mensah et al., 2024; Adesina et al., 2019). By neutralising these free radicals, Aidan fruit reduces oxidative damage to neuronal lipids, proteins, and nucleic acids, thereby maintaining cellular integrity (Kumar & Pandey, 2013).

In addition to direct free radical scavenging, bioactive compounds in *T. tetraptera* may enhance endogenous antioxidant defence systems. Experimental studies have reported increased activity of antioxidant enzymes such as superoxide dismutase, catalase, and glutathione peroxidase following treatment with plant extracts (Mensah et al., 2024). This dual action helps counteract oxidative stress, a central driver of neuronal injury in neurodegenerative disorders (Singh et al., 2019).

Anti-inflammatory Effects

Chronic neuroinflammation significantly contributes to the progression of neurodegeneration. Aidan fruit exhibits notable anti-inflammatory properties that may mitigate inflammation-mediated neuronal damage. Phytochemicals such as flavonoids, saponins, and essential oil constituents have been shown to inhibit the production and release of pro-inflammatory cytokines (Mensah et al., 2024; Aderibigbe et al., 2017).

By suppressing microglial and astrocyte overactivation, Aidan fruit may limit sustained inflammatory responses that exacerbate neuronal loss. Furthermore, the reduction of inflammation indirectly decreases oxidative stress, supporting neuronal survival and preserving synaptic function (Heneka et al., 2015).

Modulation of Neurotransmitters

Proper neurotransmitter balance is essential for normal brain function, and its disruption is a hallmark of many neurological disorders. Alkaloids and flavonoids present in Aidan fruit may influence neurotransmitter synthesis, release, and receptor activity, modulating cholinergic, dopaminergic, and glutamatergic pathways (Wink, 2015; Mensah et al., 2024).

Anti-apoptotic Pathways

Apoptosis is a key mechanism of neuronal loss in neurodegenerative disorders. Polyphenols and saponins in Aidan fruit have been shown to inhibit pro-apoptotic factors while enhancing anti-apoptotic proteins in experimental models, thereby preserving neuronal populations (Podolak et al., 2010; Mensah et al., 2024).

Metal Chelation and Reduction of Excitotoxicity

Phenolic compounds and tannins in Aidan fruit possess metal-chelating properties that reduce iron- and copper-mediated oxidative stress (Kumar & Pandey, 2013). Modulation of glutamate signalling further reduces excitotoxic injury, enhancing the neuroprotective profile of *Tetrapleuratetraptera* (Dong et al., 2009).

DISCUSSION

Aidan fruit possesses a broad spectrum of bioactive compounds capable of targeting multiple pathological processes implicated in neurodegenerative and neurological disorders. The convergence of traditional medicinal use with emerging experimental data strengthens the scientific plausibility of its neuroprotective role (Chinedu S. Ogunwa et al., 2024; Nutritive Value et al., 2025).

One of its most significant insights is the strong antioxidant capacity, which aligns closely with current understanding of neurodegeneration. Oxidative stress is a central mechanism underlying neuronal damage in disorders such as Alzheimer's disease, Parkinson's disease, stroke, and diabetic neuropathy (Smith et al., 2025). The high content of polyphenols, flavonoids, and tannins in *Tetrapleuratetraptera* provides a biochemical basis for its ability to scavenge reactive oxygen species, reduce lipid peroxidation, and enhance endogenous antioxidant defenses (Nwafor et al., 2024; Nutritive Value et al., 2025).

In addition to antioxidant effects, the anti-inflammatory properties of Aidan fruit represent another critical neuroprotective mechanism. Chronic neuroinflammation, mediated by sustained activation of microglia and astrocytes, plays a major role in progressive neuronal loss (Smith et al., 2025). Phytochemicals identified in *T. tetraptera*, including flavonoids and saponins, have demonstrated the ability to suppress pro-inflammatory cytokines and inflammatory mediators in experimental models (Nwafor et al., 2024). By dampening neuroinflammatory responses, Aidan fruit may help interrupt the vicious cycle between oxidative stress and inflammation that accelerates neurodegeneration.

The modulation of neurotransmitter systems further expands its neuroprotective relevance. Alkaloids and flavonoids present in the fruit are known to interact with cholinergic, dopaminergic, and glutamatergic pathways, which are critically involved in cognition, mood regulation, and motor control (Chinedu S. Ogunwa et al., 2024). This multi-neurotransmitter influence is particularly important because many neurodegenerative diseases involve widespread neurotransmitter dysregulation rather than isolated pathway dysfunction.

Experimental evidence from in vitro and in vivo studies provides further support for these mechanistic insights. In vitro studies consistently demonstrate that Aidan fruit extracts protect neuronal cells against oxidative stress-induced cytotoxicity and preserve antioxidant enzyme activity (Chinedu S. Ogunwa et al., 2024). These cellular-level findings are reinforced by animal studies showing improved brain antioxidant status, reduced neuroinflammatory markers, preserved neuronal architecture, and enhanced

behavioural and cognitive outcomes in models of neurological injury (Adeyemi et al., 2025). Improvements in learning, memory, locomotion, and anxiety-related behaviours suggest that the observed neuroprotection is not merely structural but also functionally meaningful (Adeyemi et al., 2025).

The potential application of Aidan fruit across multiple neurological disorders highlights its value as a multi-target neuroprotective agent. In Alzheimer's disease, its antioxidant, anti-inflammatory, and cholinergic-modulating properties may collectively address key aspects of disease pathology (Smith et al., 2025). In Parkinson's disease, protection against oxidative stress and inflammation may help preserve dopaminergic neurons. Similarly, in stroke and epilepsy, the ability of Aidan fruit to mitigate oxidative damage, excitotoxicity, and inflammatory injury supports its potential role as an adjunctive therapeutic option (Adeyemi et al., 2025).

Despite these promising findings, the study also underscores important limitations in current research. The most notable gap is the scarcity of clinical studies. While preclinical models provide valuable mechanistic and proof-of-concept evidence, the available data cannot fully capture the complexity of human neurological diseases. The lack of randomised controlled trials limits conclusions regarding clinical efficacy, optimal dosing, and long-term safety. Additionally, significant variability in extraction methods and phytochemical composition across studies complicates result comparison and reproducibility (Chinedu S. Ogunwa et al., 2024).

Another key limitation is the incomplete understanding of molecular mechanisms. Many studies rely on general biochemical markers rather than detailed pathway analysis. The absence of comprehensive molecular, genomic, and proteomic investigations restricts insight into how individual compounds or synergistic phytochemical interactions drive neuroprotection. Furthermore, challenges related to standardisation, bioavailability, and pharmacokinetics must be addressed before clinical translation is feasible (Nutritive Value et al., 2025).

Conclusion

Aidan fruit (*Tetrapleuratetraptera*) exhibits considerable neuroprotective potential, as supported by its rich phytochemical composition and multi-target mechanisms of action. The presence of polyphenols, flavonoids, alkaloids, saponins, tannins, and essential oils enables the fruit to combat oxidative stress, suppress neuroinflammation, modulate neurotransmitter systems, inhibit apoptosis, and reduce excitotoxic and metal-induced neuronal damage. Experimental evidence from in vitro and in vivo studies demonstrates that these bioactive compounds can preserve neuronal structure, improve cognitive and behavioural outcomes, and enhance biochemical markers of brain health.

Disclaimer (Artificial intelligence)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

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Analytical Solutions of Nonlinear Initial-Boundary Value Problems

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Abstract

This work presents solutions to initial-boundary value problems that are generally nonlinear exploring Taylor's theorem in a decomposition technic. Not until few decades back, real world phenomena in many fields of human endeavours were modelled into initial and boundary value problems. Due to identified deficiencies in doing that, initial-boundary value equations are used to achieve the aim. The problems are mostly nonlinear. Solution to such category of problems mostly defies the use of the established analytical methods. Most researchers employ numerical approaches that result in approximate solutions. The present approach uses Taylor's theorem alongside Adomian decomposition method. The nonlinear aspect is treated by decomposing it into polynomials, while the resulting unknown terms are evaluated through Taylor's expansion. The method is applied to eight problems in literature, all of which give exact solution. Mathematica 13.3 is employed for the simulations.

Keywords: Adomian polynomials, Boundary value problem, Initial value problem, Taylor's theorem for two variables.

Introduction

The importance of differential equations in the understanding of the physical world around us, especially as it affects modelling of phenomena that affect human and animal existence (Jimoh & Oyediji, 2020).

Since the resulting differential equations are mostly nonlinear, solving them by the traditional methods (otherwise referred to as analytical methods) becomes very difficult and in most cases outrightly impossible. Thus, the need to employ other approaches such as numerical methods or better still semi-analytical methods. The latter is in many instances preferred since they give exact solution whenever such exists in closed form. When the problem does not have an exact solution, the truncated series solution that semi-analytical methods will give is always better than what numerical approach would give. Prominent among those methods are Adomian decomposition

method (ADM) Adomian (1994), variational iteration method (VIM) in (He, 1999), homotopy analysis method (HAM) introduced by (Liao, 1997), just to mention a few. The cornerstone of ADM lies in the decomposition of the nonlinear operator present in the equation into a series of specific polynomials, known as Adomian polynomials, which depend on the series components of the solution (Elsaid, 2012). The current study solves non-linear partial differential equations using Adomian decomposition method.

Boundary value problems (BVPs) as expressed by (Boyce, 2017; Agmon, 2010; Ladyzhenskaya, 2013; Gakhov, 2014) are the physical application that often led to another problem in which the value of the dependent variable or its derivatives is specified at two different points. Boundary conditions are described as the values or relationship prescribed for the function or its derivatives at the special boundary of the region. But when the conditions are specified at the same point, it is referred to as initial value problems (IVPs) (Sarbach & Tiglio, 2012; Synge, 2012; Keller, 2018).

(Ahmed & Dmitry, 2011) and few other scientists proved the convergence of Adomian decomposition method for various types of problems. The researchers made use of Majorants, which is a tool in Cauchy-Kovalevsky theorem for differential equations with analytic vector fields, others used Picard iterative approach to establish its convergence. ADM does not require any small or large parameter in its application, nor does it require any control parameter that guarantees convergence of its solution.

To simplify the derivation of Adomian polynomials for strongly nonlinear terms, (Yisa, 2019) presented solutions of ordinary differential equations with special nonlinearities. In that work, the author derived the Adomian polynomials for some selected difficult nonlinear terms and implemented them in the solutions of nonlinear initial value problems using ADM.

In the present work, ADM is used to solve initial-boundary value problems of one, two and three dimensions. The category of problems considered cannot be handled without incorporating Taylor's theorem involving two variables.

Literature Review

In this section, the definitions relevant to the discussion in what follows, statement of the problem considered, and some other useful things are presented.

The Definition of Taylor's Theorem for Two Variables

Let f be a function of two variables ξ and η . If all partial derivatives of order m are continuous in a closed region and if all the $(m + 1)$ partial derivatives exist in the open region, then

$$\begin{aligned} f(\xi_0 + h, \eta_0 + k) = & f(\xi_0, \eta_0) + \left[h \frac{\partial}{\partial \xi} + k \frac{\partial}{\partial \eta} \right] f(\xi_0, \eta_0) + \frac{1}{2!} \left[h \frac{\partial}{\partial \xi} + k \frac{\partial}{\partial \eta} \right]^2 f(\xi_0, \eta_0) + \dots \\ & + \frac{1}{m!} \left[h \frac{\partial}{\partial \xi} + k \frac{\partial}{\partial \eta} \right]^m f(\xi_0, \eta_0) + R_m, \end{aligned} \quad (1)$$

where

$$R_m = \frac{1}{(m+1)!} \left[h \frac{\partial}{\partial \xi} + k \frac{\partial}{\partial \eta} \right]^{m+1} f(\xi_0 + \theta h, \eta_0 + \theta k), \quad 0 < \theta < 1 \text{ (Wrede, 2010)}$$

Definition of Adomian polynomials

A less cumbersome formula for the derivation of polynomials corresponding to respective nonlinear terms was reported in Adomian (1994), as

$$A_n = \frac{1}{n!} \frac{\partial^n}{\partial \lambda^n} \left[N \left(\sum_{k=0}^n \lambda^k u_k \right) \right]_{\lambda=0}, \quad n = 0, 1, 2, \dots \quad (2)$$

Considered the nonlinear n th-order initial-boundary value problem:

$$Lu(\xi, \eta) + Ru(\xi, \eta) + N(u(\xi, \eta)) = g(\xi, \eta) \quad (3)$$

with boundary conditions: $u(0, \eta) = \beta_0$, $u(L, \eta) = \beta_1$

and associated initial conditions: $u(\xi, 0) = \alpha_0$, $u_\eta(\xi, 0) = \alpha_1, \dots, u_{\eta \dots \eta}(\xi, 0) = \alpha_{n-1}$

Methodology

Generation of Adomian polynomials for some nonlinear terms

In this section, Adomian polynomials for nonlinear terms were generated for the first few polynomials A_0, A_1, A_2 .

Consider the nonlinear term $N(u) = (u_{\xi\xi})^2$

$$N(u) = (u_{\xi\xi})^2$$

$$A_n = \frac{1}{n!} \frac{\partial^n}{\partial \lambda^n} \left[N \left(\sum_{k=0}^n \lambda^k u_k \right) \right]_{\lambda=0},$$

For $n = 0$:

$$A_0 = N(u_0) = (u_0)_{\xi\xi}^2$$

For $n = 1$:

$$A_1 = \frac{\partial}{\partial \lambda} \left[N \left(\sum_{k=0}^1 \lambda^k u_k \right) \right]_{\lambda=0},$$

$$A_1 = \frac{\partial}{\partial \lambda} [N(u_0 + \lambda u_1)]_{\lambda=0}$$

$$A_1 = \frac{\partial}{\partial \lambda} [(u_0)_{\xi\xi} + \lambda (u_1)_{\xi\xi}]^2_{\lambda=0}$$

$$A_1 = 2(u_1)_{\xi\xi} [(u_0)_{\xi\xi} + \lambda (u_1)_{\xi\xi}]_{\lambda=0}$$

$$A_1 = 2(u_0)_{\xi\xi} (u_1)_{\xi\xi}$$

For $n = 2$:

$$A_2 = \frac{1}{2!} \frac{\partial^2}{\partial \lambda^2} \left[N \left(\sum_{k=0}^2 \lambda^k u_k \right) \right]_{\lambda=0},$$

$$A_2 = \frac{1}{2!} \frac{\partial^2}{\partial \lambda^2} [N(u_0 + \lambda u_1 + \lambda^2 u_2)]_{\lambda=0}$$

$$A_2 = \frac{1}{2!} \frac{\partial^2}{\partial \lambda^2} [(u_0)_{\xi\xi} + \lambda (u_1)_{\xi\xi} + \lambda^2 (u_2)_{\xi\xi}]^2_{\lambda=0}$$

$$A_2 = \frac{\partial}{\partial \lambda} [((u_0)_{\xi\xi} + \lambda (u_1)_{\xi\xi} + \lambda^2 (u_2)_{\xi\xi})((u_1)_{\xi\xi} + 2\lambda (u_2)_{\xi\xi})]_{\lambda=0}$$

$$[A_2 = [((u_1)_{\xi\xi} + 2\lambda (u_2)_{\xi\xi})^2 + ((u_0)_{\xi\xi} + \lambda (u_1)_{\xi\xi} + \lambda^2 (u_2)_{\xi\xi})(2(u_2)_{\xi\xi})]_{\lambda=0}]$$

$$A_2 = (u_1)_{\xi\xi}^2 + 2(u_0)_{\xi\xi} (u_2)_{\xi\xi}$$

Consider the nonlinear term $N(u) = \sin(u_\xi)$

$$N(u) = \sin(u_\xi)$$

$$A_n = \frac{1}{n!} \frac{\partial^n}{\partial \lambda^n} \left[N \left(\sum_{k=0}^n \lambda^k u_k \right) \right]_{\lambda=0},$$

For $n = 0$:

$$A_0 = N(u_0) = \sin(u_0)_\xi$$

For $n = 1$:

$$A_1 = \frac{\partial}{\partial \lambda} \left[N \left(\sum_{k=0}^1 \lambda^k u_k \right) \right]_{\lambda=0},$$

$$A_1 = \frac{\partial}{\partial \lambda} [N(u_0 + \lambda u_1)]_{\lambda=0}$$

$$A_1 = \frac{\partial}{\partial \lambda} [\sin(u_0 + \lambda u_1)_\xi]_{\lambda=0}$$

$$A_1 = (u_1)_\xi \cos(u_0 + \lambda u_1)_\xi \big|_{\lambda=0}$$

$$A_1 = (u_1)_\xi \cos(u_0)_\xi$$

For $n = 2$:

$$A_2 = \frac{1}{2!} \frac{\partial^2}{\partial \lambda^2} \left[N \left(\sum_{k=0}^2 \lambda^k u_k \right) \right]_{\lambda=0},$$

$$A_2 = \frac{1}{2!} \frac{\partial^2}{\partial \lambda^2} [N(u_0 + \lambda u_1 + \lambda^2 u_2)]_{\lambda=0}$$

$$A_2 = \frac{1}{2!} \frac{\partial^2}{\partial \lambda^2} [\sin((u_0)_\xi + \lambda(u_1)_\xi + \lambda^2(u_2)_\xi)]_{\lambda=0}$$

$$A_2 = \frac{1}{2!} \frac{\partial}{\partial \lambda} [\cos((u_0)_\xi + \lambda(u_1)_\xi + \lambda^2(u_2)_\xi)((u_1)_\xi + 2\lambda(u_2)_\xi)]_{\lambda=0}$$

$$A_2 = \frac{1}{2} [-\sin((u_0)_\xi + \lambda(u_1)_\xi + \lambda^2(u_2)_\xi)((u_1)_\xi + 2\lambda(u_2)_\xi)^2 + \cos((u_0)_\xi + \lambda(u_1)_\xi + \lambda^2(u_2)_\xi) 2(u_2)_\xi]_{\lambda=0}$$

$$A_2 = \cos(u_0)_\xi (u_2)_\xi - \frac{1}{2} \sin(u_0)_\xi (u_1)_\xi^2$$

A Review of Adomian Decomposition Method

Consider equation (3)

$$u_0(\xi, \eta) = \psi_0(\xi, \eta) + \beta(\xi, \eta) \quad (4)$$

where $\psi_0(\xi, \eta)$ is derived from Taylor's series using the boundary conditions as follows:

$$\psi_0(\xi, \eta) = u(0, \eta) + \xi u_\xi(0, \eta) + \frac{\xi^2}{2!} u_{\xi\xi}(0, \eta) + \dots + \frac{\xi^{n-1}}{(n-1)!} u^{(n-1)}(0, \eta) \quad (5)$$

Applying the boundary conditions, to have

$$\psi_0(\xi, \eta) = \beta_0 + \xi h(\eta) + \frac{\xi^2}{2!} h'(\eta) + \dots + \frac{\xi^{n-1}}{(n-1)!} h^{(n-2)}(\eta) \quad (6)$$

where $h(\eta)$ denotes $u_\xi(0, t)$, $h'(\eta) = u_{\xi\xi}(0, \eta) \dots$ and

$\beta(\xi, \eta)$ is obtained from application of inverse of the linear differential operator L to $g(\xi, \eta)$ in (3). That is,

$$\beta(\xi, \eta) = L^{-1} g(\xi, \eta) \quad (7)$$

Therefore, adding (6) and (7) yields the initial approximation $u_0(\xi, \eta)$,

$$u_0(\xi, \eta) = \beta_0 + \xi h(\eta) + \frac{\xi^2}{2!} h'(\eta) + \dots + \frac{\xi^{n-1}}{(n-1)!} h^{(n-2)}(\eta) + L^{-1}g(\xi, \eta) \quad (8)$$

The recurrence relation

$$u_{n+1}(\xi, \eta) = - \int_0^\xi \int_0^{\xi_{n-1}} \dots \int_0^{\xi_2} \int_0^{\xi_1} M u_n(\varepsilon, \eta) d\varepsilon d\xi_1 \dots d\xi_{n-1} \\ - \int_0^\xi \int_0^{\xi_{n-1}} \dots \int_0^{\xi_2} \int_0^{\xi_1} A_n(\varepsilon, \eta) d\varepsilon d\xi_1 \dots d\xi_{n-1}, \quad n = 0, 1, 2, \dots \quad (9)$$

where A_n is the Adomian polynomial of the nonlinear term given. Other members, that is, $u_1(\xi, \eta)$, $u_2(\xi, \eta)$... are obtained from the recurrence relation.

The final solution is obtained as,

$$u(\xi, \eta) = u_0(\xi, \eta) + u_1(\xi, \eta) + \dots \quad (10)$$

The initial conditions are then implemented in (6), after which the coefficients of like powers of ξ are compared. Through that process, the Taylor expansion produces the unknown term. That is then substituted back into (8) to get the exact solution.(Yisa, 2019).

Numerical Experiments

In this section, numerical examples are presented to demonstrate the method explained in section 3.

PROBLEM 1(Mohyud-Din, 2010)

Solve the two-dimensional initial-boundary value problem

$$u_{\eta\eta} = \frac{1}{2}\tau^2 u_{\xi\xi} + \frac{1}{2}\xi^2 u_{\tau\tau}, \quad 0 < \xi, \tau < 1, \eta > 0$$

with boundary conditions

$$u(0, \tau, \eta) = \tau^2 e^{-\eta}, \quad u(1, \tau, \eta) = (1 + \tau^2)e^{-\eta}$$

$$u(\xi, 0, \eta) = \xi^2 e^{-\eta}, \quad u(\xi, 1, \eta) = (1 + \xi^2)e^{-\eta}$$

and initial conditions

$$u(\xi, \tau, 0) = \xi^2 + \tau^2, \quad u_\eta(\xi, \tau, 0) = -(\xi^2 + \tau^2)$$

The exact solution for this problem is

$$u(\xi, \tau, \eta) = (\xi^2 + \tau^2)e^{-\eta}.$$

Solution

Solving in ξ -direction

$$u_{\xi\xi} = \frac{2}{\tau^2}u_{\eta\eta} - \frac{\xi^2}{\tau^2}u_{\tau\tau}$$

The initial approximation $u_0(\xi, \tau, \eta)$ is obtained as

$$u_0(\xi, \tau, \eta) = \psi_0(\xi, \tau, \eta) + \beta(\xi, \tau, \eta)$$

$$\psi_0(\xi, \tau, \eta) = u(0, \tau, \eta) + \xi u_\xi(0, \tau, \eta) = \tau^2 e^{-\eta} + \xi h(\eta),$$

where $h(\eta) = u_\xi(0, \tau, \eta)$

$$\beta(\xi, \tau, \eta) = 0$$

$$u_0(\xi, \tau, \eta) = \tau^2 e^{-\eta} + \xi h(\eta)$$

And the recurrence relation is derived as

$$u_{n+1}(\xi, \tau, \eta) = \int_0^\xi \int_0^s \left(\frac{2}{\tau^2} (u_n)_{\eta\eta} - \frac{\varepsilon^2}{\tau^2} (u_n)_{\tau\tau} \right) d\varepsilon ds, \quad n = 0, 1, 2, 3 \dots$$

When $n = 0$:

$$u_1(\xi, \tau, \eta) = \int_0^\xi \int_0^s \left(\frac{2}{\tau^2} (u_0)_{\eta\eta} - \frac{\varepsilon^2}{\tau^2} (u_0)_{\tau\tau} \right) d\varepsilon ds$$

$$u_1(\xi, \tau, \eta) = \int_0^\xi \int_0^s \left(\frac{2}{\tau^2} (\tau^2 e^{-\eta} + \varepsilon h(\eta))_{\eta\eta} - \frac{\varepsilon^2}{\tau^2} (\tau^2 e^{-\eta} + \varepsilon h(\eta))_{\tau\tau} \right) d\varepsilon ds$$

$$u_1(\xi, \tau, \eta) = \int_0^\xi \int_0^s \left(2e^{-\eta} + \frac{2\varepsilon}{\tau^2} h''(\eta) - \frac{2\varepsilon^2}{\tau^2} e^{-\eta} \right) d\varepsilon ds$$

$$u_1(\xi, \tau, \eta) = \xi^2 e^{-\eta} + \frac{\xi^3}{3\tau^2} h''(\eta) - \frac{\xi^4}{6\tau^2} e^{-\eta}$$

When $n = 1$:

$$u_2(\xi, \tau, \eta) = \int_0^\xi \int_0^s \left(\frac{2}{\tau^2} (u_1)_{\eta\eta} - \frac{\varepsilon^2}{\tau^2} (u_1)_{\tau\tau} \right) d\varepsilon ds$$

$$\begin{aligned} &= \int_0^\xi \int_0^s \left(\frac{2}{\tau^2} \left(\varepsilon^2 e^{-\eta} + \frac{\varepsilon^3}{3\tau^2} h''(\eta) - \frac{\varepsilon^4}{6\tau^2} e^{-\eta} \right)_{\eta\eta} \right. \\ &\quad \left. - \frac{\varepsilon^2}{\tau^2} \left(\varepsilon^2 e^{-\eta} + \frac{\varepsilon^3}{3\tau^2} h''(\eta) - \frac{\varepsilon^4}{6\tau^2} e^{-\eta} \right)_{\tau\tau} \right) d\varepsilon ds \end{aligned}$$

$$= \int_0^\xi \int_0^s \left(\frac{2\varepsilon^2}{\tau^2} e^{-\eta} + \frac{2\varepsilon^3}{3\tau^4} h''''(\eta) - \frac{2\varepsilon^4}{6\tau^4} e^{-\eta} + \frac{6\varepsilon^5}{3\tau^6} h''(\eta) + \frac{6\varepsilon^6}{6\tau^6} e^{-\eta} \right) d\varepsilon ds$$

$$u_2(\xi, \tau, \eta) = \frac{\xi^4}{6\tau^2} e^{-\eta} + \frac{2\xi^5}{60\tau^4} h''''(\eta) - \frac{2\xi^6}{180\tau^4} e^{-\eta} + \frac{6\xi^7}{126\tau^6} h''(\eta) + \frac{6\xi^8}{336\tau^6} e^{-\eta}$$

$$\text{Since } u_1(\xi, \tau, \eta) = \xi^2 e^{-\eta} + \frac{\xi^3}{3\tau^2} h''(\eta) - \frac{\xi^4}{6\tau^2} e^{-\eta}$$

This implies there exist noise terms in $u_1(\xi, \tau, \eta)$ and $u_2(\xi, \tau, \eta)$ terms, the exact solution is obtained as

$$u(\xi, \tau, \eta) = \xi^2 e^{-\eta} + \tau^2 e^{-\eta} + \xi h(\eta) + \frac{\xi^3}{3\tau^2} h''(\eta)$$

Applying the initial condition $u(\xi, \tau, 0) = \xi^2 + \tau^2$

$$u(\xi, \tau, 0) = \xi^2 + \tau^2 + \xi h(0) + \frac{\xi^3}{3\tau^2} h''(0) = \xi^2 + \tau^2$$

Comparing the coefficients of powers of ξ , we obtain

$$h(0) = 0, \quad h''(0) = 0$$

The Taylors expansion of $h(\eta) = 0$

Substitute $h(\eta) = 0$ in to $u(\xi, \tau, \eta)$

$$u(\xi, \tau, \eta) = \xi^2 e^{-\eta} + \tau^2 e^{-\eta} = (\xi^2 + \tau^2) e^{-\eta}$$

which is the exact solution.

Problem 2(Mohyud-Din, 2010)

Consider the following three-dimensional initial-boundary value problem

$$u_{\eta\eta} = \frac{1}{45} \xi^2 u_{\xi\xi} + \frac{1}{45} \tau^2 u_{\tau\tau} + \frac{1}{45} \vartheta^2 u_{\vartheta\vartheta} - u, \quad 0 < \xi, \tau < 1, \quad \eta < 0$$

Subject to Neumann boundary conditions:

$$u_\xi(0, \tau, \vartheta, t) = 0, \quad u_\xi(1, \tau, \vartheta, \eta) = 6\tau^6 \vartheta^6 \sinh \eta$$

$$u_\tau(\xi, 0, \vartheta, \eta) = 0, \quad u_\tau(\xi, 1, \vartheta, \eta) = 6\xi^6 \vartheta^6 \sinh \eta$$

$$u_\vartheta(\xi, \tau, 0, \eta) = 0, \quad u_\vartheta(\xi, \tau, 1, \eta) = 6\xi^6 \tau^6 \sinh \eta$$

and initial condition:

$$u(\xi, \tau, \vartheta, 0) = 0, \quad u_\eta(\xi, \tau, \vartheta, 0) = \xi^6 \tau^6 \vartheta^6$$

The exact solution for this problem is $u(\xi, \tau, \vartheta, \eta) = \xi^6 \tau^6 \vartheta^6 \sinh \eta$.

Solution

$$u_{\eta\eta} = \frac{1}{45} (\xi^2 u_{\xi\xi} + \tau^2 u_{\tau\tau} + \vartheta^2 u_{\vartheta\vartheta}) - u$$

The initial approximation $u_0(\xi, \tau, \vartheta, \eta)$ is obtained as

$$\begin{aligned} u_0(\xi, \tau, \vartheta, \eta) &= \psi_0(\xi, \tau, \vartheta, \eta) + \beta(\xi, \tau, \vartheta, \eta) \\ \psi_0(\xi, \tau, \vartheta, \eta) &= u(\xi, \tau, \vartheta, 0) + \eta u_\eta(\xi, \tau, \vartheta, 0) = (\xi^6 \tau^6 \vartheta^6) \eta, \\ \beta(\xi, \tau, \vartheta, \eta) &= 0 \\ u_0(\xi, \tau, \vartheta, \eta) &= (\xi^6 \tau^6 \vartheta^6) \eta \end{aligned}$$

And the recurrence relation is derived as

$$\begin{aligned} u_{n+1}(\xi, \tau, \vartheta, \eta) &= \int_0^\eta \int_0^s \left(\frac{1}{45} (\xi^2 (u_n)_{\xi\xi} + \tau^2 (u_n)_{\tau\tau} + \vartheta^2 (u_n)_{\vartheta\vartheta}) - u_n \right) d\epsilon ds, \quad n = 0, 1, 2, \dots \\ u_1(\xi, \tau, \vartheta, \eta) &= \int_0^\eta \int_0^s \left(\frac{1}{45} (\xi^2 (u_0)_{\xi\xi} + \tau^2 (u_0)_{\tau\tau} + \vartheta^2 (u_0)_{\vartheta\vartheta}) - u_0 \right) d\epsilon ds, \\ u_1(\xi, \tau, \vartheta, \eta) &= (\xi^6 \tau^6 \vartheta^6) \frac{\eta^3}{6} \\ u_2(\xi, \tau, \vartheta, \eta) &= (\xi^6 \tau^6 \vartheta^6) \frac{\eta^5}{120} \end{aligned}$$

The solution is given by

$$\begin{aligned} u(\xi, \tau, \vartheta, \eta) &= u_0(\xi, \tau, \vartheta, \eta) + u_1(\xi, \tau, \vartheta, \eta) + u_2(\xi, \tau, \vartheta, \eta) + \dots \\ u(\xi, \tau, \vartheta, \eta) &= (\xi^6 \tau^6 \vartheta^6) \eta + (\xi^6 \tau^6 \vartheta^6) \frac{\eta^3}{6} + (\xi^6 \tau^6 \vartheta^6) \frac{\eta^5}{120} \\ u(\xi, \tau, \vartheta, \eta) &= (\xi^6 \tau^6 \vartheta^6) \sinh \eta \end{aligned}$$

which is the exact solution.

Problem 3 (Mohyud-Din, 2010)

Consider the two-dimensional non-linear inhomogeneous initial-boundary value problem

$$u_{\eta\eta} = 2\xi^2 + 2\tau^2 + \frac{15}{2} (\xi u_{\xi\xi}^2 + \tau u_{\tau\tau}^2), \quad 0 < \xi, \tau < 1, \quad \eta > 0$$

with boundary conditions:

$$\begin{aligned} u(0, \tau, \eta) &= \tau^2 \eta^2 + \tau \eta^6, & u(1, \tau, \eta) &= (1 + \tau^2) \eta^2 + (1 + \tau) \eta^6 \\ u(\xi, 0, \eta) &= \xi^2 \eta^2 + \xi \eta^6, & u(\xi, 1, \eta) &= (1 + \xi^2) \eta^2 + (1 + \xi) \eta^6 \end{aligned}$$

and initial condition

$$u(\xi, \tau, 0) = 0, \quad u_\eta(\xi, \tau, 0) = 0.$$

The exact solution for this problem is

$$u(\xi, \tau, \eta) = (\xi^2 + \tau^2)\eta^2 + (\xi + \tau)\eta^6.$$

Solution

The initial approximation $u_0(\xi, \tau, \eta)$ is obtained as

$$u_0(\xi, \tau, \eta) = (\xi^2 + \tau^2)\eta^2$$

And the recurrence relation is derived as

$$u_{n+1}(\xi, \tau, \eta) = \frac{15}{2} \int_0^\eta \int_0^s (\xi A_n(\varepsilon) + \xi B_n(\varepsilon)) d\varepsilon ds, \quad n = 0, 1, 2, 3 \dots$$

where $A_n(\varepsilon)$ and $B_n(\varepsilon)$ are the Adomian polynomials of the nonlinear terms $u_{\xi\xi}^2$ and $u_{\tau\tau}^2$ respectively

$$\begin{aligned} u_1(\xi, \tau, \eta) &= (\xi + \tau)\eta^6 \\ u_2(\xi, \tau, \eta) &= 0 \end{aligned}$$

The result is given

$$u(\xi, \tau, \eta) = (\xi^2 + \tau^2)\eta^2 + (\xi + \tau)\eta^6.$$

Problem 4(Mohyud-Din, 2010)

Consider the three-dimensional nonlinear in homogeneous initial-boundary value problem

$$u_{\eta\eta} = (2 - \eta^2) + u - (e^{-\xi} u_{\xi\xi}^2 + e^{-\tau} u_{\tau\tau}^2 + e^{-\vartheta} u_{\vartheta\vartheta}^2), \quad 0 < \xi, \tau < 1, \quad \eta > 0$$

Subject to Neumann boundary conditions:

$$\begin{aligned} u_\xi(0, \tau, \vartheta, \eta) &= 1, & u_\xi(1, \tau, \vartheta, \eta) &= e \\ u_\tau(\xi, 0, \vartheta, \eta) &= 0, & u_\tau(\xi, 1, \vartheta, \eta) &= e. \\ u_\vartheta(\xi, \tau, 0, \eta) &= 1, & u_\vartheta(\xi, \tau, 1, \eta) &= e \end{aligned}$$

and initial conditions:

$$u(\xi, \tau, \vartheta, 0) = e^\xi + e^\tau + e^\vartheta, \quad u_\eta(\xi, \tau, \vartheta, 0) = 0$$

The exact solution for this problem is

$$u(\xi, \tau, \vartheta, \eta) = (e^\xi + e^\tau + e^\vartheta) + \eta^2.$$

Solution

The initial approximation $u_0(\xi, \tau, \vartheta, \eta)$ is obtained as

$$u_0(\xi, \tau, \vartheta, \eta) = e^\xi + e^\tau + e^\vartheta + \eta^2 - \frac{\eta^4}{12}$$

And the recurrence relation is derived as

$$u_{n+1}(\xi, \tau, \vartheta, \eta) = \int_0^\eta \int_0^s \left(u_n - e^{-\xi} A_n(\varepsilon) - e^{-\tau} B_n(\varepsilon) - e^{-\vartheta} C_n(\varepsilon) \right) d\varepsilon ds, \quad n = 0, 1, 2, 3 \dots$$

$$u_1(\xi, \tau, \vartheta, \eta) = \frac{\eta^4}{12} - \frac{\eta^6}{360}$$

Since there exist noise terms in u_1 and u_0 . Therefore, the exact solution is

$$u(\xi, \tau, \vartheta, \eta) = (e^\xi + e^\tau + e^\vartheta) + \eta^2$$

Problem 5 (Wazwaz, 2009)

Solve the one-dimensional initial-boundary value problem:

$$\begin{aligned} u_\eta &= u_{\xi\xi} - u, & 0 < \xi < \pi, \eta > 0, \\ u(0, \eta) &= 0, & u(\pi, \eta) &= 0, & u(\xi, 0) &= \sin \xi. \end{aligned}$$

Solution

Solving in the ξ -direction,

$$u_{\xi\xi} = u_\eta + u$$

The initial approximation $u_0(\xi, \eta)$ is obtained as

$$u_0(\xi, \eta) = \xi h(\eta),$$

And the recurrence relation is derived as

$$u_{n+1}(\xi, \eta) = \int_0^\xi \int_0^s \left((u_n)_\eta + u_n \right) d\varepsilon ds, \quad n = 0, 1, 2, 3 \dots$$

$$u_1(\xi, \eta) = \frac{\xi^3}{6} h'(\eta) + \frac{\xi^3}{6} h(\eta)$$

$$u_2(\xi, \eta) = \frac{\xi^5}{120} h''(\eta) + \frac{\xi^5}{60} h'(\eta) + \frac{\xi^5}{120} h(\eta)$$

The result is obtained as

$$u(\xi, \eta) = \xi h(\eta) + \frac{\xi^3}{3!} (h'(\eta) + h(\eta)) + \frac{\xi^5}{5!} (h''(\eta) + 2h'(\eta) + h(\eta))$$

Applying initial condition $u(\xi, 0) = \sin \xi$, we have,

$$\begin{aligned} u(\xi, 0) &= \xi h(0) + \frac{\xi^3}{3!} (h'(0) + h(0)) + \frac{\xi^5}{5!} (h''(0) + 2h'(0) + h(0)) \\ &= \xi - \frac{\xi^3}{3!} + \frac{\xi^5}{5!} - \dots \end{aligned}$$

Comparing the coefficients of powers of ξ , we obtain

$$h(0) = 1, \quad h'(0) = -2, \quad h''(0) = 4$$

The Taylor expansion of $h(\eta)$ gives

$$h(\eta) = e^{-2\eta}$$

Substituting in to $u(\xi, \eta)$,

$$\begin{aligned} u(\xi, \eta) &= e^{-2\eta} \left(\xi - \frac{\xi^3}{3!} + \frac{\xi^5}{5!} \right) \\ u(\xi, \eta) &= e^{-2\eta} \sin \xi \end{aligned}$$

which is the exact solution.

Problem 6(Wazwaz, 2010)

Solve the one-dimensional initial-boundary value problem:

$$\begin{aligned} u_\eta &= u_{\xi\xi} - 2\pi^2 u, \quad 0 < \xi < \pi, \quad \eta > 0, \\ u(0, \eta) &= 0, \quad u(1, \eta) = 0, \quad u(\xi, 0) = \sin(\pi\xi). \end{aligned}$$

Solution

Solving in the ξ -direction,

$$u_{\xi\xi} = u_\eta + 2\pi^2 u$$

The initial approximation $u_0(\xi, \eta)$ is obtained as

$$u_0(\xi, \eta) = \xi h(\eta).$$

And the recurrence relation is derived as

$$\begin{aligned} u_{n+1}(\xi, \eta) &= \int_0^\xi \int_0^s ((u_n)_\eta + 2\pi^2 u_n) d\epsilon ds, \quad n = 0, 1, 2, 3 \dots \\ u_1(\xi, \eta) &= \frac{\xi^3}{6} h'(\eta) + \frac{\pi^2 \xi^3}{3} h(\eta) \\ u_2(\xi, \eta) &= \frac{\xi^5}{120} h''(\eta) + \frac{2\pi^2 \xi^5}{60} h'(\eta) + \frac{2\pi^4 \xi^5}{60} h(\eta) \end{aligned}$$

The result is obtained as

$$u(\xi, \eta) = \xi h(\eta) + \frac{\xi^3}{3!} (h'(\eta) + 2\pi^2 h(\eta)) + \frac{\xi^5}{5!} (h''(\eta) + 4\pi^2 h'(\eta) + 4\pi^4 h(\eta))$$

Applying initial condition $u(\xi, 0) = \sin(\pi\xi)$, we have,

$$\begin{aligned} u(\xi, 0) &= \xi h(0) + \frac{\xi^3}{3!} (h'(0) + 2\pi^2 h(0)) + \frac{\xi^5}{5!} (h''(0) + 4\pi^2 h'(0) + 4\pi^4 h(0)) \\ &= \pi\xi - \frac{(\pi\xi)^3}{3!} + \frac{(\pi\xi)^5}{5!} - \dots \end{aligned}$$

Comparing the coefficients of powers of ξ , we obtain

$$h(0) = \pi, \quad h'(0) = -3\pi^3, \quad h''(0) = 9\pi^5$$

The Taylor expansion of $h(\eta)$ gives

$$h(\eta) = \pi e^{-3\pi^2\eta}$$

Substituting in to $u(\xi, \eta)$,

$$\begin{aligned} u(\xi, \eta) &= e^{-3\pi^2\eta} \left(\pi\xi - \frac{(\pi\xi)^3}{3!} + \frac{(\pi\xi)^5}{5!} \right) \\ u(\xi, \eta) &= e^{-3\pi^2\eta} \sin(\pi\xi) \end{aligned}$$

which is the exact solution.

Problem 7(Wazwaz, 2010)

Solve the one-dimensional initial-boundary value problem:

$$\begin{aligned} u_\eta &= u_{\xi\xi} + \sin(2\xi), \quad 0 < \xi < \pi, \quad \eta > 0, \\ u(0, \eta) &= 0, \quad u(\pi, \eta) = 0, \quad u(\xi, 0) = \sin(\xi) + \frac{1}{4} \sin(2\xi). \end{aligned}$$

Solution

Solving in the ξ -direction,

$$u_{\xi\xi} = u_\eta - \sin(2\xi)$$

The initial approximation $u_0(\xi, \eta)$ is obtained as,

$$u_0(\xi, \eta) = \xi h(\eta) + \frac{1}{4} \sin(2\xi)$$

And the recurrence relation is derived as

$$u_{n+1}(\xi, \eta) = \int_0^\xi \int_0^s (u_n)_\xi d\epsilon ds, \quad n = 0, 1, 2, 3 \dots$$

$$u_1(\xi, \eta) = \frac{\xi^3}{6} h'(\eta)$$

$$u_2(\xi, \eta) = \frac{\xi^5}{120} h''(\eta)$$

The result is obtained as,

$$u(\xi, \eta) = \xi h(\eta) + \frac{1}{4} \sin(2\xi) + \frac{\xi^3}{3!} h'(\eta) + \frac{\xi^5}{5!} h''(\eta)$$

Applying initial condition $u(\xi, 0) = \sin(\xi) + \frac{1}{4} \sin(2\xi)$, we have,

$$u(\xi, 0) = \xi h(0) + \frac{1}{4} \sin(2\xi) + \frac{\xi^3}{3!} h'(0) + \frac{\xi^5}{5!} h''(0) = \xi - \frac{\xi^3}{3!} + \frac{\xi^5}{5!} + \frac{1}{4} \sin(2\xi).$$

Comparing the coefficients of powers of ξ , we obtain

$$h(0) = 1, \quad h'(0) = -1, \quad h''(0) = 1$$

The Taylor expansion of $h(\eta)$ gives

$$h(\eta) = e^\eta$$

Substituting in to $u(\xi, \eta)$,

$$u(\xi, \eta) = e^{-\eta} \left(\xi - \frac{\xi^3}{3!} + \frac{\xi^5}{5!} \right) + \frac{1}{4} \sin(2\xi)$$

$$u(\xi, \eta) = e^{-\eta} \sin \xi + \frac{1}{4} \sin(2\xi)$$

which is the exact solution.

Problem 8(Wazwaz, 2009)

Solve the one-dimensional initial-boundary value problem:

$$u_\eta = u_{\xi\xi} - 6, \quad 0 < \xi < \pi, \quad \eta > 0,$$

$$u(0, \eta) = e^{-\eta}, \quad u(\pi, \eta) = 3\pi^2 - e^{-\eta}, \quad u(\xi, 0) = 3\xi^2 + \cos(\xi)$$

Solution

Solving in the ξ -direction,

$$u_{\xi\xi} = u_\eta + 6$$

The initial approximation $u_0(\xi, \eta)$ is

$$u_0(\xi, \eta) = e^{-\eta} + \xi h(\eta) + 3\xi^2$$

And the recurrence relation is derived as

$$u_{n+1}(\xi, \eta) = \int_0^\xi \int_0^s (u_n)_\eta d\epsilon ds, \quad n = 0, 1, 2, 3 \dots$$

$$u_1(\xi, \eta) = -\frac{\xi^2}{2} e^{-\eta} + \frac{\xi^3}{6} h'(\eta)$$

$$u_2(\xi, \eta) = \frac{\xi^4}{24} e^{-\eta} + \frac{\xi^5}{120} h''(\eta)$$

The result is obtained as,

$$u(\xi, \eta) = e^{-\eta} + \xi h(\eta) + 3\xi^2 - \frac{\xi^2}{2} e^{-\eta} + \frac{\xi^3}{6} h'(\eta) + \frac{\xi^4}{24} e^{-\eta} + \frac{\xi^5}{120} h''(\eta)$$

Applying initial condition $u(\xi, 0) = 3\xi^2 + \cos(\xi)$, we have,

$$\begin{aligned} u(\xi, 0) &= 1 + \xi h(0) + 3\xi^2 - \frac{\xi^2}{2} + \frac{\xi^3}{6} h'(0) + \frac{\xi^4}{24} + \frac{\xi^5}{120} h''(0) \\ &= 3\xi^2 + 1 - \frac{\xi^2}{2} + \frac{\xi^4}{4!} - \frac{\xi^6}{6!} \end{aligned}$$

Comparing the coefficients of powers of ξ , we obtain

$$h(0) = 0, \quad h'(0) = 0, \quad h''(0) = 0$$

The Taylor expansion of $h(\eta)$ gives

$$h(\eta) = 0.$$

and the solution give

$$\begin{aligned} u(\xi, \eta) &= 3\xi^2 + e^{-\eta} \left(1 - \frac{\xi^2}{2} + \frac{\xi^4}{4!} - \frac{\xi^6}{6!} \right) \\ u(\xi, \eta) &= 3\xi^2 + e^{-\eta} \cos(\xi) \end{aligned}$$

which is the exact solution.

Discussion of Results

The results obtained for the entire problems 1, 2, 3, and 4 are all exact solutions. The first two problems are linear initial-boundary value PDEs, while the following two are nonlinear. Adomian polynomials are used to overcome the nonlinear terms in problems 3 and 4. The four problems were earlier solved by (Mohyud-Din, 2010) when

he used variation of parameter approach. While our approach gives exact solutions for both linear and nonlinear problems, the approach proposed in the referenced literature is cumbersome and really gives exact solutions. Problems 5, 6, 7, and 8 are taken from (Abdul-Majid, 2009) where he used ADM to solve the problems. Our results tally with the author's results, being exact solutions in the four cases. The present method is ADM fused with Taylor's series of two variables has proven to be a powerful tool for providing solutions to problems of linear and nonlinear partial differential equations.

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Effect of Varying Concentrations of Plant Growth Regulators on *In-Vitro* Multiplication of Sugarcane (*Saccharum Officinarum* L.) using Shoot-Tip Culture

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Abstract

Despite the introduction of improved sugarcane varieties, their large-scale propagation and distribution remain limited due to the inefficiency of conventional propagation methods. Plant tissue culture enables rapid propagation of high-quality, disease-free planting materials. This research evaluated the effects of different concentrations of plant growth regulators on the establishment of in vitro culture and shoot multiplication of the sugarcane variety CO-002 using shoot tip explants. Four Murashige and Skoog (MS) media supplemented with varying concentrations of plant growth regulators were tested: TI (control) lacks growth hormones, TII (2.0 mg/L BAP, 0.5 mg/L kinetin, and 0.5 mg/L IAA); TIII (3.0 mg/L BAP, 1.5 mg/L kinetin, and 1.0 mg/L IAA), and TIV (4.0 mg/L BAP, 2.5 mg/L kinetin, and 1.5 mg/L IAA). Data were subjected to analysis of variance and the significant means were separated using Duncan multiple-range test at ($p \leq 0.05$). The earliest shoot emergence, the highest number of shoots per explant, and shoot length were observed at TII, followed by TIII, whilst TIV demonstrated the lowest performance, and all compared better than the control TI.

Keywords: Sugarcane, Shoot-tip, 6-Benzylaminopurine, Indole acetic acid, Explants

Introduction

Sugarcane (*Saccharum officinarum* L.) is a member of the grass family; it is a tall, perennial plant species grown worldwide (Hailu et al., 2018). It is a major crop that is the primary source of sugar production, accounting for 80% of global consumption, followed by ethanol as the second-largest producer and consumer (Hailu et al., 2018). Sugarcane is widely cultivated in tropical and sub-tropical regions and originated from New Guinea (Thibane et al., 2023). Brazil is the world's largest producer of sugarcane, primarily due to its soil-related and environmental conditions, which contribute substantially to global sugar and ethanol production. In contrast, India is ranked second among sugarcane producers, largely due to advancements in plant biotechnology (Krishanu et al., 2023).

In Nigeria, sugarcane is predominantly propagated through conventional methods using stem cuttings (setts), resulting in low propagation efficiency with a multiplication ratio of approximately 1:8 due to the slow multiplication rate of newly introduced varieties (Usman, 2015; Ajadi et al., 2018). The rapid propagation of sugarcane seeds through traditional methods usually exceeds ten years for a new or improved sugarcane seedling to be adequately multiplied for planting in a large expanse of land (Sharma et al., 2015; Krishanu et al., 2023).

The repeated use of sugarcane setts for propagation allows the accumulation of pathogens over successive cycles, leading to reduced performance of improved varieties (Abdu et al., 2012; Usman, 2015). The prevalence of disease and low cane yield are major constraints hindering the mass production of commercial and chewing sugarcane in Nigeria (Usman, 2015).

To address the multiple problems associated with the non-biotechnological approach, sugarcane factories should adopt new advances in plant biotechnology, specifically in vitro propagation, which enables rapid proliferation of sugarcane varieties to produce high-quality, disease-resistant sugarcane (Hailu et al., 2018).

Micropropagation, also referred to as in vitro propagation, is a method for generating new plants under controlled environmental conditions by culturing selected tissues (explants) of the target plant on a sterile nutrient medium, performed under aseptic techniques (Krishanu et al., 2023). In vitro propagation offers the most effective and viable approach for generating top-quality, mass-produced sugarcane seedlings from organs, tissues, or cells that are true to the type of a desired plant as the original plant, using plant tissue culture techniques (Krishanu et al., 2023). On the other hand, in traditional methods, where a single bud develops 4-5 shoots, the tissue culture technique can develop hundreds to thousands of shoot formations and thousands of seedlings after repeated cycles of culturing from a single shoot tip in a few months (Tesfa et al., 2016). Therefore, there is a need to supply disease-free and pest/pathogen-resistant planting materials, and provision of high-yielding and quality sugarcane varieties with rapid mass proliferation of newly released varieties within a short period (Usman, 2015; Tesfa et al., 2016).

Tissue culture of sugarcane planting material relies on specific genotypes and plant growth hormone/regulator (PGR) compositions, which are used and required to develop PGR protocol combinations for specific genotypes (Melaku et al., 2016; Amente&Feyissa, 2022). PGR is an essential component of in vitro sugarcane propagation, and responses differ among varieties depending on the composition and combination of PGR in the nutrient culture medium (Amente&Feyissa, 2022). It is of utmost importance to develop an efficient, productive protocol for the newly introduced or improved sugarcane variety to promote rapid shoot initiation and shoot proliferation (Amente&Feyissa, 2022). Thus, this study was designed to investigate the effect of varying concentrations of PGR on in vitro multiplication of the sugarcane variety CO-002 using shoot-tip explants.

Materials and Methods

The study was conducted at the Plant Tissue Culture Laboratory, C.G. Bhakta Institute of Biotechnology, Uka Tarsadia University, Gujarat, India. The plant material used for the experiment was sugarcane (*Saccharum officinarum*) shoot tips. The explants were obtained from the variety CO-002, sourced from the Plant Tissue Culture Farm, Uka Tarsadia University, Gujarat, India. The shoot tips were selected as explants due to their high regenerative potential and suitability for in vitro culture studies.

Culture Media Preparation and Sterilisation

Murashige and Skoog (1962) medium was used as the basal culture medium. The standard composition of macronutrients, micronutrients, iron source, vitamins, and organic compounds was maintained without modification. Sucrose was adjusted to 25 g/L and kept constant throughout the experiment.

The medium was prepared both without hormones (control) and supplemented with varying concentrations of plant growth regulators: BAP (2, 3, and 4 mg/L), Kinetin (0.5, 1.5, and 2.5 mg/L), and IAA (0.5, 1.0, and 1.5 mg/L). Stock solutions of each PGR were prepared by dissolving the appropriate amount of the pure chemical in a small volume of solvent (BAP and Kinetin in 1 N NaOH, IAA in a small volume of ethanol), and then diluted to the desired concentration with double-distilled water. Stock solutions were stored at 4°C in amber bottles until use. All medium components were prepared from stock solutions and dissolved in double-distilled water. The final volume of the medium was divided into appropriate portions, and the required amounts of PGRs from the stock solutions were added to achieve the desired working concentrations.

Before gelling with agar, the pH of the medium was adjusted to 5.75 using 1N NaOH or 1N HCl. Agar was then added, and the mixture was heated on a magnetic hot plate until completely dissolved. The prepared medium was dispensed into 50 mL culture jars or McCartney bottles, which had been cleaned and dried, and the containers were properly closed. The medium was then autoclaved at 121°C under 15 psi for 15 minutes to ensure sterility.

Explant Collection and Surface Sterilisation

This study used only one sugarcane cultivar, CO-002. Shoot tips were collected from healthy, six-month-old mother plants cultivated in the greenhouse. Removing the leaf sheath revealed a healthy young shoot tip. It was subsequently taken to the Plant Tissue Culture Laboratory at Uka Tarsadia University, Gujarat, India, for explant preparation.

The shoot tips were cut and trimmed to 8 centimetres. The explants were thoroughly washed with clean tap water for 20 to 30 minutes, 0.2% bavistin was added for 10 minutes. They were then washed with 0.5 millilitres of Tween 20 (an organic solvent) and subsequently rinsed with distilled water to fully remove the foam. Sterile water was mixed with a small amount of citric and ascorbic acid, and an additional 2 grams of Bavistin. The liquid was then stirred for 20 minutes. A small amount of ascorbic acid and citric acid was added. The shoot tips were then clipped at both ends and resized to form explants measuring 1.0 cm to 1.5 cm. The explants were surface-sterilised by pouring 50 mL of sodium hypochlorite into the conical flask containing them, stirring for 15 minutes, and discarding the solution. After being transferred to a laminar airflow cabinet, it was treated for an additional 5 minutes with 0.1% (w/v) mercuric chloride (HgCl₂), rinsed with sterile water, and injected into sterile nutritional agar media that had already been packed in culture tubes. All of the aforementioned operations were performed aseptically in a laminar airflow cabinet.

Shoot Establishment

Shoot tips containing apical meristems from the sugarcane genotype CO-002 were cultured on Murashige and Skoog (MS) medium supplemented with sucrose (25 g/L), which remained constant throughout the experiment. The medium was further supplemented with four hormonal treatments selected from the literature for optimal sugarcane shoot emergence, including a control treatment without plant growth regulators: BAP (2.0, 3.0, 4.0 mg/L), Kinetin (0.5, 1.5, 2.5 mg/L), and IAA (0.5, 1.0, 1.5 mg/L). Each treatment was applied to six McCartney bottles, with one explant per bottle, providing six technical replicates, of which three were used for data collection. Cultures were subcultured every 21 days, a standard interval for sugarcane culture to ensure nutrient availability and healthy shoot growth.

Shoot establishment was assessed three weeks after culture by recording the percentage of explants remaining alive and shoot emergence.

***In vitro* Shoot Proliferation**

For the experiment, microshoots developed from comparable-sized shoot tip explants were used after the initiation stage. To avoid the carryover effect of the initiation medium, explants were grown on plant growth regulator-free media.

Murashige & Skoog's Media (TI) lack growth regulators TII, MS was utilized incorporated with different combinations and concentrations of Benzylaminopurine (BAP) (2mg/l), kinetin (0.5mg/l) and IAA (0.5mg/l); TIII, MS was enriched with BAP

(3mg/l), kinetin (1.5mg/l) and IAA (1mg/l); and finally TIV, MS was supplemented with BAP (4mg/l), kinetin (2.5mg/l) and IAA (1.5mg/l). In contrast, sucrose concentration remained constant at 25 g/L in all the treatment groups.

After adjusting the pH to 5.75, the culture media were autoclaved at 121°C under 15 psi for 15 minutes to ensure sterility. The study was conducted at 24°C ± 2°C, with 16 hrs of light and 8 hrs of dark, under fluorescent light at 2500-3000 lux, and in a growth chamber with relative humidity of 65% to 70%. After 30 days of culture, data on shoot emergence, shoot count per culture, and shoot length were taken and carefully documented after each subculturing. The collected data were analysed using analysis of variance (ANOVA).

Data Analysis

The experiment was conducted using MS medium supplemented with four different PGR treatments. Data were collected on shoot initiation and development parameters. The data were analyzed using analysis of variance (ANOVA), and significant differences among means were determined using Duncan's multiple range test at $p \leq 0.05$. Results were expressed as mean ± standard error of the mean (SEM) based on three replicates.

Results & Discussion

Effect of Varying Concentrations of 6-Benzylaminopurine on Culture Establishment from Shoot Tips Explants of CO-002 Sugarcane Variety

The results from shoot tip explants of the CO-002 sugarcane variety showed that percentage culture initiation was influenced by varying concentrations of BAP (Table 1). The highest culture initiation (88.67%) was recorded for the BAP treatment (2.00 mg/l), a subsequent higher culture initiation (72.33%) was observed at the BAP treatment (3.00 mg/l), and the BAP treatment (4.00 mg/l) had 54.00%, while the BAP treatment at (0.00 mg/l) recorded the lowest culture initiation, 41.33%. The grand mean culture initiation was 64.08% with a coefficient of variation (CV) of 19.40%. The results indicate that the optimal culture initiation concentration was 2.00 mg/L BAP.

Culture initiation of sugarcane shoot tip explants (CO-002) was significantly influenced by BAP concentration, as indicated by the variation among treatments. The superior performance observed at 2.00 mg/L suggests it is the optimal concentration for culture initiation, producing a significantly higher response compared to the control (0.00 mg/L) and higher concentrations. The reduced initiation at 3.00 and 4.00 mg/L indicates that increasing BAP beyond the optimal level may have an inhibitory effect on explant responsiveness. Overall, the results demonstrate that moderate BAP concentration significantly enhances culture initiation, while both lower (absence of hormone) and higher concentrations result in comparatively reduced responses.

Table 1: Effect of Varying Concentrations of 6-Benzylaminopurine on Culture Establishment from Shoot Tips Explants of CO-002 Sugarcane Variety

BAP (Treatments)	Culture Initiation $\pm$ SEM
TI (0.00 mg/dl BAP)	41.33 $\pm$ 2.33 ^a
TII (2.00 mg/dl BAP)	88.67 $\pm$ 0.88 ^d
TIII (3.00 mg/dl BAP)	72.33 $\pm$ 1.46 ^c
TIV (4.00 mg/dl BAP)	54.00 $\pm$ 1.15 ^b
Mean	64.08
CV	19.40

Values are indicated as mean $\pm$ SEM (n = 3). Data values with different superscripts differ significantly (p < 0.05).

The shoot tip explants on treatment TI, which is MS media (control) lacking BAP, recorded the lowest culture initiation rate (41.33%), suggesting that the endogenous cytokinin present in the shoot tip was inadequate to effectively activate meristematic effect and support the establishment of the explant in an artificial environment. However, the enhanced shoot establishment was observed in MS media enriched with various BAP concentrations (0.00, 2.00, 3.00, and 4.00 mg/l). The highest culture establishment (88.67%) was observed in MS medium enriched with 2.0 mg/l BAP, indicating that the cytokinin concentrations provide the optimal level required to stimulate cell division and growth and to aid axillary shoot multiplication in CO-002 sugarcane variety shoot tip explants. An additional increase in BAP concentration above the optimal level resulted in a significant decline in the percentage of culture establishment. The TIII, which is the BAP supplemented at 3.00 mg/l, the culture initiation reduced to 72.33% whilst the 4.00 mg/l concentration TIV was decreased to 54.00%. The decline in culture initiation at BAP concentrations above the optimal level may be attributed to inhibitory effects, hormonal disruption, and physiological stress, which can suppress organised meristematic shoot formation.

Several authors have demonstrated that the percentage of culture establishment relies on cytokinin and auxin concentrations and on the genotypes of sugarcane varieties (Biradar et al., 2008; Talihun et al., 2014). In this study, optimal culture initiation was recorded at 2.00 mg/l BAP, consistent with the report by Tilahun and colleagues (2014), who found that 2.00 mg/l BAP + 0.50 mg/l IBA produced normal establishment in two different sugarcane cultivars, Co449 and Co678. A previous study by Biradar et al. (2008) also reported that shoot initiation/establishment produced optimal outcomes at 2.00 mg/L BAP for CoC-671 sugarcane cultivars.

Effect of Varying Hormonal Concentrations on the Number of Days to Shoot Emergence from Shoot Tip Explants of CO-002 Sugarcane Variety

The results showed that the days required to shoot emergence from shoot tip explants of the CO-002 were influenced by varying hormonal levels of BAP, Kinetin, and Indole-3-acetic acid (IAA) (Table 2). The earliest shoot emergence (13.00 $\pm$ 0.58^c days)

was found at 2.00 mg/l BAP + 0.50 mg/l kinetin + 0.50 mg/l IAA (TII), indicating that lower hormonal concentrations can accelerate shoot establishment in culture. In contrast, the slower emergence days 20.00 ± 0.58 were observed at 3.00 mg/l BAP + 1.50 mg/l kinetin + 1.00 mg/l IAA (TIV), suggesting a delayed response to shoot emergence. At the same time, the TI and TIII showed shoot-emergence days that were not significantly different ($p < 0.05$). The grand mean across all treatment groups was 16.67%, and the coefficient of variation (CV) was 5.75%. The result suggests that TII treatments enhanced faster shoot emergence when compared with other treatment groups.

Table 2: Effect of Varying Hormonal Concentrations from Shoot tips Explants on Shoot Emergence of Sugarcane Variety CO-002

Treatments	Hormones			Mean Shoot $\pm$ SEM
	BAP	Kinetin	IAA	
T I	0.00	0.00	0.00	17.33 ± 0.67^b
T II	2.00	0.50	0.50	13.00 ± 0.58^c
T III	3.00	1.50	1.00	16.33 ± 0.33^b
T IV	4.00	2.50	1.50	20.00 ± 0.58^a
Grand Mean				16.67
CV (%)				5.75

Values are indicated as mean $\pm$ SEM ($n = 3$). Data values with distinct superscripts are significantly different ($p < 0.05$).

The earliest shoot emergence was observed in the MS media enriched with PGR at varying concentrations and combinations. However, a slow response was recorded in MS media (TI) without PGR. Earlier studies reported that delayed shoot emergence from shoot tip explants of the C86-12 sugarcane variety was observed in medium without PGRs (Hailu et al., 2018). The previous study reported that endogenous hormones present in the shoot tip were insufficient to activate optimal shoot growth. In this research, the shortest time to shoot response was 13 days for the TII treatment, with hormonal concentrations of 2.00 mg/l BAP, 0.50 mg/l kinetin, and 0.50 mg/l IAA, which demonstrated the best shoot response. The TIII recorded a duration of 16 days to shoot emergence, followed by the TI (control), which is PGR-free, with 17 days, and lastly, the TIV recorded 20 days.

The results showed that varying hormonal concentrations and combinations can influence the number of days to shoot response. Therefore, the shoot response that produced optimal shoots at the shortest time required minimal hormone concentrations of 2.00 mg/l BAP + 0.50 mg/l kinetin + 0.50 mg/l IAA, whilst concentrations above the optimal level increased the number of days to shoot response. Thus, suggesting a delay in the shoot response due to higher PGR concentrations and their combination. The findings align with Hailu et al. (2018), who reported that the sugarcane cultivar C86-165 on media enriched with 2.00 mg/l and 0.50 mg/l NAA showed the best shoot emergence. The study showed that the earliest shoot response occurred at lower hormone concentrations

observed at TII; however, concentrations above the optimal level resulted in a slower response time required to shoot emergence.

Effect of Varying Hormonal Concentrations on the Shoot Number per Explant of the CO-002 Sugarcane Variety

The results showed that the number of shoot tips per explant of the CO-002 sugarcane variety was influenced by varying concentrations of BAP, kinetin, and IAA (Table 3). The maximum response for the number of shoots was 23.00 at 2.00 mg/l BAP + 0.50 mg/l kinetin + 0.50 mg/l IAA (TII), suggesting that the hormone concentrations are optimal for shoot formation. The increase in hormonal concentrations above this level (TIII and TIV) significantly decreased ($p < 0.05$) the shoot count to 18.00 and 14.67, respectively, indicating that higher concentrations of hormones reduced shoot development. The grand mean across all treatment groups was 16.17%, and the coefficient of variation (CV) was 5.65%. There was a significant increase ($p < 0.05$) in the shoot number per explant when compared with the treatment group lacking PGR (TI); however, TII produced the highest shoot count per explant.

Table 3: Effect of Varying Hormonal Concentrations on the Shoot Number per Explant of Sugarcane Variety CO-002

Treatments	Hormones			Number of shoots per explant $\pm$ SEM
	BAP	Kinetin	IAA	
T I	0.00	0.00	0.00	9.00 $\pm$ 0.58 ^a
T II	2.00	0.50	0.50	23.00 $\pm$ 0.57 ^d
T III	3.00	1.50	1.00	18.00 $\pm$ 0.58 ^c
T IV	4.00	2.50	1.50	14.67 $\pm$ 0.33 ^b
Grand Mean				16.17
CV (%)				5.65

Values are indicated as mean $\pm$ SEM ($n = 3$). Data values with distinct superscripts are significantly different ($p < 0.05$).

The results demonstrated that the treatments TII had the highest number (23.00 ± 1.00) of shoots per explant in the CO-002 sugarcane variety, while the TI treatment lacking PGR had the lowest number (9.00 ± 0.58) of shoots per explant. The TII treatment enriched with 2.00 mg/l BAP + 0.50 mg/l kinetin + 0.50 mg/l IAA produced the best outcome, indicating that it contains the required concentration and combination of cytokinin and auxin to support axillary shoot multiplication. Importantly, cytokinin, especially BAP, has been reported to promote cell division and shoot proliferation while a minimal concentration of auxin was also known to enhance meristematic activity (Krishanu et al. 2023).

The TIII and TIV, supplemented with PGR concentration beyond the optimal level results in a decline in shoot count per explant. The decrease in the shoot count per

explant may be ascribed to supra-optimal cytokinin and auxin levels, which may hinder shoot growth. This is in agreement with previous studies that reported that higher concentrations of BAP and kinetin result in a decline in shoot formation (Kumar et al. 2020; Azu et al. 2022; Krishanu et al., 2023). The findings showed that a varying concentration and combination of 2.00 mg/l BAP + 0.50 mg/l kinetin + 0.50 mg/l IAA is essential for optimal shoot generation per explant for the CO-002 sugarcane variety, with TII recorded as the most ideal PGR combination for optimal shoot outcome, while TI with no PGR exhibited the lowest shoot formation per explant.

Effect of Varying Hormonal Concentrations on the Shoot Length of the CO-002 Sugarcane Variety

The results showed the shoot length of the CO-002 sugarcane variety with the varying hormonal concentrations (Table 4). The shoot length was significantly enhanced ($p < 0.05$) in shoot tip explants treated with plant growth regulators (PGRs) than in explant culture treated with the free PGRs. The maximum shoot length was observed with TII at 2.00 mg/l BAP + 0.50 mg/l kinetin + 0.50 mg/l IAA, demonstrating that hormone concentration can influence explant growth and improve shoot length. However, the shortest shoot length was 3.37 ± 0.12 at 0.00 mg/l BAP + 0.00 mg/l kinetin + 0.00 mg/l IAA (TI), while TIII exhibited an intermediate shoot length of 5.70 ± 0.20 and was followed by TIV at 4.10 ± 0.20 . All treatment groups had an average of 4.98% and a coefficient of variation (CV) of 3.45%.

Table 4: Effect of Varying Hormonal Concentrations on the Shoot Length of Sugarcane Variety CO-002

Treatments	Hormones			Shoot Length $\pm$ SEM
	BAP	Kinetin	IAA	
T I	0.00	0.00	0.00	3.37 ± 0.07^a
T II	2.00	0.50	0.50	6.77 ± 0.09^d
T III	3.00	1.50	1.00	5.70 ± 0.12^c
T IV	4.00	2.50	1.50	4.10 ± 0.12^b
Grand Mean				4.98
CV (%)				3.45

Values are indicated as mean $\pm$ SEM ($n = 3$). Data values with distinct superscripts are significantly different ($p < 0.05$).

The results demonstrated significant improvement ($p < 0.05$) in the shoot length per explant treated with PGR compared with explants not treated with PGR. Significant difference ($p < 0.05$) was observed among explants treated with varying concentrations and combinations of PGR. The cultured explants on TII, MS media containing 2.00 mg/l BAP + 0.50 mg/l kinetin + 0.50 mg/l IAA, demonstrated significantly higher ($p < 0.05$) shoot length with an average of 6.77, when compared to all other PGR-treated groups. This suggests that these concentrations and combinations produced the best outcome for

explant shoot elongation, which indicates synergistic interaction between the optimal level of cytokinin and auxin. Previous research had reported that higher shoot length was observed in cultured samples on MS media enriched with 2.0 mg/l BAP and 0.5 mg/l NAA compared to all other treatments for the variety C 86-165 (Hailu et al. 2018).

However, the TIII augmented with 3.00 mg/L BAP, 1.50 mg/L kinetin, and 1.00 mg/L IAA recorded a slight decrease in shoot length with a mean of 5.70 when compared with TII. In addition, an increase in the concentration of hormones in TIV leads to a significant decline ($p < 0.05$) in the shoot length of the explant with an average of 4.10 for the sugarcane variety CO-002. The decline in shoot length may be due to higher concentrations of cytokinin and auxin beyond the optimal for the CO-002 sugarcane variety, which may suppress the elongation of the explant. Hailu et al. (2018) also reported that shoot length decreased with increasing BAP concentration exceeding the optimum (2.0 mg/l BAP).

The significantly lower shoot length observed in the control (without PGR supplementation) indicates that plant growth regulators are essential for promoting shoot elongation under in vitro conditions. However, although PGR supplementation enhanced shoot growth, the results also suggest that increasing hormone concentrations beyond the optimal level may reduce shoot elongation, indicating a concentration-dependent effect. This observation is consistent with previous findings of Hailu et al. (2018), which report that supra-optimal hormone levels can adversely affect in vitro shoot morphology. The findings showed that optimal concentrations of cytokinin and auxin produced the best outcome for shoot length in sugarcane variety CO-002, with TII giving the best result.

Conclusion

The findings demonstrate that plant growth regulators significantly affected the in vitro establishment, shoot emergence, shoot number, and shoot length of sugarcane variety CO-002. The treatment TII with MS media supplementation of 2.0 mg/l BAP, 0.50 mg/l kinetin, and 0.50 mg/l IAA gave the optimal results for shoot emergence, shoot count, and shoot length. However, higher PGR concentrations resulted in a decrease in the number of days to shoot emergence, the number of shoot formation and shoot length. Thus, the use of optimum PGR levels in MS media is more effective for micropropagation of the CO-002 sugarcane variety.

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Convergence Analysis of Some Iterative Schemes in Banach Space

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Abstract

A fixed point is a point that remains unchanged under a given mapping and plays a fundamental role in mathematical analysis and its applications. Fixed-point iterative methods are widely used to obtain approximate solutions to complex mathematical problems that may not admit explicit solutions. This paper examines three iterative schemes within the framework of Banach spaces. It focuses on the rate of convergence and uniqueness of fixed points. The study provides analysis on the conditions under which these iterative processes converge to a unique solution. Numerical examples and tabulated results are presented to illustrate and validate the theoretical findings. Furthermore, new results concerning the existence and convergence of Mann iteration, S-iteration, and Noor iterative schemes are established.

Keywords: Fixed point, Iterative schemes, Zamfirescu operator, Banach space, Convergence.

Introduction

A fixed point is a point that remains unchanged under the application of a given mapping. It plays a fundamental role in mathematical analysis and has wide-ranging applications across various fields. Such a point is also referred to as an invariant point. For example, g is a fixed point of the function $f(g)$ if $f(g) = g$. Many problems can be solved by finding fixed point of a particular operator and algorithms for finding such points, Berinde (2004).

An iterative method in computational Mathematics, is a mathematical procedure that generates a sequence of improving approximate solutions for a class of problems. An iterative method is called convergent if the corresponding sequence converges for given initial approximations.

Fixed point method is used to find approximate solutions to algebraic and transcendental equations. Despite the significant progress made in fixed point theory, the study of topological fixed point properties, fixed points in partially ordered structures, and logic and combinatorics remains underdeveloped. More importantly, there is a clear lack of rigorous and systematic results addressing the rate of convergence of iterative schemes, particularly for single-step, double-step, and triple-step methods with errors

using Zamfirescu operator in Banach space. This gap underscores the need for more comprehensive analytical frameworks in this area. Some new results concerning the existence of Mann iteration, S-iteration and Noor iterative schemes would be considered. Dhage (1999) proved some common fixed point theorems for pairs of condensing mappings in an ordered Banach space.

Hussain *et.al.* (2010) extended the results of Dhage to 1-set contractive mappings. Agarwal *et.al.* (2012) proved some new common fixed point theorems for a pair of nonlinear mappings defined on an ordered Banachspace. They also proved the existence of nonnegative integrable solutions for an implicit integral equation and presented some common fixed point results using the weak topology of a Banachspace. Hang and Changfeng (2014) presented a fixed-point iterative method for solving systems of nonlinear equations.

Bucur (2017) stated that fixed point theory is essential to various theoretical and applied fields, such as variational and linear inequalities, approximation theory, nonlinear analysis, integral and differential equations and inclusions, dynamic systems theory, mathematics of fractals, mathematical economics (game theory, equilibrium problems, and optimization problems) and mathematical modelling. In his paper, he presented a few benchmarks regarding the applications of the fixed point theory and also debates if the results of the fixed point theory can be applied to mathematical modelling. He therefore concluded that fixed point theory has many applications in the last decades and its applications are very useful and interesting to optimization theory, to the game theory, to conflict situations, and also to mathematical modelling.

Rauf *et.al.* (2019) proved the convergence and stability of Mann and Ishikawa iterative procedures by applying λ -generalized, Geraghty and Ćirić contractive conditions in a complete G-metric space.

Mannan *et al.* (2021) introduced Banach fixed point theorem for mapping in normed space. They also developed ideas that many of known fixed point theorems can easily be derived from the Banach fixed point theorem and further extended some recent works with Banach contraction principle from metric space to norm space.

Berinde and Pacurar (2021) introduced Krasnoselskij-type algorithm for variational inequality problems and fixed point concept in higher dimensions and applications.

Sahin *et.al.* (2023) obtained some fixed-point results for the KF-iteration process in hyperbolic metric spaces. Sharma *et.al.* (2023) constructed a new three-step fixed point iteration scheme with strong convergence and application. Kamalov and Leung (2023) stated that fixed points represent equilibrium states, stability, and solutions to a range of problems. They said further that it has been an active field of research. In their paper, they provided an overview of the main branches of fixed point theory. They further discussed the key results and applications.

Wahab *et.al.* (2016) proved some theorems to approximate fixed point of Zamfirescu operators on normed spaces for some two-step iterative schemes. Rauf and Oyekanmi (2024) showed weak and strong convergence theorems for a Krasnoselskij-

type iterative method to approximate coupled solution of non-expansive operator $E : F \times F \rightarrow F$, where F is non-empty, depending on two variables. They further obtained a fixed point for an almost-contraction operator (b, θ, L) -almost contraction operator $E : X \times X \rightarrow X$ using the Krasnoselskij iteration. Among the authors used are; Agarwal *et.al.* (2007), Zamfirescu (1972), Berinde (2002) and Imoru and Olatinwo (2003).

The iterations used in this study are; Mann iteration which is single-step, S-iteration known as double-step and Noor iteration refer to as triple-step.

In this study, the strong convergence and convergence rate results of S-iterative, Mann and Noor iterations in Banach space using the Zamfirescu operator will be concerned. The aim of this research work is to establish the strong convergence, convergence rate of iterative schemes with errors in Banach space as well as to find a common iterative scheme for some iterative algorithms using Zamfirescu operator.

PRELIMINARY RESULT

The following Lemma and Theorem are required to prove our main results.

Lemma 1: (Berinde, 2002): Let δ be a real number such that $\delta \in [0, 1]$ and $\{e_n\}_{n=0}^{\infty}$ is a sequence of non-negative numbers such that $\lim_{n \rightarrow \infty} e_n = 0$; then, for any sequence of positive numbers $\{U_n\}_{n=0}^{\infty} \subset X$ satisfying

$$U_{n+1} \leq \delta U_n + e_n \text{ for all } n \in \mathbb{N} \quad (1.1)$$

Moreover, the Zamfirescu operator used the following theorem to prove the strong convergence of Picard iteration.

Theorem 1: (Zamfirescu, 1972): Let (X, d) be a complete metric space and let $T : X \rightarrow X$ be a mapping. Suppose that there exist constants $a, b, c \in [0, 1]$ such that for every $x, y \in X$, at least one of the following conditions holds:

1. $d(Tx, Ty) \leq ad(x, y)$;
2. $d(Tx, Ty) \leq b[d(x, Tx) + d(y, Ty)]$; and
3. $d(Tx, Ty) \leq c[d(x, Ty) + d(y, Tx)]$

Then, T has a unique fixed point $x^* \in X$ (i.e., $Tx^* = x^*$) and for any initial point $x_0 \in X$, the iterative sequence defined by $x_{n+1} = Tx_n$.

The three-step iteration which is called Noor iteration was introduced by Noor (2000) as follows:

$$\begin{aligned} x_{n+1} &= (1 - \alpha_n)x_n + \alpha_n Ty_n \\ y_n &= (1 - \beta_n)x_n + \beta_n Tz_n \\ z_n &= (1 - \gamma_n)x_n + \gamma_n Tx_n, \quad \text{for all } n \geq \mathbb{N} \end{aligned} \quad (1.2)$$

where $\{\alpha_n\}_n^\infty, \{\beta_n\}_n^\infty$ and $\{\gamma_n\}_n^\infty$ are sequences of positive numbers in $[0, 1]$ such that their sum is infinity. Also, observe that if $\gamma_n = 0$ for each n , then the Noor iteration process (1.2) reduces to the Ishikawa iterative scheme.

Another iteration called S-iteration process, was introduced by Agarwal et al (2007) which is independent of Mann and Ishikawa iterations. It is defined in a normed linear space as follows: If $x_0 \in E, E$ is a normed space, the sequence $\{x_n\}$ in E is defined as:

$$\begin{aligned} x_{n+1} &= (1 - \alpha_n)y_n + \alpha_n T y_n \\ y_n &= (1 - \beta_n)x_n + \beta_n T x_n, \quad \text{for all } n \geq 0 \end{aligned} \quad (1.4)$$

where $\alpha_n, \beta_n \subseteq [0, 1]$ with $\sum \alpha_n = \infty$.

Mann (1953) defined a more general iteration in a Banach space setting satisfying quasi-non expansive operators. The Mann iteration is given as:

$$x_n = (1 - \alpha_n)x_{n-1} + \alpha_n T x_{n-1} \quad (1.1)$$

where α_n is a sequence of positive numbers $[0, 1]$. By putting $\alpha_n = 1$ into (1.1) yields Picard iteration.

Liu (1995) introduced the concept of Mann iteration process with errors by the sequence $\{x_n\}$ defined as follows:

$$x_n = (1 - \alpha_n)x_{n-1} + \alpha_n T x_{n-1} + U_n \quad (1.2)$$

where $\{x_n\}$ satisfies $\sum \|U_n\| < \infty$. This certainly contain (1.1). The proof of the convergence of (1.1) and (1.2) in normed linear setting can be found in Rhoades (1995).

MAIN RESULT

In this section, we study convergence and convergence rate of some iterative schemes. We present our main result using Zamfirescu operator on normed spaces for the iterative schemes with errors and at the end, computational results for the rate of convergence of S-iteration, Mann iteration and Noor iteration were tabulated.

The Results of the schemes are given in the following theorems:

Theorem 3.1: Let F be a non-empty subset of a normed linear space $(F, \|\cdot\|)$. Let $T: F \rightarrow F$ be a self-map of F satisfying the Zamfirescu operator for each $x, y \in F$. Let x_n be the Mann iterative scheme, if $F(T) \neq \emptyset$, $\sum_{n=1}^\infty \alpha_n = \infty$ and $\|V_n\| = 0$. Then

- (i) T defined by Theorem 1 has a unique fixed point, p ; and
- (ii) Mann iterative scheme converges strongly to a fixed point of T .

Proof:

- (i) We shall first establish that the mapping T satisfying the condition of Theorem 1 has a unique fixed point.

Suppose there exist $p_1, p_2 \in F_T$, and that $p_1 = p_2$, with $\|p_1 - p_2\| > 0$, then $0 < \|p_1 - p_2\| = \|Tp_1 - Tp_2\| \leq a\|p_1 - p_2\| + 2a(\|p_1 - Tp_1\|)$

This implies,

$$\|Tp_1 - Tp_2\| \leq a\|p_1 - p_2\|$$

Thus,

$$(1 - a)\|p_1 - p_2\| \leq 0$$

Since $a \in [0, 1]$, we have $1 - a > 0$ and $\|p_1 - p_2\| \leq 0$.

Since the norm is non-negative we have $\|p_1 - p_2\| = 0$.

That is $p_1 = p_2 = p$. Thus, T has a unique fixed point p .

(ii) Next, we shall establish that $\lim_{n \rightarrow \infty} x_n = p$. That is, we show that Mann iterative scheme converges strongly to p of T .

In view of Mann iteration and Zamfirescu operator where V_n is an error term, we have

$$\begin{aligned} \|x_{n+1} - p\| &= \|(1 - a_n)x_n + a_nTx_n - p + V_n\| \\ &= \|(1 - a_n)(x_n - p) + a_n(Tx_n - p) + V_n\| \quad (2) \\ &\leq (1 - a_n)\|x_n - p\| + a_n\|Tx_n - p\| + \|V_n\| \end{aligned}$$

Now, using Theorem 1 in (2), we have

$$\begin{aligned} \|x_{n+1} - p\| &\leq (1 - a_n)\|x_n - p\| + a_n\delta\|x_n - p\| + \|V_n\| \\ (3) &= [1 - (1 - \delta)a_n]\|x_n - p\| + \|V_n\|, n = 1, 2, 3, \dots \end{aligned}$$

Using Lemma 1 in (3), the fact that

$$0 \leq \delta < 1, \sum_{n=1}^{\infty} a_n = \infty, \text{ and } \|V_n\| = 0,$$

we find the result that

$$\lim_{n \rightarrow \infty} \|x_{n+1} - p\| = 0$$

Therefore $x_{n+1} \rightarrow p \in F$. That is $\{x_n\}_{n=0}^{\infty}$ converges strongly to p .

Theorem 3.2: Let F be a non-empty subset of a normed linear space, $(F, \|\cdot\|)$. Let $K : F \rightarrow F$ be a self-map of F satisfying the Zamfirescu operator for each $x, y \in F$.

Let $\{x_n\}_{n=0}^{\infty}$ be the S-iterative scheme. If $F(T) \neq \emptyset$, $\sum_{n=1}^{\infty} a_n = \infty$ and $\|V_n\| = 0$. Then

- (i) T defined by Theorem 1 has a unique fixed point p
- (ii) the S-iterative scheme converges strongly to a fixed point of T .

Proof:

(i) We shall first establish that the mapping T satisfying the condition Theorem has a unique fixed point.

Suppose there exist $p_1, p_2 \in F_T$ and that $p_1 \neq p_2$ with $\|p_1 - p_2\| > 0$, then $0 < \|p_1 - p_2\| = \|Tp_1 - Tp_2\| \leq a\|p_1 - p_2\| + 2a(\|p_1 - Tp_1\|)$ this implies

$$\|Tp_1 - Tp_2\| \leq a\|p_1 - p_2\|$$

Thus,

$$(1 - a)\|p_1 - p_2\| \leq 0$$

Since $a \in [0, 1]$ we have $(1 - a)$ and $\|p_1 - p_2\| \leq 0$.

Since the norm is non-negative we have $\|p_1 - p_2\| = 0$. That is, $p_1 = p_2 = p$.

Thus, T has a unique fixed point p .

(ii) Next we shall establish that $\lim_{n \rightarrow \infty} x_n = p$. That is, we show that S-iterative scheme converges strongly to p of T .

From Theorem 1, we have

$$\|T_x - T_y\| \leq \delta\|x - y\| + 2\delta\|x - T_x\| \text{ for all } x, y \in C$$

Also from Theorem 2, we have

$$\|Tx_n - q\| \leq d\|x_n - q\|$$

In view of Theorem 1 and S-iteration, we obtained

$$\begin{aligned} \|x_{n+1} - p\| &\leq \|(1 - a_n)Tx_n - a_nTy_n - p + U_n\| \\ &= \|(1 - a_n)(Tx_n - p) + a_n(Ty_n - p) + U_n\| \\ &\leq (1 - a_n)\|(Tx_n - p)\| + a_n\|Ty_n - p\| + \|U_n\| \\ &\leq (1 - a_n)\delta\|x_n - p\| + a_n\delta\|y_n - p\| + \|U_n\| \end{aligned} \quad (4)$$

also, from S-iteration, we have

$$\begin{aligned} \|y_n - p\| &\leq \|(1 - b_n)x_n - b_nTx_n - p + V_n\| \\ &= \|(1 - b_n)(x_n - p) + b_n(Tx_n - p) + V_n\| \\ &\leq (1 - b_n)\|(x_n - p)\| + b_n\delta\|x_n - p\| + \|V_n\| \\ &= [(1 - b_n) + b_n\delta]\|x_n - p\| + \|V_n\| \end{aligned} \quad (5)$$

Substituting (5) in (4), we have

$$\|x_{n+1} - p\| \leq [(1 - a_n)\delta + a_n\delta((1 - b_n) + b_n\delta)]\|x_n - p\| + \|U_n\| + \|V_n\| \quad (6)$$

Inequality (6) becomes

$$\|x_{n+1} - p\| \leq [(1 - (1 - \delta)a_n)]\|x_n - p\| + \|U_n\| + \|V_n\| \quad n = 1, 2, \dots \quad (7)$$

By putting $\|e_n\| = \|U_n\| + \|V_n\|$ and using Lemma 1 in (7), we find the result that

$$\lim_{n \rightarrow \infty} \|x_{n+1} - p\| = 0$$

Hence, $x_{n+1} \rightarrow p \in F_T$.

That is $\{x_n\}_{n=0}^{\infty}$ converges strongly to p . This ends the proof

Theorem 3.3: Let F be a non-empty subset of normed linear space $(F, \|\cdot\|)$. Let $K : F \rightarrow F$ be a self-map of F satisfying the Zamfirescu operator for each $x, y \in F$. If $F(T) \neq \Phi$, $\sum_{n=1}^{\infty} a_n = \infty$, $\|V_n\| = 0$. Then

- (i) T defined by Theorem 1 has a unique fixed point p ; and
- (ii) the Noor iterative scheme converges strongly to a fixed point of T .

Proof:

(i) We shall first establish that the mapping T satisfying the condition Theorem 1 has a unique fixed point. Suppose there exist $p_1, p_2 \in F_T$, and that $p_1 \neq p_2$, with $\|p_1 - p_2\| > 0$, then

$$\begin{aligned} 0 < \|p_1 - p_2\| &= \|Tp_1 - Tp_2\| \\ &\leq 2a\|p_1 - Tp_1\| + a\|p_1 - p_2\| \end{aligned}$$

this implies

$$\|p_1 - p_2\| \leq a\|p_1 - p_2\|$$

Thus

$$(1 - a)\|p_1 - p_2\| \leq 0$$

Since $a \in [0, 1]$, we have $(1 - a) > 0$ and $\|p_1 - p_2\| \leq 0$. Since the norm is non-negative we have

$$\|p_1 - p_2\| = 0. \text{ That is } p_1 = p_2 = p \text{ (say).}$$

Thus, T has a unique fixed point p .

(ii) Next we shall establish that $\lim_{n \rightarrow \infty} x_n = p$. That is, we show that Noor iterative scheme converges strongly to p of T .

From Theorem 1, we have

$$\|Tx - Ty\| \leq \delta\|x - y\| + 2\delta\|x - Tx\| \text{ for all } x, y \in C$$

Also from Theorem 1, we have

$$\|Tx_n - q\| \leq \delta\|x_n - q\|$$

By applying Theorem 1 to Noor iteration with error term U_n yield

$$\begin{aligned} \|x_{n+1} - p\| &\leq \|(1 - a_n)x_n + a_nTy_n + U_n - p\| \\ &= \|(1 - a_n)x_n + a_nTy_n - a_np + a_np + U_n - p\| \\ &= \|(1 - a_n)(x_n - p) + a_n(Ty_n - p) + U_n\| \end{aligned} \tag{9}$$

$$\begin{aligned} &\leq (1 - a_n)\|x_n - p\| + a_n\|Ty_n - p\| + \|U_n\| \\ &\leq (1 - a_n)\|x_n - p\| + a_n\delta\|y_n - p\| + \|U_n\| \end{aligned}$$

Also, from Noor iteration, we have

$$\begin{aligned} \|y_n - p\| &\leq \|(1 - b_n)x_n - b_nTz_n + V_n - p\| \\ &= \|(1 - b_n)x_n + b_nTz_n - b_np + b_np + V_n - p\| \\ &= \|(1 - b_n)(x_n - p) + b_n(Tz_n - p) + V_n\| \tag{10} \\ &\leq (1 - b_n)\|x_n - p\| + b_n\|Tz_n - p\| + \|V_n\| \\ &\leq (1 - b_n)\|x_n - p\| + b_n\delta\|z_n - p\| + \|V_n\| \end{aligned}$$

Then, from Noor iteration with W_n as error term implies

$$\begin{aligned} \|z_n - p\| &\leq \|(1 - c_n)x_n - p + c_nTx_n + V_n - W_n\| \\ &= \|(1 - c_n)x_n - p + c_nTz_n - c_np + c_np + W_n\| \\ &= \|(1 - c_n)(x_n - p) + c_n(Tx_n - p) + W_n\| \tag{11} \\ &= (1 - c_n)\|x_n - p\| + c_n\delta\|Ty_n - p\| + \|W_n\| \\ &\leq [(1 - c_n) + c_n\delta]\|x_n - p\| + \|W_n\| \end{aligned}$$

Substituting (11) in (10), we have

$$\begin{aligned} \|y_n - p\| &\leq \|(1 - b_n)\|x_n - p\| + b_n\delta((1 - c_n + c_n\delta)\|x_n - p\| + \|W_n\| + \|V_n\|) \\ &\leq [(1 - b_n) + b_n\delta(1 - c_n + c_n\delta)]\|x_n - p\| + \|W_n\| + \|V_n\| \end{aligned} \tag{12}$$

Similarly substituting (12) in (9), we obtain

$$\begin{aligned} \|x_{n+1} - p\| &\leq (1 - a_n)\|x_n - p\| + a_n\delta((1 - b_n + b_n\delta(1 - c_n + c_n\delta))\|x_n - p\| + \|W_n\| + \|V_n\| + \|U_n\|) \\ &\leq (1 - a_n) + a_n\delta(1 - b_n + b_n\delta - b_nc_n\delta + b_nc_n\delta^2)]\|x_n - p\| + \|W_n\| + \|V_n\| + \|U_n\| \tag{13} \end{aligned}$$

By letting $\|\epsilon_n\| = \|W_n\| + \|V_n\| + \|U_n\|$ and by Lemma 1, using the condition of the theorem 1, we have

$$\lim_{n \rightarrow \infty} \|x_{n+1} - p\| = 0$$

Hence, $x_{n+1} \rightarrow p \in F(T)$ implying $\{x_n\}_{n=0}^\infty$ converges strongly to p . The proof is complete

Corollary 3.4: Let F be a non-empty subset of a normed linear space, $(F, \|\cdot\|)$. Let $K : F \rightarrow F$ be a self-map of F satisfying the Zamfirescu operator for each $x, y \in F$. If

$F(T) \neq \Phi$, $\sum_{n=1}^{\infty} a_n = \infty$, $\|V_n = 0\|$. Then

- (i) T defined by Theorem 1 has a unique fixed point;
- (ii) the Mann iterative scheme converges strongly to p of T ;
- (iii) the S-iterative scheme converges strongly to p of T ;
- (iv) the Noor iterative scheme converges strongly to p of T .

Proof:

The proof of this corollary is direct from Theorem 3.1 and theorem 3.2 when $b_n = 1$ in Mann iteration and S-iteration.

Moreso, we use example to compare our iterative schemes with the help of MATLAB.

Numerical Example

The function defined by $f(x) = (1 - x)^2$ is a function with multiple zero. By taking the initial approximation $x_0 = 0.9$, $a_n = b_n = \frac{1}{2}$, the comparison of the convergence of the iterative schemes to the exact fixed point $p = 0.38196601$ is shown in the Table 1 below.

TABLE 1: The results for various iterations of the example

S/N	Mann Iteration	Noor Iteration	SIteration
0	0.90000000	0.90000000	0.90000000
1	0.45500000	0.53059611	0.15351250
2	0.37601250	0.41092632	0.51786791
3	0.38268645	0.38648213	0.31143842
4	0.38188123	0.38263205	0.42141796
5	0.38197602	0.38396925	0.36076612
6	0.38196483	0.38225969	0.39361130
7	0.38196615	0.38200889	0.37564509
8	0.38196599	0.38197227	0.38541937
9	0.38196601	0.38196692	0.38008599
10	-	0.38196614	0.38299148
11	-	0.38196603	0.38140725
12	-	0.38196601	0.38227064
13	-	-	0.38179998
14	-	-	0.38205652
15	-	-	0.38191668
16	-	-	0.38199290

17	-	-	0.38195135
18	-	-	0.38197400
19	-	-	0.38196166
20	-	-	0.38196839
21	-	-	0.38196472
22	-	-	0.38196672
23	-	-	0.38196563
24	-	-	0.38196622
25	-	-	0.38196590
26	-	-	0.38196607
27	-	-	0.38196598
28	-	-	0.38196603
29	-	-	0.38196600
30	-	-	0.38196601

The error estimation (ϵ_n) in the Table 1 choosing n at an interval of 5 iterations is presented in Table 2, where $\epsilon_n = \|q - x_n\|$.

TABLE 2: The results error estimation for various iterations of the example

S/N	Mann Iteration	S Iteration	Noor Iteration
0	0.00001001	0.00211999	0.00200323
1	0.00000000	0.00102547	0.00000013
2	-	0.00002689	0.00000000
3	-	0.00000238	-
4	-	0.00000011	-
5	-	0.00000000	-

CONCLUSION

In conclusion, we have proved the convergence and convergence rate of some iterative schemes with errors using Zamfirescu operator in Banach space. It was observed that Mann iterative converges faster than both Noor iteration and S-iteration and Noor iteration is faster than S-iteration in term of convergence rate.

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***Amaranthus spinosus* (Green Variant) Restores Bone Marrow Architecture and Preserves Hepatic Integrity in Experimentally Induced Anaemic Rats**

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Abstract

Anaemia remains one of the most prevalent nutritional and public health disorders globally, particularly in developing countries. Although iron supplementation is widely used for treatment, prolonged use may cause adverse effects including gastrointestinal irritation and hepatic stress. Consequently, attention has increasingly focused on plant-based functional foods rich in micronutrients and bioactive compounds that can support haematopoiesis with minimal toxicity. *Amaranthus spinosus*, a widely consumed leafy vegetable in tropical regions, is known for its high nutritional value and diverse phytochemical constituents. This study evaluated the haematopoietic and hepatoprotective effects of *Amaranthus spinosus* (green variant) in experimentally induced anaemic rats through histological, phytochemical, and nutritional analyses. Anaemia was experimentally induced in rats, after which animals were fed diets containing 50% and 100% *A. spinosus*. Control groups included non-anaemic rats, untreated anaemic rats, and ferrous sulfate-treated rats. Bone marrow and liver tissues were examined histologically at 40 h and 14 days post-treatment. Phytochemical composition was determined using quantitative phytochemical assays, GC–MS, and FTIR analyses. Proximate composition, mineral content, and vitamin levels were also evaluated. Anaemia induction resulted in marked bone marrow hypercellularity characterized by erythroid proliferation, while hepatic architecture remained largely preserved. Ferrous sulfate treatment promoted erythropoietic recovery but was associated with mild hepatic inflammatory changes. In contrast, *A. spinosus*-fed groups exhibited restoration of normocellular bone marrow architecture and preserved hepatic histology without inflammatory lesions. Phytochemical analysis revealed high levels of phenolics (329.09 ± 0.46 mg/100 g), tannins (663.50 ± 0.94 mg/100 g), and alkaloids ($6.95 \pm 0.05\%$). GC–MS identified 22 bioactive compounds dominated by fatty acids and hydrocarbons. Nutritional analysis showed high protein (31.95%), iron (49.37 mg/100 g), and vitamins A and C contents. *Amaranthus spinosus* demonstrated significant haematopoietic-supportive and hepatoprotective properties in anaemic rats, likely due to its rich phytochemical and micronutrient composition. These findings support the potential of *A. spinosus* as a functional food for the dietary management of anaemia.

Keywords:

Anaemia; *Amaranthus spinosus*; haematopoiesis; phytochemicals; liver histology; functional food.

INTRODUCTION

Anaemia remains one of the most widespread global public health challenges, affecting an estimated two billion people worldwide and disproportionately impacting

populations in low- and middle-income countries (World Health Organization, 2021). The condition is characterized by a reduction in haemoglobin concentration, red blood cell count, or packed cell volume, leading to diminished oxygen-carrying capacity of the blood and impaired delivery of oxygen to body tissues. Consequently, anaemia is associated with fatigue, reduced physical performance, impaired cognitive development, and increased morbidity and mortality, particularly among children and pregnant women (World Health Organization, 2021; Centers for Disease Control and Prevention, 2022). Among the various etiological factors, nutritional deficiencies—particularly iron deficiency—represent the most common cause of anaemia worldwide. Iron is an essential micronutrient required for haemoglobin synthesis, erythropoiesis, and numerous enzymatic processes involved in cellular metabolism (Ross *et al.*, 2020). In addition to nutritional deficiencies, other contributing factors include parasitic infections, chronic inflammatory diseases, genetic haemoglobin disorders, and inadequate dietary intake of essential vitamins such as folate, vitamin B12, and vitamin A (Food and Agriculture Organization, 2019).

The conventional management of anaemia primarily involves iron supplementation, dietary modification, and in severe cases, blood transfusion. Ferrous sulfate and other iron salts are widely prescribed due to their effectiveness in restoring haemoglobin levels and replenishing body iron stores. However, prolonged iron supplementation has been associated with several adverse effects, including gastrointestinal irritation, nausea, constipation, and oxidative stress resulting from excessive iron accumulation (Black *et al.*, 2013). Excess iron may also contribute to hepatic oxidative damage through the generation of reactive oxygen species, which can compromise liver function and cellular integrity (Bonkovsky, 2012). These limitations have stimulated increasing interest in exploring alternative therapeutic strategies based on plant-derived nutraceuticals and functional foods capable of supporting erythropoiesis while minimizing adverse effects.

Leafy vegetables are widely recognized as important dietary sources of essential nutrients, including proteins, minerals, vitamins, and diverse phytochemicals that contribute to overall health and hematological function. In many developing regions, traditional vegetables serve as affordable and accessible dietary resources that help alleviate micronutrient deficiencies (Food and Agriculture Organization, 2019). Among these, species belonging to the genus *Amaranthus* are extensively cultivated and consumed in tropical and subtropical regions. These plants are valued for their high nutritional quality, rapid growth, and adaptability to diverse environmental conditions. One important species within this genus is *Amaranthus spinosus*, commonly known as spiny amaranth. The plant is widely used in traditional medicine and local diets across Africa, Asia, and Latin America. Ethnomedicinal reports attribute several therapeutic properties to *A. spinosus*, including anti-inflammatory, antioxidant, antimicrobial, and hematinic activities (Rahmatullah *et al.*, 2010). Previous phytochemical investigations have revealed that *Amaranthus* species contain substantial amounts of iron, calcium, magnesium, and potassium, as well as vitamins A and C and bioactive compounds such as phenolics, flavonoids, alkaloids, tannins, and saponins (Shahidi, 2015). These phytochemicals possess antioxidant and anti-inflammatory properties that may contribute to improved erythropoiesis and protection against oxidative stress associated with anaemia.

Despite the nutritional richness and ethnomedicinal importance of *A. spinosus*, relatively few studies have comprehensively evaluated its potential role in anaemia management using integrated experimental approaches. In particular, limited research has examined the combined histological effects of *Amaranthus*-based dietary supplementation on both bone marrow structure and hepatic tissue during anaemia

recovery. This is significant because the bone marrow serves as the primary site of erythropoiesis, while the liver plays a crucial role in iron metabolism, storage, and detoxification processes essential for maintaining systemic homeostasis (Greer *et al.*, 2019).

Therefore, the present study investigated the haematopoietic and hepatoprotective effects of the green variant of *Amaranthus spinosus* in experimentally induced anaemic rats. Specifically, the study aimed to: (i) evaluate histological changes in bone marrow and liver tissues following dietary supplementation with *A. spinosus*; (ii) determine the phytochemical composition of the plant using chromatographic and spectroscopic techniques; and (iii) assess its nutritional value through proximate, mineral, and vitamin analyses. By integrating histological, phytochemical, and nutritional assessments, this study seeks to provide comprehensive insights into the potential of *A. spinosus* as a functional dietary resource for the nutritional management of anaemia.

MATERIALS AND METHODS

Experimental Animals and Ethical Approval

All experimental procedures involving animals were conducted in accordance with the institutional guidelines for laboratory animal care and were approved by the University of Ilorin Ethical Review Committee.

A total of forty-two (42) healthy adult Wistar rats (*Rattus norvegicus*), weighing 150–200 g, were obtained from the Animal Holding Unit of the Department of Physiology, University of Ilorin, Nigeria. The animals were acclimatized for two weeks under standard laboratory conditions (temperature 25 ± 2 °C, relative humidity $55 \pm 5\%$, and a 12 h light/dark cycle) with free access to feed and water.

Rats were housed in polypropylene cages lined with sterile wood shavings and maintained under hygienic conditions throughout the experimental period.

Experimental Design

The experiment followed a completely randomized design consisting of five treatment groups, each comprising six rats (three males and three females). The groups included two dietary treatments involving *Amaranthus spinosus* (green variant) and three control groups.

After acclimatization and anaemia induction, the animals were assigned to the following groups:

1. Non-anaemic control: rats maintained on standard commercial feed.
2. Anaemic control (untreated): anaemic rats receiving standard feed without treatment.
3. Anaemic + ferrous sulfate: anaemic rats treated with ferrous sulfate.
4. Anaemic + 50%*Amaranthus spinosus*diet: anaemic rats fed a diet containing 50% *A. spinosus* leaf powder.
5. Anaemic + 100%*Amaranthus spinosus*diet: anaemic rats fed a diet containing 100% *A. spinosus* leaf powder.

Dietary treatment continued for 14 days following confirmation of anaemia. Tissue samples were collected at two experimental time points: 40 h post-induction and 14 days post-treatment, corresponding to the histological analyses presented in the Results section.

Plant Material Collection and Authentication

Fresh leaves of *Amaranthus spinosus* (green variant) were collected from the Botanical Garden of the University of Ilorin and nearby cultivated fields. The plant

material was authenticated by a plant taxonomist in the Department of Plant Biology, University of Ilorin. A voucher specimen (UILH/001/1851/2026) was deposited in the departmental herbarium for future reference.

Preparation of Plant Material and Diet Formulation

The harvested leaves were thoroughly washed with distilled water to remove debris and then air-dried at room temperature (25–28 °C) for 7–10 days. The dried leaves were pulverized into fine powder using a laboratory mill and stored in airtight containers until use.

Experimental diets were prepared by incorporating *A. spinosus* leaf powder into standard rat chow at inclusion levels of 50% and 100%, replacing equivalent proportions of the basal diet. The control groups received standard commercial rat feed. Feed and water were supplied ad libitum throughout the experimental period.

Prior to feeding, proximate and mineral compositions of the formulated diets were analyzed to ensure nutrient adequacy.

Induction of Anaemia

Anaemia was experimentally induced using cyclophosphamide (CP) (Sigma-Aldrich, USA) administered intraperitoneally at a dose of 40 mg/kg body weight for two consecutive days. This protocol was adapted from previously established procedures for inducing haemolytic anaemia in rodents.

Forty-eight hours after the final injection, blood samples were collected to confirm anaemia through assessment of haemoglobin concentration and packed cell volume.

Ferrous Sulfate Treatment

Rats assigned to the positive control group received ferrous sulfate as a standard anti-anaemic treatment. Ferrous sulfate was administered orally at a therapeutic dose consistent with established experimental protocols for iron supplementation in rodents.

Tissue Collection

At each experimental time point (40 h post-induction and 14 days post-treatment), rats were euthanized under light chloroform anaesthesia followed by cervical dislocation. Bone marrow samples were obtained from the femoral shafts, while liver tissues were carefully excised and rinsed in normal saline to remove blood residues. The tissues were immediately fixed in 10% neutral buffered formalin for histological processing.

Histological Processing and Examination

Fixed tissues were processed following standard histological procedures. Samples were dehydrated through graded ethanol series, cleared in xylene, and embedded in paraffin wax. Paraffin blocks were sectioned at 5 µm thickness using a rotary microtome (Leica RM2235, Germany).

The sections were mounted on glass slides and stained with hematoxylin and eosin (H&E) to evaluate general tissue architecture.

Stained sections were examined under a light microscope (Leica DM500, Germany), and representative photomicrographs were captured using a digital imaging system. Histological assessment focused on bone marrow cellularity, erythroid and myeloid lineage distribution, megakaryocyte presence, sinusoidal structure, and hepatic architecture, including hepatocyte morphology, portal triads, congestion, and inflammatory changes.

Representative micrographs were documented as Figures 1–8.

Phytochemical and Nutritional Analysis

Quantitative Phytochemical Analysis

Quantitative phytochemical constituents including phenolics, flavonoids, tannins, saponins, alkaloids, terpenoids, and glycosides were determined using standard spectrophotometric and gravimetric methods.

Proximate Composition

Proximate components, including moisture content, ash, crude protein, crude fat, crude fibre, and carbohydrate, were determined according to standard procedures described by the **Association of Official Analytical Chemists (AOAC)**.

Mineral Analysis

Mineral elements such as iron (Fe), calcium (Ca), magnesium (Mg), potassium (K), copper (Cu), manganese (Mn), and zinc (Zn) were quantified using an Atomic Absorption Spectrophotometer (Buck Scientific Model 210 VGP, USA).

Vitamin Determination

Vitamins A, C, and E, as well as selected B-complex vitamins, were quantified using high-performance liquid chromatograph (HPLC) with appropriate standards.

GC–MS Analysis

Bioactive compounds present in *A. spinosus* leaf and seed extracts were further characterized using gas chromatography-mass spectrometry (GC-MS) (Shimadzu QP2010 Plus, Japan). Compound identification was based on comparison of mass spectra with reference spectra from the NIST library.

FTIR Analysis

Fourier transform infrared (FTIR) spectroscopy was used to identify major functional groups present in the plant samples. Spectral scans were recorded across the range of 400–4000 cm⁻¹.

Statistical Analysis

All quantitative data were expressed as mean $\pm$ standard deviation (SD). Statistical analyses were performed using SPSS version 26.0 (IBM Corporation, USA). Differences among treatment groups were evaluated using one-way analysis of variance (ANOVA) followed by Tukey's post-hoc test for multiple comparisons.

Statistical significance was considered at $p < 0.05$. Results were presented in tables and figures corresponding to the histological and biochemical observations described in the Results section.

RESULTS

Histological Evaluation of Bone Marrow and Liver Tissues

Forty Hours Post-Induction (Control Groups)

Histological examination conducted 40h after anaemia induction revealed distinct differences in bone marrow cellularity among the control groups, while hepatic architecture remained largely preserved (Figs. 1–3).

In non-anaemic female rats maintained on a normal diet, bone marrow sections showed normal bony trabeculae with wide inter-trabecular spaces containing sparse myeloid elements and occasional megakaryocytes (Fig. 1a). Areas of inter-trabecular haemorrhage were present. Liver sections from the same animals (Fig. 1b) exhibited preserved lobular organization with hepatocyte cords radiating from the central vein.

Hepatocytes displayed granular cytoplasm and intact nuclei, while portal triads appeared normal without inflammatory infiltration.

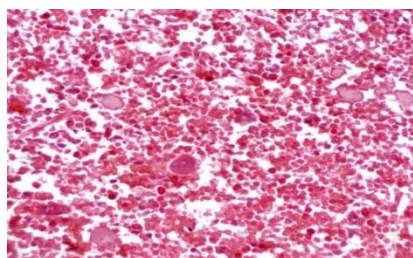
Untreated anaemic female rats demonstrated markedly increased bone marrow cellularity (Fig. 2a). The marrow consisted of densely packed erythroid, myeloid, and lymphoid precursors with numerous sinusoidal channels and scattered megakaryocytes. Comparable features were observed in untreated anaemic male rats (Fig. 2c), where proliferating erythroid precursors and prominent megakaryocytes dominated the marrow architecture.

Despite the marked marrow hypercellularity observed in the anaemic groups, liver sections from both male and female rats (Figs. 2b–2d) retained normal lobular configuration with intact hepatocytes and preserved portal triads. Mild cytoplasmic vacuolation was occasionally observed but without inflammatory or degenerative changes.

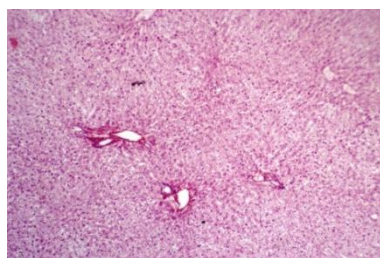
In ferrous sulfate-treated rats (Figs. 3a–3b), bone marrow remained densely cellular and contained clearly defined maturation stages of erythroid and myeloid cells, numerous megakaryocytes, and patent sinusoids. Liver sections displayed intact hepatic cords and normal hepatocyte morphology without necrosis or structural disruption.

Table 1: Histological observations at 40 hours post-induction (Control Groups)

Group	Organ	Major Histological Features	Interpretation
Non-anaemic female (control)	Bone marrow	Sparse myeloid cells, few megakaryocytes, extensive haemorrhage	Normal marrow quiescence
Non-anaemic female (control)	Liver	Normal hepatocytes, preserved lobular pattern, no inflammation	Normal hepatic structure
Anaemic female (untreated)	Bone marrow	Hypercellular marrow, maturing erythroid/myeloid series, active sinusoids	Compensatory erythropoiesis
Anaemic female (untreated)	Liver	Normal hepatocytes, vacuolated cytoplasm, intact portal triads	No hepatic damage
Anaemic male (untreated)	Bone marrow	Dense marrow, abundant megakaryocytes, distinct lineages	Erythroid hyperplasia
Anaemic male (untreated)	Liver	Normal hepatocytes, minimal vacuolation	Preserved hepatic function
Anaemic female (FeSO ₄ -treated)	Bone marrow	Compact cellular marrow, distinct lineages, numerous sinusoids	Regenerative response
Anaemic female (FeSO ₄ -treated)	Liver	Normal hepatocytes, eosinophilic cytoplasm	Normal hepatic function



a



b

Fig. 1: Histological sections of bone marrow and liver from non-anaemic control rats at 40 h post-induction.

- (a) Bone marrow showing normal trabecular bone with wide inter-trabecular spaces containing sparse myeloid cells and scattered megakaryocytes.
- (b) Liver section displaying preserved hepatic lobular architecture with hepatocyte cords radiating from the central vein and intact portal triads. (H&E stain; ×400).

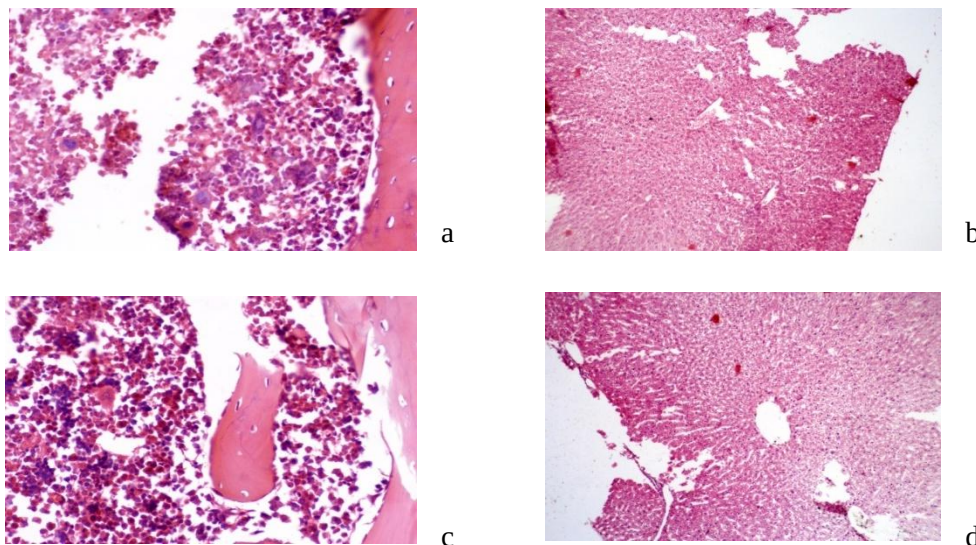


Fig. 2: Histological sections of bone marrow and liver from untreated anaemic rats at 40 h post-induction.

- (a) Bone marrow of anaemic female rat showing marked hypercellularity with numerous erythroid and myeloid precursors.
- (b) Liver section of anaemic female rat showing preserved hepatic architecture.
- (c) Bone marrow of anaemic male rat demonstrating dense erythroid proliferation and prominent megakaryocytes.
- (d) Liver section of anaemic male rat with intact hepatocyte cords and portal structures. (H&E stain; ×400).

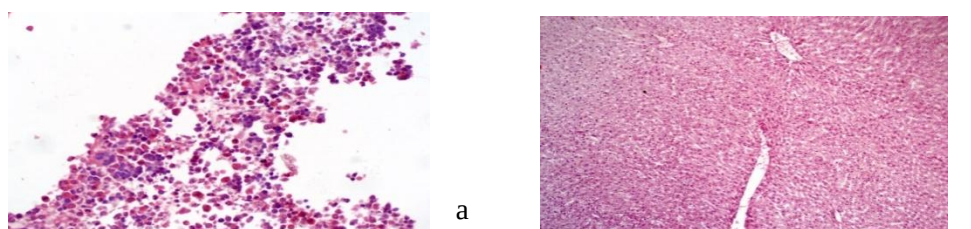


Fig. 3: Histological sections of bone marrow and liver from ferrous sulfate-treated anaemic rats at 40 h post-treatment.

- (a) Bone marrow showing dense cellularity with distinct erythroid and myeloid maturation stages and numerous megakaryocytes.
- (b) Liver section displaying preserved hepatocytes and intact lobular organization without structural damage. (H&E stain; ×400).

Fourteen Days Post-Treatment (Control Groups)

Histological observations at 14 days showed that most control groups maintained their characteristic tissue architecture with minor variations (Figs. 4–6).

In non-anaemic male rats, liver sections exhibited normal hepatic lobules with intact hepatocytes and portal triads (Fig. 4a). Bone marrow sections were normocellular with identifiable erythroid and myeloid precursors and scattered megakaryocytes (Fig. 4b).

Untreated anaemic female rats continued to display hypercellular marrow with abundant erythroid precursors and megakaryocytes (Fig. 5a). Liver sections from these

animals maintained normal hepatic architecture with preserved hepatocytes and no inflammatory lesions (Fig. 5b).

In ferrous sulfate-treated groups, liver sections demonstrated mild histological alterations characterized by portal inflammatory infiltration and occasional central venous congestion, particularly in anaemic males (Figs. 6a–6b). Bone marrow sections appeared moderately cellular with loosely arranged haematopoietic elements, open sinusoids, and distinct maturation stages of erythroid and myeloid lineages (Figs. 6c–6d).

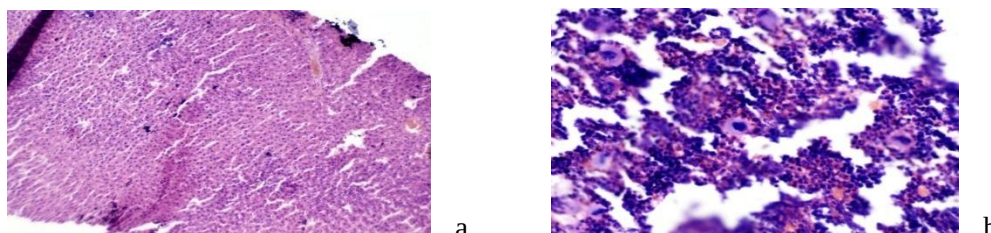


Fig. 4: Histological sections of tissues from non-anaemic rats at 14 days.

- (a) Liver showing normal lobular architecture with intact hepatocytes and portal triads.
- (b) Bone marrow displaying normocellular haematopoietic tissue with identifiable erythroid and myeloid lineages. (H&E stain; ×400).

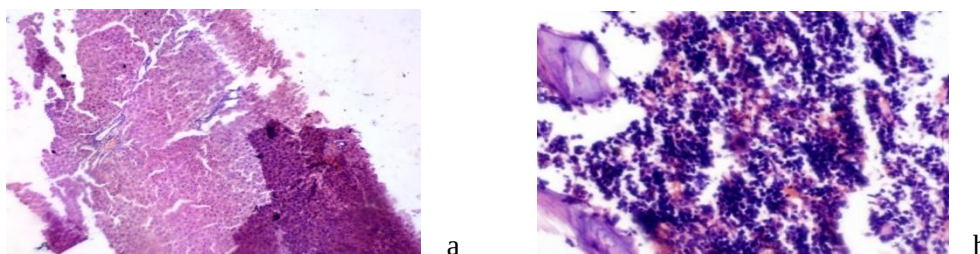


Fig. 5: Histological sections of tissues from untreated anaemic rats at 14 days post-induction.

- (a) Bone marrow showing persistent hypercellularity with erythroid predominance.
- (b) Liver section maintaining normal architecture without inflammatory changes. (H&E stain; ×400).

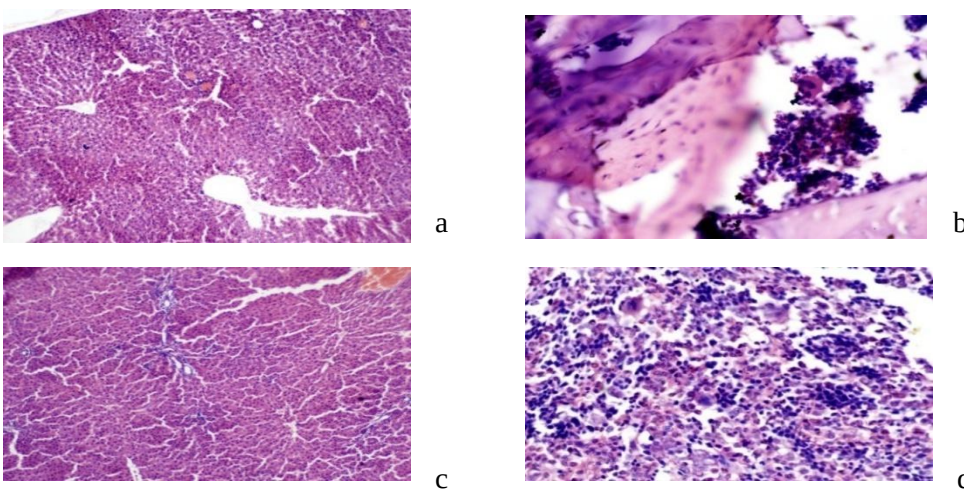


Fig. 6: Histological sections of tissues from ferrous sulfate-treated rats at 14 days.

- (a–b) Liver sections showing mild portal inflammation and occasional central venous congestion.
- (c–d) Bone marrow sections demonstrating moderately cellular marrow with identifiable maturation stages of haematopoietic cells. (H&E stain; $\times 400$).

Table 2: Histological observations at 14 days post-treatment (Control Groups)

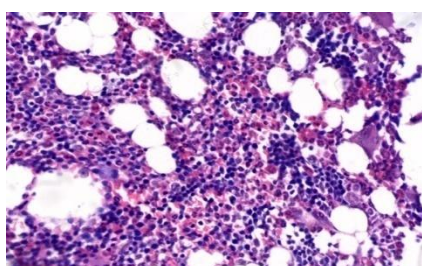
Group	Organ	Major Histological Features	Interpretation
Non-anaemic male	Liver	Normal hepatocytes, intact portal triads, no damage	Normal liver
Non-anaemic male	Bone marrow	Normocellular marrow, distinct myeloid/erythroid lineages	Normal haematopoiesis
Anaemic female (untreated)	Liver	Normal architecture, granular cytoplasm	No hepatic damage
Anaemic female (untreated)	Bone marrow	Dense marrow, erythroid hyperplasia	Compensatory erythropoiesis
Anaemic female (FeSO ₄ -treated)	Liver	Mild–moderate portal inflammation	Hepatic irritation due to FeSO ₄
Anaemic female (FeSO ₄ -treated)	Bone marrow	Loose cellular marrow, numerous sinusoids	Active regeneration
Anaemic male (FeSO ₄ -treated)	Liver	Central venous congestion, portal inflammation	Mild hepatocellular stress
Anaemic male (FeSO ₄ -treated)	Bone marrow	Loose cellular marrow, distinct maturing lineages	Erythropoietic recovery

Forty Hours Post-/Induction (*Amaranthus spinosus*-Fed Groups)

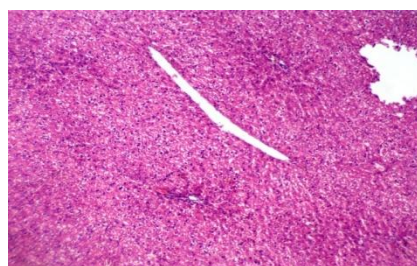
Anaemic rats fed diets containing the green variant of *Amaranthus spinosus* (50% and 100%) exhibited bone marrow and hepatic architectures comparable to normal histological patterns within 40 h (Figs. 7a–7f).

Bone marrow sections from both male and female rats showed normocellular intramedullary tissue composed of adipocytes, granulocytes, megakaryocytes, and vascular sinusoids. Cellular distribution and marrow organization appeared comparable across both dietary levels.

Liver sections from these animals displayed preserved hepatic lobular architecture with hepatocyte cords radiating from the central vein and intact portal tracts. Hepatocytes exhibited eosinophilic cytoplasm and distinct nuclei without evidence of inflammatory infiltration, necrosis, or structural distortion.



a



b

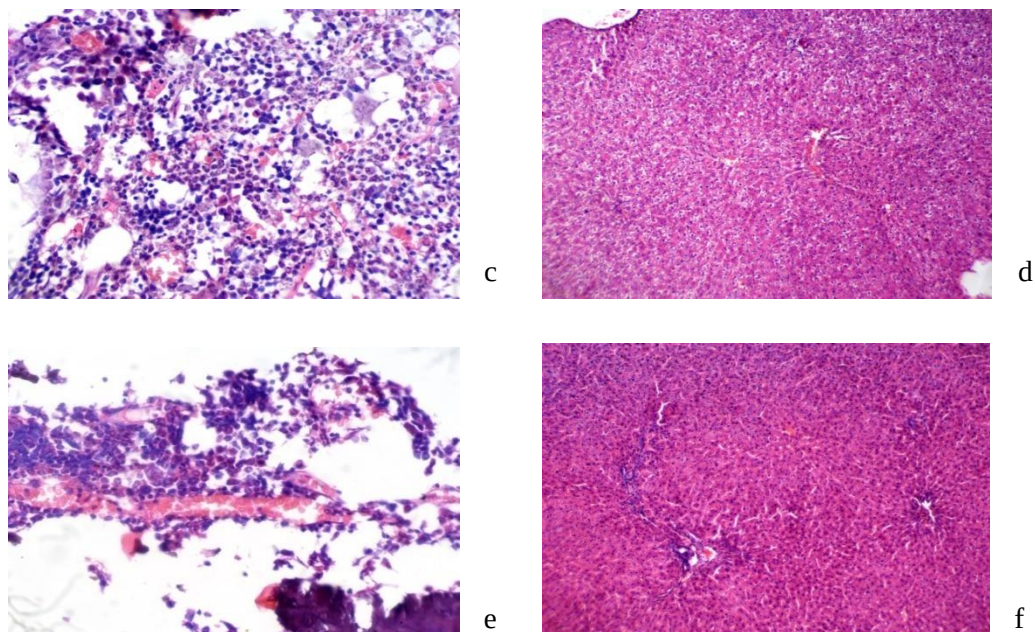


Fig. 7: Histological sections of bone marrow and liver from anaemic rats fed *Amaranthus spinosus* (green variant) diets at 40 h post-treatment.

- (a–c) Bone marrow sections showing normocellular intramedullary tissue with adipocytes, granulocytes, and megakaryocytes.
- (d–f) Liver sections displaying preserved hepatocyte cords and intact portal tracts without inflammatory lesions. (H&E stain; $\times 400$).

Fourteen Days Post-Treatment (*Amaranthus spinosus*-Fed Groups)

At 14 days post-treatment, histological examination revealed sustained normalization of tissue architecture in rats receiving *A. spinosus* diets (Figs. 8a–8h).

In the 50% dietary group, bone marrow sections from both sexes displayed normal bone fragments with normocellular marrow composed of granulocytes, lymphoid cells, and scattered megakaryocytes. Liver sections retained intact hepatocyte cords and portal structures without inflammatory or degenerative changes.

In the 100% dietary group, bone marrow of anaemic males exhibited mild hypocellularity with reduced megakaryocyte numbers and occasional haemorrhagic foci. In contrast, female rats maintained normocellular marrow with numerous sinusoidal spaces. Liver tissues across all animals in this group preserved normal lobular organization with no observable pathological alterations.

Table 3: Histological observations at 40 hours and 14 days post-treatment (*Amaranthus spinosus* green variant)

Group	Duration	Organ	Major Histological Features	Interpretation
50% feed (male)	40 h	Bone marrow	Normocellular with granulocytes, megakaryocytes	Active, balanced haematopoiesis
50% feed (male)	40 h	Liver	Normal hepatocytes, no inflammation	Preserved hepatic function
50% feed (female)	40 h	Bone marrow	Normocellular marrow, intact sinusoids	Normal regeneration
50% feed (female)	40 h	Liver	Normal hepatocytes, no injury	Hepatic stability

100% feed (male)	40 h	Bone marrow	Normocellular marrow, adipocytes and megakaryocytes	Regenerative haematopoiesis
100% feed (male)	40 h	Liver	Normal hepatocytes	Normal hepatic architecture
50% feed (male/female)	14 d	Bone marrow	Normocellular marrow with granulocytes, megakaryocytes	Sustained regeneration
50% feed (male/female)	14 d	Liver	Normal hepatocytes, no inflammation	Hepatic protection
100% feed (male)	14 d	Bone marrow	Hypocellular marrow, mild haemorrhage	Mild erythroid suppression
100% feed (female)	14 d	Bone marrow	Normocellular marrow, numerous sinusoids	Optimal regeneration
100% feed (all)	14 d	Liver	Normal hepatocytes, intact portal tracts	Hepatoprotection maintained

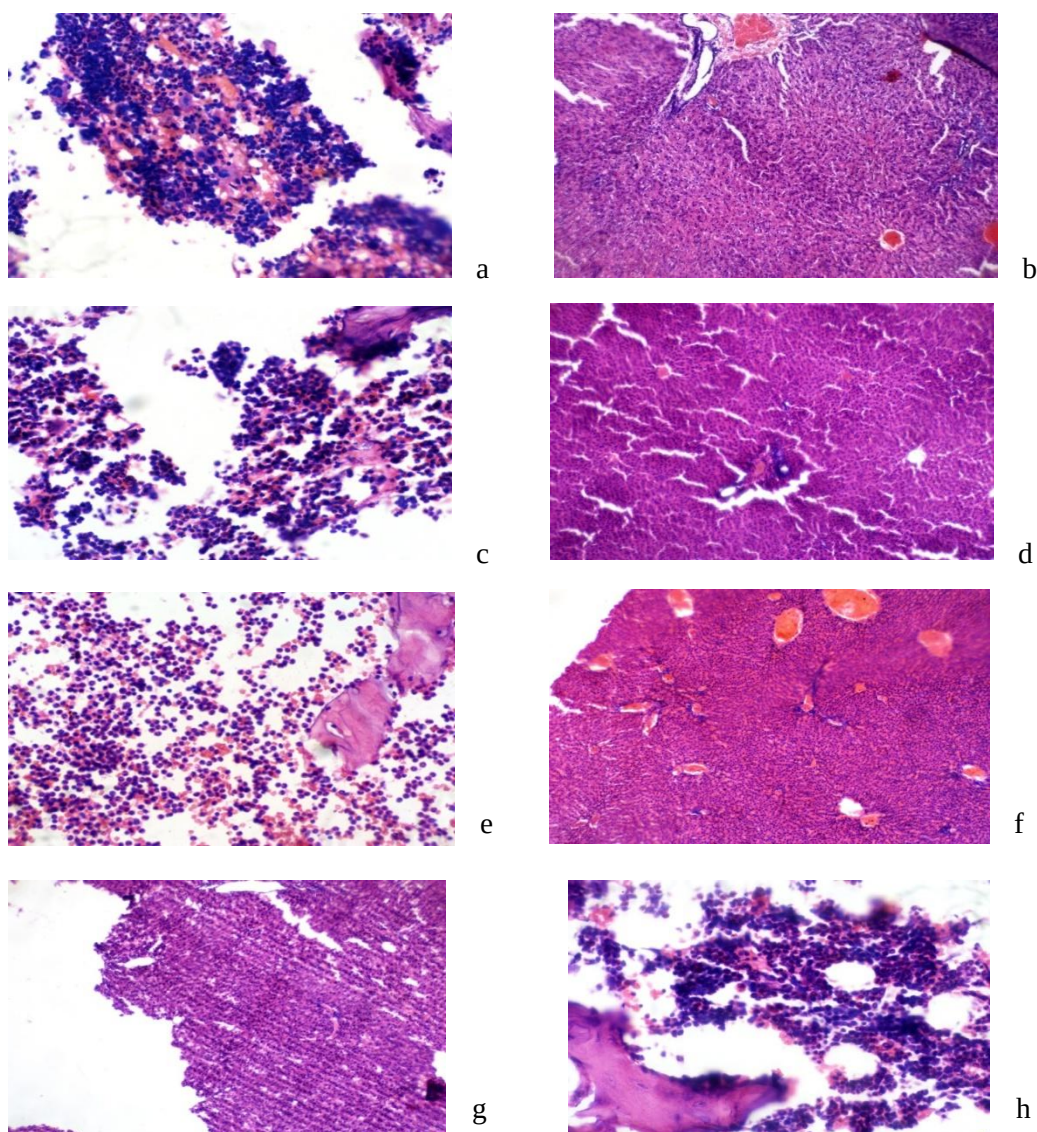


Fig. 8: Histological sections of tissues from *Amaranthus spinosus*-fed rats at 14 days post-treatment.

- (a–d) Bone marrow sections showing largely normocellular marrow with granulocytes and scattered megakaryocytes.
- (e–h) Liver sections demonstrating intact hepatic architecture with no evidence of necrosis or inflammatory infiltration. (H&E stain; $\times 400$).

Phytochemical Composition of *Amaranthus spinosus* (Green Variant)

Quantitative Phytochemical Constituents

Quantitative phytochemical analysis of the green variant of *Amaranthus spinosus* revealed a diverse composition of bioactive compounds (Table 1). Total phenolic content was 329.09 ± 0.46 mg/100 g, while tannins were present at the highest concentration (663.50 ± 0.94 mg/100 g; $P < 0.001$). Flavonoids were detected at 88.59 ± 0.44 mg/100 g, and β -carotene content was 2.17 ± 0.006 mg/100 g (Table 4).

Secondary metabolites included alkaloids ($6.95 \pm 0.05\%$), saponins ($3.51 \pm 0.001\%$), terpenoids ($3.09 \pm 0.08\%$), and glycosides ($0.56 \pm 0.006\%$). Seed phytochemical analysis revealed comparatively lower phenolic and flavonoid levels but higher alkaloid concentrations. Oxalates and phytates were present in low amounts (Table 5).

Table 4: Quantitative phytochemical composition of *A. spinosus* (green variant)

Phytochemical	Value ($\pm$ SEM)	Unit
Phenols	329.09 ± 0.46	mg/100 g
Tannins	663.50 ± 0.94	mg/100 g
Flavonoids	88.59 ± 0.44	mg/100 g
β -Carotene	2.17 ± 0.006	mg/100 g
Saponins	3.51 ± 0.001	%
Alkaloids	6.95 ± 0.05	%
Terpenoids	3.09 ± 0.08	%
Glycosides	0.56 ± 0.006	%

Table 5: Phytochemical composition of *A. spinosus* (green seeds)

Compound	Value ($\pm$ SEM)	Unit
Total tannins	145.0 ± 3.0	mg/100 g
Flavonoids	1.07 ± 0.01	mg/100 g
Total phenols	0.045 ± 0.005	mg/100 g
Alkaloids	21.2 ± 0.4	mg/100 g
Oxalates	0.105 ± 0.005	mg/100 g
Phytates	2.41 ± 0.01	mg/100 g

GC–MS Analysis

Gas chromatography–mass spectrometry (GC–MS) analysis identified 22 compounds in the leaf and seed extracts with retention times ranging from 12.34 to 28.71 min. The most abundant compounds included octadecane (18.26%), nonanoic acid (8.79%), hexadecanoic acid methyl ester (5.78%), 14,17-octadecadienoic acid methyl ester (4.71%), and octanoic acid (4.13%) (Table 6).

Compound classification indicated that hydrocarbons and fatty acids represented the dominant chemical classes, each accounting for approximately 22% of the detected compounds (Table 7). Fatty acid esters contributed 18%, while alcohols, terpenoids, and miscellaneous compounds constituted smaller proportions.

Retention time patterns indicated that polar fatty acids eluted earlier (mean retention time ≈ 13.1 min), whereas less polar hydrocarbons appeared later (mean retention time ≈ 26.7 min).

Table 6: Major GC–MS-identified compounds in *A. spinosus* (green variant)

Compound	% Abundance	Retention Time (min)
Octadecane	18.26	27.35
Nonanoic acid	8.79	13.88
Hexadecanoic acid, methyl ester	5.78	21.19
14,17-Octadecadienoic acid, methyl ester	4.71	23.26
Octanoic acid	4.13	12.34

Table 7: Compound distribution by chemical class

Chemical Class	No. of Compounds	Total (%)	Mean (%)
Fatty acids	5	22	4.4
Fatty acid esters	6	18	3.0
Terpenoids	3	8	2.7
Alcohols	4	10	2.5
Hydrocarbons	3	22	7.3
Miscellaneous	4	20	5.0

FTIR Spectroscopic Profile

FTIR spectra of *A. spinosus* leaf displayed characteristic absorption bands corresponding to several functional groups. Aliphatic C–H stretching vibrations occurred at 2919 and 2853 cm^{-1} , while an ester carbonyl band appeared at 1700 cm^{-1} . Amide I (1635 cm^{-1}) and aromatic C=C (1539 cm^{-1}) peaks were also detected. Additional absorption bands between 1240 and 1031 cm^{-1} corresponded to polysaccharide C–O–C and C–O stretching vibrations, while a peak at 678 cm^{-1} indicated aromatic C–H bending.

Proximate Composition

Proximate analysis of the leaves revealed high protein content ($31.95 \pm 0.06\%$) and ash content ($21.33 \pm 0.15\%$). Crude fibre constituted $20.65 \pm 0.04\%$, while fat content was relatively low ($4.86 \pm 0.06\%$). Carbohydrates accounted for $12.87 \pm 0.03\%$ of the dry weight (Table 8).

Table 8: Proximate composition of *A. spinosus* (green leaves)

Component	Value ($\pm$ SEM)
Moisture	8.38 ± 0.19
Ash	21.33 ± 0.15
Fibre	20.65 ± 0.04
Protein	31.95 ± 0.06
Fat	4.86 ± 0.06
Carbohydrate	12.87 ± 0.03

Mineral and Vitamin Composition

Mineral analysis of the leaves indicated high concentrations of magnesium (195.8 mg/100 g), potassium (150.6 mg/100 g), and calcium (142.2 mg/100 g). Iron content was 49.37 mg/100 g, while trace elements including copper, manganese, and zinc were present in smaller amounts (Table 9).

Seeds contained moderate levels of calcium (61.7 mg/100 g), magnesium (38.5 mg/100 g), potassium (41.3 mg/100 g), and iron (16.2 mg/100 g) (Table 10).

Vitamin analysis showed high levels of vitamins A (44.94 mg/100 g) and C (49.08 mg/100 g) in the leaves. Seeds contained lower concentrations of these vitamins (18.20 and 20.11 mg/100 g, respectively). B-complex vitamins, including thiamine (10.62 mg/100 g) and riboflavin (6.44 mg/100 g), were also present in appreciable quantities in the leaves (Table 8).

Table 9: Mineral content of *A. spinosus* (green leaves)

Element	Value (mg/100 g)
Calcium (Ca)	142.2
Magnesium (Mg)	195.8
Potassium (K)	150.6
Copper (Cu)	0.877
Manganese (Mn)	5.04
Zinc (Zn)	0.902
Iron (Fe)	49.37

Overall Summary of Experimental Findings

Anaemia induction resulted in increased bone marrow cellularity characterized by erythroid proliferation, while liver architecture remained largely preserved. Ferrous sulfate treatment was associated with moderate marrow recovery and mild hepatic alterations. Dietary supplementation with *Amaranthus spinosus* maintained normal bone marrow cellularity and preserved hepatic histology. The plant also demonstrated a diverse phytochemical composition and substantial nutritional value, including high levels of phenolics, protein, vitamins, and essential minerals.

DISCUSSION

Anaemia remains one of the most prevalent global health disorders and is characterized by reduced haemoglobin concentration and diminished oxygen-carrying capacity of the blood. It affects billions of individuals worldwide, particularly in developing regions where nutritional deficiencies and limited access to healthcare are common (McLean et al., 2021; World Health Organization [WHO], 2024). In the present study, experimental induction of anaemia resulted in marked histological alterations in bone marrow cellularity while largely preserving hepatic architecture. The pronounced marrow hypercellularity observed in untreated anaemic rats likely reflects a compensatory increase in erythropoietic activity in response to reduced circulating erythrocytes. Such erythroid hyperplasia represents a well-established physiological mechanism by which the bone marrow attempts to restore adequate oxygen transport capacity during anaemic stress (Melaku et al., 2021; Weiss & Ganz, 2019).

Treatment with ferrous sulfate produced increased bone marrow cellularity accompanied by distinct erythroid maturation, indicating active haematopoietic recovery. These findings are consistent with the established role of iron supplementation in stimulating erythropoiesis and restoring haemoglobin levels in iron-deficiency anaemia (Camaschella, 2019; Tolkien et al., 2020). However, mild hepatic inflammatory changes and central venous congestion were observed in some animals following prolonged ferrous sulfate administration. Similar hepatic alterations have been reported in previous studies and may be attributed to oxidative stress induced by excess iron deposition in hepatocytes. Iron overload can catalyze the formation of reactive oxygen species through Fenton reactions, leading to lipid peroxidation and mild hepatocellular injury (Kell & Pretorius, 2019; Nemeth & Ganz, 2021).

In contrast, dietary supplementation with the green variant of *Amaranthus spinosus* resulted in notable improvements in bone marrow histology, characterized by restoration toward normocellular marrow architecture and balanced haematopoietic activity. Unlike the ferrous sulfate-treated group, hepatic tissues from *A. spinosus*-fed animals remained structurally intact without evidence of inflammatory infiltration or hepatocellular degeneration. These observations suggest that the plant-based diet supported erythropoietic recovery while maintaining hepatic stability. Similar protective effects of *Amaranthus* species have been reported in previous studies, which attribute their hematinic properties to their high micronutrient content and antioxidant phytochemicals (Gbadamosi & Okolosi, 2021; Sarker & Oba, 2022).

The favourable histological outcomes observed in *A. spinosus*-fed rats may be partly explained by the plant's rich phytochemical profile. Quantitative analysis in the present study revealed high concentrations of phenolics, tannins, alkaloids, and saponins. Phenolic compounds and flavonoids are widely recognized for their strong antioxidant activities, which enable them to neutralize reactive oxygen species and mitigate oxidative damage associated with anaemia and iron metabolism (Batiha et al., 2020; Shahidi & Ambigaipalan, 2019). The presence of β -carotene further contributes to antioxidant defense mechanisms and supports immune function and cellular protection against oxidative stress (Sarker & Oba, 2022).

Gas chromatography–mass spectrometry (GC–MS) analysis also confirmed the presence of multiple bioactive compounds, including fatty acids, hydrocarbons, and esters. Several of these compounds have been reported to possess anti-inflammatory, antioxidant, and membrane-stabilizing properties (Wu et al., 2022). These bioactive constituents may contribute to the maintenance of hepatic integrity observed in *A. spinosus*-fed animals and may facilitate cellular recovery processes during anaemia. The nutritional composition of *A. spinosus* further supports its potential haematopoietic role. The leaves contained high levels of protein, essential minerals, and vitamins, all of which are important for erythropoiesis and general metabolic function. In particular, the relatively high iron content is significant because iron is an essential component of haemoglobin synthesis and red blood cell production (Camaschella, 2019). In addition to iron, minerals such as magnesium and calcium contribute to enzymatic processes involved in erythropoiesis and cellular metabolism (Gupta et al., 2020).

Moreover, the high concentrations of vitamins A and C observed in the leaves may enhance iron absorption and utilization. Vitamin C is known to improve non-heme iron bioavailability by reducing ferric iron to the more absorbable ferrous form in the gastrointestinal tract (Hurrell & Egli, 2019). Vitamin A has also been implicated in erythropoietic regulation and immune function, while deficiencies of this vitamin have been associated with impaired haemoglobin synthesis (Semba & Bloem, 2021). The presence of B-complex vitamins, particularly thiamine and riboflavin, further supports metabolic pathways involved in red blood cell production and energy metabolism (Gupta et al., 2020).

Taken together, the combined histological, phytochemical, and nutritional findings of this study suggest that *Amaranthus spinosus* possesses considerable potential as a functional dietary supplement capable of supporting haematopoiesis while preserving hepatic integrity. The plant's diverse bioactive constituents and micronutrient profile may act synergistically to promote erythropoietic recovery and protect tissues from oxidative damage during anaemic conditions.

Conclusion

The present study demonstrates that dietary supplementation with *Amaranthus spinosus* promotes restoration of bone marrow architecture and maintains normal hepatic histology in experimentally induced anaemic rats. The plant exhibits a rich

phytochemical and nutritional composition, including phenolics, alkaloids, vitamins, and essential minerals that collectively support haematopoiesis and antioxidant defense mechanisms. These findings highlight the potential of *A. spinosus* as a nutritionally valuable plant with promising applications in the dietary management of anaemia.

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Subsemigroup Structure of the Rhotrix Bicyclic Semigroup

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Abstract

This paper investigates the subsemigroup structure of the rhotrix bicyclic semigroup obtained as a coordinatewise extension of the classical bicyclic semigroup. Order-defined subsemigroups determined by the componentwise relations $p_i \geq q_i$, $p_i \leq q_i$ and $p_i = q_i$ are introduced and analyzed. It is shown that the order-decreasing subsemigroup forms a subsemigroup but is neither regular nor inverse, with its regular elements coinciding precisely with the diagonal elements. The order-increasing subsemigroup is established to be anti-isomorphic to the order-decreasing case, while the diagonal subsemigroup is proved to be commutative idempotent semigroup and is isomorphic to a semilattice under componentwise maximum. These results provide structural classification of order-defined subsemigroups of the rhotrix bicyclic semigroup.

Keywords: Rhotrix bicyclic semigroup; order-defined subsemigroup; regularity; idempotent structure; inverse semigroup; semilattice.

INTRODUCTION

According to Howie[4], a semigroup is non-empty set equipped with a binary operation that is closed and associative. Among the fundamental examples in semigroup theory is the classical bicyclic semigroup, whose structure plays a significant role in the study of inverse and regular semigroups see Howie[5] and continues to inspire structural extensions.

The concept of rhotrix was introduced by Ajibade[2] as an extension of matrix-ternions and matrix-noitrets earlier proposed by Atanassov and Shannon[3]. Since then, rhotrices have been widely used as underlying sets for constructing algebraic systems. For example, Mohammed, Balarabe and Imam[12] constructed semigroups based on rhotrix sets together with the rhotrix multiplication introduced by Sani[17]. Ahmed, Yisa and Ayinla[1] investigated orthogonal rhotrix groups and characterized their principal subgroups, while Mohammed and Okon[12] studied non-commutative rhotrix groups and established subgroup embedding into the classical general linear group. Further developments by Isere[6] introduced even-dimensional rhotrices and generalized multiplication for higher even dimensions, extending earlier work that focused on odd dimensions.

In our recently accepted paper, “The Concept of Rhotrix Bicyclic Semigroup”, we introduced the rhotrix bicyclic semigroup as a coordinatewise extension of the classical bicyclic semigroup and established its basic algebraic properties. Motivated in part by structural investigations of certain subgroups of the bicyclic semigroup by Makanjuola and Umar[8], the present work continues their study by examining internal subgroups defined through coordinatewise order relations.

Specifically, we analyze subsets determined by the conditions $p_i \geq q_i, p_i \leq q_i$ and $p_i = q_i$. We establish closure and associativity, investigate regularity and inverse properties, describe the idempotent structure, and determine the relationship between the order-decreasing and order-increasing subsemigroups. In particular, the diagonal subsemigroup is shown to form a commutative idempotent semigroup with a semilattice structure under componentwise maximum.

These results further clarify the internal structure of the rhotrix bicyclic semigroup and reinforce the role of rhotrices as a flexible foundation for extending classical semigroup constructions.

Preliminary

The definitions below lay the groundwork for the subsequent sections.

Definition 2.1 (Rhotrix)

A rhotrix is a rhomboidal arrangement of real numbers which are some ways, between (2×2) dimensional and (3×3) dimensional matrices of the form

$$P = \left\{ \left\langle \begin{array}{ccc} & a & \\ b & c & d \\ & e & \end{array} \right\rangle : a, b, c, d, e \in R \right\}$$

Where c is the heart of the rhotrix P denoted as $h(P)$.

Definition 2.2 (Addition of rhotrices)

The sum of two rhotrices P and Q is defined as follows:

$$P + Q = \left\langle \begin{array}{ccc} a & & \\ b & h(P) & d \\ & e & \end{array} \right\rangle + \left\langle \begin{array}{ccc} f & & \\ g & h(Q) & j \\ & k & \end{array} \right\rangle = \left\langle \begin{array}{ccc} a + f & & \\ b + g & h(P) + h(Q) & d + j \\ & e + k & \end{array} \right\rangle$$

Definition 2.3 (Multiplication of Rhotrices)

The multiplication of heart based rhotrices was first introduced by Ajibade[] and is defined as follows:

$$P \circ Q = \left\langle \begin{array}{ccc} & ah(Q) + fh(P) & \\ bh(Q) + gh(P) & h(P)h(Q) & dh(Q) + jh(P) \\ & eh(Q) + kh(P) & \end{array} \right\rangle$$

An alternative multiplication method for heart-based rhotrices, known as the “row-column” method, was introduced by Sani[16] and is defined as follows:

$$P \circ Q = \left\langle \begin{array}{ccc} & af + dg & \\ bf + eg & h(P)h(Q) & aj + dk \\ & bj + ek & \end{array} \right\rangle$$

We now recall the classical bicyclic structure and its coordinatewise extension to the rhotrix setting.

Definition 2.4 (Classical Bicyclic Semigroup)

Let $N_0 := 0, 1, 2, \dots$

The classical bicyclic semigroup is defined as

$$B := \{(m, n) : m, n \in N_0\},$$

With binary operation $\circ$ given by

$$(m, n) \circ (r, s) = (m + r - \min(n, r), n + s - \min(n, r))$$

It is well known that B is associative and forms a fundamental example in semigroup theory.

Definition 2.5 (Rhotrix Bicyclic Set)

Let $k \in N_0$ be fixed

For $P = (p_1, p_2, \dots, p_k)$ and $Q = (q_1, q_2, \dots, q_k)$ in N_0^k , define

$$B(N_0) := \{(P, Q) : P, Q \in N_0^k\}.$$

Multiplication on $B(N_0)$ is defined componentwise by

$$(P, Q) * (R, S) = ((p_i + r_i - \min(q_i, r_i), (q_i + s_i - \min(q_i, r_i)))_{i=1}^k$$

Thus, multiplication in each coordinate follows the classical bicyclic rule. Consequently, associativity of $B(N_0)$ follows from associativity in each coordinate.

Note that rhotrices are denoted by capital letters, while their entries are denoted componentwise by lower case letters.

Definition 2.6 (Order-Defined Subsemigroups)

We now introduce subsemigroups determined by coordinatewise order relations.

Order-decreasing subsemigroup

$$B^-(N_0) = \{(P, Q) \in B(N_0) : p_i \geq q_i \text{ for all } i\}.$$

Order-increasing subsemigroup

$$B^+(N_0) = \{(P, Q) \in B(N_0) : p_i \leq q_i \text{ for all } i\}$$

Diagonal(order-equality) subsemigroup

$$B^0(N_0) = \{(P, Q) \in B(N_0) : p_i = q_i \text{ for all } i\}$$

It is immediate that

$$B^0(N_0) = B^-(N_0) \cap B^+(N_0)$$

We now proceed to investigate the structural properties of these order-defined subsemigroups, beginning with $B^-(N_0)$.

Subsemigroup Structure of $B^-(N_0)$

We begin by establishing that $B^-(N_0)$ forms a subsemigroup of the rhotrix bicyclic semigroup $B(N_0)$ which is the coordinatewise extension of the classical bicyclic semigroup.

Theorem 3.1

$B^-(N_0)$ is closed under the operation of $B(N_0)$.

Proof

Scalar Case:

Let (m, n) and (r, s) be elements of the classical bicyclic semigroup satisfying $m \geq n$ and $r \geq s$.

Their product

$$(m, n) \circ (r, s) = (m + r - \min(n, r), n + s - \min(n, r)).$$

$$\text{Let } x = m + r - \min(n, r), \quad y = n + s - \min(n, r)$$

We show that $x \geq y$

$$\text{Observe that } x - y = (m - n) + (r - s).$$

Since $m \geq n$ and $r \geq s$, both terms on the right-hand side are non-negative. Hence, $x - y \geq 0$, so $x \geq y$.

Thus, the order condition is preserved in the scalar case.

Componentwise Case:

Let $(P, Q), (R, S) \in B^-(N_0)$. Then for every coordinate i , $p_i \geq q_i$ and $r_i \geq s_i$. By scalar argument above, $p_i + r_i - \min(q_i, r_i) \geq q_i + s_i - \min(q_i, r_i)$.

Hence, the product $(P, Q) * (R, S)$ satisfies the order condition in every coordinate. Therefore, $(P, Q) * (R, S) \in B^-(N_0)$.

Thus, $B^-(N_0)$ is closed under the binary operation $*$.

Theorem 3.2

The operation $*$ on $B^-(N_0)$ is associative.

Proof

Since $B(N_0)$ is defined componentwise from the classical bicyclic semigroup, and the classical bicyclic semigroup is associative, it follows that multiplication in each coordinate is associative.

Therefore, for all

$$(P, Q), (R, S), (T, U) \in B^-(N_0),$$

$$((P, Q) * (R, S)) * (T, U) = (P, Q) * ((R, S) * (T, U))$$

Hence, $B^-(N_0)$ is associative.

Corollary 3.1

$(B^-(N_0), *)$ is a subsemigroup of $(B(N_0), *)$.

Example 3.1

$$\left[\left(\begin{array}{c} \langle 3 \\ 2 \ 3 \ 4 \\ 5 \end{array} \right), \left(\begin{array}{c} \langle 2 \\ 2 \ 2 \ 3 \\ 4 \end{array} \right) * \left(\begin{array}{c} \langle 4 \\ 3 \ 2 \ 1 \\ 4 \end{array} \right), \left(\begin{array}{c} \langle 3 \\ 2 \ 2 \ 1 \\ 3 \end{array} \right) \right] * \left[\left(\begin{array}{c} \langle 1 \\ 3 \ 2 \ 4 \\ 3 \end{array} \right), \left(\begin{array}{c} \langle 1 \\ 3 \ 2 \ 3 \\ 1 \end{array} \right) \right]$$

$$\begin{aligned}
 &= \left(\begin{array}{c} \left(\begin{array}{ccc} & 5 & \\ 3 & 3 & 4 \\ & 5 & \end{array} \right), \left(\begin{array}{ccc} & 3 & \\ 2 & 2 & 3 \\ & 3 & \end{array} \right) * \left(\begin{array}{ccc} & 1 & \\ 3 & 2 & 4 \\ & 3 & \end{array} \right), \left(\begin{array}{ccc} & 1 & \\ 3 & 2 & 3 \\ & 1 & \end{array} \right) \end{array} \right) \\
 &= \left(\begin{array}{c} \left(\begin{array}{ccc} & 5 & \\ 4 & 3 & 5 \\ & 5 & \end{array} \right), \left(\begin{array}{ccc} & 3 & \\ 3 & 2 & 3 \\ & 1 & \end{array} \right) \end{array} \right)
 \end{aligned}$$

Similarly,

$$\begin{aligned}
 &\left(\begin{array}{c} \left(\begin{array}{ccc} & 3 & \\ 2 & 3 & 4 \\ & 5 & \end{array} \right), \left(\begin{array}{ccc} & 2 & \\ 2 & 2 & 3 \\ & 4 & \end{array} \right) * \left[\left(\begin{array}{ccc} & 4 & \\ 3 & 2 & 1 \\ & 4 & \end{array} \right), \left(\begin{array}{ccc} & 3 & \\ 2 & 2 & 1 \\ & 3 & \end{array} \right) * \left(\begin{array}{ccc} & 1 & \\ 3 & 2 & 4 \\ & 3 & \end{array} \right), \left(\begin{array}{ccc} & 1 & \\ 3 & 2 & 3 \\ & 1 & \end{array} \right) \right] \end{array} \right) \\
 &= \left(\begin{array}{c} \left(\begin{array}{ccc} & 3 & \\ 2 & 3 & 4 \\ & 5 & \end{array} \right), \left(\begin{array}{ccc} & 2 & \\ 2 & 2 & 3 \\ & 4 & \end{array} \right) * \left(\begin{array}{ccc} & 4 & \\ 4 & 2 & 4 \\ & 4 & \end{array} \right), \left(\begin{array}{ccc} & 3 & \\ 3 & 2 & 3 \\ & 1 & \end{array} \right) \end{array} \right) \\
 &= \left(\begin{array}{c} \left(\begin{array}{ccc} & 5 & \\ 4 & 3 & 5 \\ & 5 & \end{array} \right), \left(\begin{array}{ccc} & 3 & \\ 3 & 2 & 3 \\ & 1 & \end{array} \right) \end{array} \right)
 \end{aligned}$$

Theorem 3.3

The scalar order-decreasing subsemigroup of B^- is not regular.

Proof

Let $a = (m, n) \in B^-$ with $m > n$. In the full bicyclic semigroup, the unique inverse of (m, n) is

$$(m, n)^{-1} = (n, m).$$

Regularity requires the existence of $x \in B^-$ such that $a = axa$.

From bicyclic structure theory, any element satisfying $a = axa$ must be the inverse (n, m) .

However,

$$(n, m) \in B^- \Leftrightarrow n \geq m,$$

which contradicts $m > n$. Hence no such $x \in B^-$ exists. Therefore, a is not regular in B^- . Since there exists at least one regular element, B^- is not regular.

Proposition 3.1 (Characterization of Regular Elements)

An element $(m, n) \in B^-$ is regular if and only if $m = n$.

Proof

If $m = n$, then $(m, m)^\circ(m, m)^\circ(m, m) = (m, m)$, so the element is regular. Conversely, If $m > n$, from Theorem 3.3 the element is not regular.

Now let us pass to the rhotrix structure.

Let

$$B^-(N_0) = \{(P, Q) \in B(N_0) : p_i \geq q_i \text{ for all } i\},$$

where multiplication is defined componenewise from the scalar bicyclic operation.

Theorem 3.4

The rhotrix order-decreasing bicyclic subsemigroup $B^-(N_0)$ is not regular.

Proof

Let $A = (P, Q) \in B^-(N_0)$, so that $p_i \geq q_i$ for every component. Regularity would require the existence of $X = (R, S) \in B^-(N_0)$ such that $A = AXA$.

Since multiplication is defined componenewise, this equality holds if and only if for each i ,

$$(p_i, q_i) = (p_i, q_i)(r_i, s_i)(p_i, q_i)$$

in the scalar bicyclic semigroup. But from Proposition 3.1, this is possible only when $p_i = q_i$ for each i . Hence, if there exists at least one index i with $p_i > q_i$, then A is not regular. Therefore, $B^-(N_0)$ is not regular.

Corollary 3.2

An element $(P, Q) \in B^-(N_0)$ is regular if and only if $P = Q$ componenewise.

Idempotent Structure of $B^-(N_0)$

Theorem 3.5

The idempotents of the bicyclic semigroup B are precisely the elements of the form (n, n) , $n \in N_0$.

Proof

Let $(m, n) \in B$ be idempotent. Then $(m, n)^2 = (m, n)$.

Compute:

$$(m, n)(m, n) = (m + m - \min(n, m), n + n - \min(n, m))$$

Let $k = \min(n, m)$. Then, $(m, n)^2 = (2m - k, 2n - k)$.

For idempotency, we require $(2m - k, 2n - k) = (m, n)$. Thus, $2m - k = m$ and $2n - k = n$.

Hence, $m = k$ and $n = k$. So $m = n$.

Conversely, if $m = n$, then $(n, n)^2 = (n, n)$. Thus, the idempotents are exactly (n, n) .

Idempotents in the Scalar Order-Decreasing Subsemigroup:

Recall

$$B^- := \{(m, n) \in B : m \geq n\}.$$

Since the scalar idempotents are (n, n) , and these satisfy $n \geq n$, we obtain:

Corollary 3.3

The idempotents of B^- are precisely

$$E(B^-) = \{(n, n) : n \in N_0\}$$

Thus, the order restriction does not change the idempotent set.

Proposition 3.2 (Commutativity of Idempotents)

For $m, n \in N_0$

$$(m, m)^\circ(n, n) = (m + n - \min(m, n), m + n - \min(m, n)).$$

This simplifies to $(\max(m, n), \max(m, n))$, since the expression is symmetric in m, n , we have

$$(m, m)^\circ(n, n) = (n, n)^\circ(m, m)$$

Hence the idempotents commute.

Idempotents in the Rhotrix Order-Decreasing Subsemigroup

Now consider the rhotrix order-decreasing subsemigroup

$$B^-(N_0) := \{(P, Q) : p_i \geq q_i \text{ for all } i\}.$$

Multiplication is defined componentwise.

Theorem 3.6

The idempotents of $B^-(N_0)$ are precisely the elements of the form: (P, P) .

Proof

Let $A = (P, Q) \in B^-(N_0)$ be idempotent. Then $A^2 = A$. Since multiplication is componentwise,

This holds if and only if

$$(p_i, q_i)^2 = (p_i, q_i) \text{ for all } i$$

From the scalar result, this implies $p_i = q_i$ for all i . Hence $P = Q$.

Conversely, if $P = Q$, then $(P, P)^2 = (P, P)$.

Thus, idempotents are exactly those of the form (P, P) .

Inverse Property of the Order-Decreasing Subsemigroup

Theorem 3.7

The scalar order decreasing subsemigroup B^- is not an inverse semigroup.

Proof

Let $a = (m, n) \in B^-$ with $m > n$.

If B^- were inverse, there would exist $b \in B^-$ such that $a = aba$ and $b = bab$. But in the bicyclic semigroup, the inverse of (m, n) is uniquely (n, m) . Since $(n, m) \notin B^-$, no such b exists in B^- . Hence B^- is not inverse.

Proposition 3.3

An element $(m, n) \in B^-$ possesses an inverse in B^- if and only if $m = n$.

Proof

If $m = n$, then $(m, m)^{-1} = (m, m)$, and clearly $(m, m) \in B^-$.

Conversely, if $m > n$ its inverse lies outside B^- .

Now consider the rhotrix order-decreasing subsemigroup

$$B^-(N_0) = \{(P, Q) \in B(N_0) : p_i \geq q_i \text{ for all } i\}.$$

Multiplication is defined componentwise.

Theorem 3.8

The rhotrix order-decreasing bicyclic subsemigroup $B^-(N_0)$ is not an inverse semigroup.

Proof

Let $A = (P, Q) \in B^-(N_0)$ with $p_j = q_j$ for some index j . In full rhotrix bicyclic semigroup, the inverse is $A^{-1} = (Q, P)$.

But $(Q, P) \in B^-(N_0) \Leftrightarrow q_i \geq p_i$ for all i , which contradicts $p_j > q_j$. Hence, A does not possess an inverse in $B^-(N_0)$. Therefore, $B^-(N_0)$ is not inverse.

Corollary 3.4

An element $(P, Q) \in B^-(N_0)$ has an inverse in $B^-(N_0)$ if and only if $P = Q$ componenewise.

Commutative of the Order-Decreasing Subsemigroup

Theorem 3.9

The scalar order-decreasing subsemigroup B^- is not commutative.

Proof

To prove non-commutativity, it suffices to prove a counter example.

Take $a = (2, 1)$, $b = (1, 0)$. Both satisfy $m \geq n$, hence belong to B^- .

Compute $ab: (2, 1)(1, 0) = (2 + 1 - \min(1, 1), 1 + 0 - \min(1, 1)) = (2, 0)$, since $\min(1, 1) = 1$

Compute $ba: (1, 0)(2, 1) = (1 + 2 - \min(0, 2), 0 + 1 - \min(0, 2)) = (3, 1)$, since $\min(0, 2) = 0$

Clearly $ab \neq ba$.

Hence B^- is not commutative.

Theorem 3.10

The rhotrix order-decreasing bicyclic subsemigroup $B^-(N_0)$ is not commutative.

Proof

Since multiplication is defined componenewise, commutativity in $B^-(N_0)$ would imply commutativity in scalar coordinate. But from Theorem 3.9, the scalar subsemigroup B^- is not commutative. Hence, there exist scalar components a_i, b_i such that $a_i b_i \neq b_i a_i$.

Construct rhotrix elements whose entries agree with this counter example in one coordinate and are equal elsewhere. Then the resulting rhotrix products differ in that coordinate. Therefore, $AB \neq BA$. Hence $B^-(N_0)$ is not commutative.

The Order-Increasing Rhotrix Bicyclic Subsemigroup

We now show that the order-increasing structure is completely determined by the order-decreasing one via an anti-isomorphism. Consequently, no repetition of proofs is required.

Scalar Anti-Isomorphism

Let $B := \{(m, n): m, n \in N_0\}$ be the classical bicyclic semigroup.

Define map $\delta: B \rightarrow B$ by $\delta(m, n) = (n, m)$.

Proposition 4.1

The mapping δ is an anti-isomorphism of B .

Proof

Let $(m, n), (r, s) \in B$. Recall the multiplication:

$(m, n) \circ (r, s) = (m + r - \min(n, r), n + s - \min(n, r))$. Then

$\delta((m, n) \circ (r, s)) = (n + s - \min(n, r), m + r - \min(n, r))$.

On the other hand,

$$\delta(r, s)\delta(m, n) = (s, r)(n, m)$$

Compute: $(s, r) \circ (n, m) = (s + r - \min(r, n), r + m - \min(r, n))$. Since $\min(n, r) = \min(r, n)$, both expressions coincide. Hence,

$$\delta((m, n) \circ (r, s)) = \delta(r, s)\delta(m, n).$$

Thus δ reverses multiplication order and being clearly bijective, it is an anti-isomorphism.

Between B^- and B^+

Define: $B^- := \{(m, n) \in B : m \geq n\}$ and $B^+ := \{(m, n) \in B : m \leq n\}$

Theorem 4.2

$$\delta(B^-) = B^+$$

Hence B^- and B^+ are anti-isomorphic.

Proof

Let $(m, n) \in B^-$. Then $m \geq n$. Thus, $\delta(m, n) = (n, m)$, and since $n \leq m$, we have $(n, m) \in B^+$.

Conversely, if $(a, b) \in B^+$, then $a \leq b$, so $(b, a) \in B^-$, and $\delta(b, a) = (a, b)$. Thus, $\delta(B^+) = B^-$.

Since δ is an anti-isomorphism of B , its restriction gives an anti-isomorphism between B^- and B^+ .

Now define the rhotrix order-increasing subsemigroup

$$B^+(N_0) := \{(P, Q) \in B(N_0) : p_i \leq q_i \text{ for all } i\}$$

Define $\beta : B(N_0) \rightarrow B(N_0)$ by $\beta(P, Q) = (Q, P)$

Theorem 4.3

The mapping β is an anti-isomorphism of the rhotrix bicyclic semigroup, and $\beta(B^-(N_0)) = B^+(N_0)$.

Since multiplication is defined componentwise, for any $(P, Q), (R, S)$,

$$\beta((P, Q), (R, S)) = \beta(R, S)\beta(P, Q) \text{ componenetwise by Proposition 4.1.}$$

Moreover,

$$(P, Q) \in B^-(N_0) \Leftrightarrow p_i \geq q_i \text{ for all } i,$$

$$\Leftrightarrow q_i \leq p_i \text{ for all } i, \Leftrightarrow (Q, P) \in B^+(N_0).$$

The order-increasing rhotrix bicyclic subsemigroup $B^+(N_0)$ is anti-isomorphic to the order-decreasing subsemigroup $B^-(N_0)$.

Therefore, all structural properties established for $B^-(N_0)$ holds dually for $B^+(N_0)$.

Order-Equality Rhotrix Bicyclic Semigroup

We now analyse the order-defined subsemigroup

$$B^0(N_0) := \{(P, Q) \in B(N_0) : p_i = q_i \text{ for all } i\}.$$

Scalar Diagonal Subsemigroup

Let $B := \{(m, n) : m, n \in N_0\}$ be the classical bicyclic semigroup. Define the diagonal subset

$$B^0 := \{(n, n) : n \in N_0\}.$$

Theorem 5.1

B^0 is a commutative subsemigroup of B .

Proof

Take $(m, m), (n, n) \in B^0$

Compute: $(m, m) \circ (n, n) = (m + n - \min(m, n), m + n - \min(m, n))$

$$\text{Since } m + n - \min(m, n) = \max(m, n)$$

$(m, m)(n, n) = (\max(m, n), \max(m, n))$. Thus, the product remains diagonal, so the set is closed.

Because $\max(m, n) = \max(n, m)$,

$(m, m)(n, n) = (n, n)(m, m)$. Hence, it is commutative.

The structural interpretation of this result is that the operation reduces to:

$(m, m)(n, n) = (\max(m, n), \max(m, n))$. Thus, B^0 is isomorphic to $(N_0, \max)$.

Theorem 5.2

$B^0(N_0)$ is a commutative idempotent subsemigroup of $B(N_0)$.

Proof

Let $(P, P), (R, R) \in B^0(N_0)$. Since multiplication is componentwise,

$(P, P) * (R, R) = (\max(p_i, r_i), \max(p_i, r_i))$. Thus:

- (i) The product is diagonal
- (ii) Every element satisfies $(P, P)^2 = (P, P)$

Therefore, $B^0(N_0)$ is a commutative band (indeed semilattice).

$$B^0(N_0) = B^-(N_0) \cap B^+(N_0)$$

So, the diagonal is the intersection of the order-decreasing and order-increasing subsemigroups.

Conclusion

This paper extends our earlier work on the rhotrix bicyclic semigroup by examining internal subsemigroups defined through coordinatewise order relations. We showed that the order-decreasing subset forms subsemigroup but is neither regular nor

inverse in general, and that its regular elements coincide with the diagonal elements. The order-increasing subsemigroup was shown to possess corresponding structural properties, while the diagonal subsemigroup was identified as a commutative idempotent semigroup admitting a semilattice structure under componentwise maximum.

These findings provide clearer structural decomposition of the rhotrix bicyclic semigroup and further illustrate how order constraints shape the algebraic behavior of coordinatewise extensions of the classical bicyclic semigroup.

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**Assessment of knowledge of Hypertension among Non-Teaching Staff of
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Abstract

Hypertension is considered a major global health issue, resulting in approximately 9.4 million deaths annually. Hypertension also known as a silent killer, it is often diagnosed incidentally. Untreated hypertension can lead to severe complications such as cardiovascular disease, kidney failure, and stroke resulting in disabilities. Awareness and knowledge of hypertension have been validated as effective method for the prevention and better management of the condition. This study aimed to assess the knowledge of hypertension amongst non-teaching staff of College of Health Sciences, University of Ilorin.

The study used a cross-sectional design and a purposeful sampling technique. A self-administered questionnaires were used to assess the perceived knowledge and awareness of Hypertension among the participants using Hypertension Evaluation of Lifestyle and Management Scale (HELM). Descriptive statistics were used to analysed the data using frequency and simple percentage presented in a bar chart.

A total of eighty-one (81) questionnaires were administered, 67 copies were appropriately filled and returned, corresponding to 82.7% response rate. Meanwhile 58.2%, 17.9% and 23.9% of the participants show high, moderate and low overall degree of knowledge of hypertension respectively. 85.0% of the participants declare high awareness of risk factors of hypertension, 74.6% showed high knowledge on methods of reducing hypertension and 61.2% showed high level of knowledge of complications of hypertension. Non-academic staff participants with the highest education qualification demonstrated highest degree of knowledge of hypertension.

In this survey, there was high knowledge and awareness of hypertension among non-academic staff of college of Health Sciences, University of Ilorin. Continuous workplace advocacy is essential with greater emphasis on ways of reducing hypertension and complications of hypertension.

Keywords: hypertension, prevention, knowledge, awareness, risk factors,

Introduction

The development of heart disease, stroke, renal failure, premature death, and disabilities are all influenced by hypertension, a global public health concern [1]. Cardiovascular diseases (CVDs) are the leading cause of death globally. An estimated 19.8 million people died from CVDs in 2022, representing approximately 32% of all global deaths. Of these deaths, 85% were due to heart attack and stroke. Over three quarters of CVD deaths take place in low- and middle-income countries [2]. In reaction to this danger, International and local organization have created and maintained an updating policy linked to hypertension management and strategies for promoting health in managing hypertension to lessen or avoid its potential from leading to additional disabilities [1]. These efforts have reduced the occurrence of hypertension in high income countries, while the prevalence have been increasing in middle and low-income countries [3].

The incidence and prevalence of hypertension keep rising around the world [4], the number of adults with hypertension has risen from 650 million in 1990 to 1.4 billion in 2024. Estimate suggest that the prevalence of hypertension among adults may rise between 1.5 billion to 1.56 billion by the year 2025 [5]. Currently, an estimate of 1.23 billion adults aged 30-79 years worldwide have hypertension, 46% were not aware that they had it [2]. In Nigeria, research indicates varying yet significant prevalence rates, such as 27.1% among employees in southwest Nigeria [6] and an overall age-standardized prevalence of 38.1% across different geopolitical zones [7]. Another study was conducted in Nigeria on the evaluation of knowledge risk factors for hypertension among staff of tertiary institution and it showed that most of the staff (62.1%) lacked awareness of their blood pressure status [8]. Unfortunately, despite the high prevalence of hypertension in Africa, there is limited number of scholarly research on understanding hypertension in Africa including Nigeria. This raised a serious apprehension to both the researchers and the academicians in the country.

Despite the high prevalence of hypertension, there remain significant gaps in awareness, treatment, and control on a global scale, with 50–75% of hypertensive individuals not receiving the necessary care [9, 10]. In certain contexts, such as the data from Iran in 2011, it was found that 43.2% of individuals were not aware of their condition, 34.8% were receiving treatment, and 38.6% had managed their hypertension, with variation across different regions. In Nigeria, a lack of awareness is particularly pronounced, as demonstrated by a study indicating that 62.1% of staff at tertiary institutions were ignorant of their blood pressure readings [11].

Africa, and Nigeria in particular, is confronted with a significant burden of hypertension; however, there is a scarcity of academic research addressing knowledge levels, which raises concerns among scholars and researchers. Hypertension was identified as a global health challenge by the WHO in 2023, contributing to 64 million disability-adjusted life years (DALYs) in both developed and developing nations [2]. This condition adversely affects productivity, results in workforce shortages, and leads to early retirements, thereby having economic repercussions [11, 2].

Existing empirical studies indicate a scarcity of research regarding hypertension awareness, particularly in Kwara State, Nigeria, which underscores a significant gap [12]. Consequently, the study intends to assess the understanding of hypertension among non-teaching personnel at the College of Health Sciences, University of Ilorin, Kwara State, Nigeria, to tackle this vital issue considering the concerning increases observed both globally and locally.

Consequently, the study is specifically sought to; determine the awareness and understanding of hypertension among non-teaching staff of College of Health Science, University of Ilorin, Kwara State, Nigeria.

Methodology

A cross-sectional study design was employed to evaluate awareness and knowledge of hypertension among non-teaching-staff of College of Health Sciences University of Ilorin. The study did not include any high-risk groups; thus, ethical approval was waived. Participants received clear and comprehensive information through informed consent that outlined the study's purpose and benefits. Confidentiality was strictly maintained by safeguarding personal information, and anonymity was prioritized to protect participants' privacy and encourage honest responses. Participants were free to leave the study at any time without facing any repercussions because voluntary participation was strictly enforced.

Participants

The study adopted a purposeful sampling technique and participant were approached physically and notified ahead of time through verbal word of mouth communication. Eligibility required participants to be non-teaching staff of College of Health Sciences University of Ilorin, who had obtained a minimum of O level certificate. Excluded from this investigation were academic staff members, as they were considered medical personnel who are already likely to possess adequate knowledge and awareness of hypertension.

Study Setting

The research was carried out between January and March of 2024, and participants were drawn from the college irrespective of the specialization and job role provided they are non-teaching staff of the college of health sciences, of the University. The University of Ilorin is situated in Ilorin town, State Capital of Kwara State, Nigeria. It was established in 1975. College of Health sciences is a section of University of Ilorin that handles medical courses it consists of three faculties with all most two dozen departments. The faculties include Faculty of Clinical Sciences, Faculty of Basic Clinical Sciences and Faculty of Basic Medical Sciences.

Data Collection Procedure

Self-administered questionnaires were utilised for data collection. The questionnaire was structured to assess participants perceived knowledge and awareness of Hypertension, lifestyle and management Skill among non-teaching staff across all the faculties in College of Health Sciences, University of Ilorin. The early section of the questionnaire covers questions on basic participant's demographics, while the other section covers questions to evaluate knowledge and awareness of hypertension. Questions were formulated based on the Hypertension Evaluation of Lifestyle and Management (HELM) scale. It is a validated 14-item instrument designed to assess a patient's knowledge of hypertension self-management. HELM addresses three main areas that assist patients in actively managing their condition: general knowledge of hypertension, lifestyle/medication, and treatment goals.

Data analysis

Data analysis was done with Microsoft excel version 2016 and Statistical Package for the Social Sciences (SPSS). The responses of the participants were analyzed with descriptive statistics (frequency and percentage). Standard deviations and means were used to display categorical data. Respondents were ranked according to their level

of knowledge on awareness of hypertension risk factor using the following parameters: high (above 50%), moderate (above 20%) and low (below 20%) awareness of risk factors for hypertension. Sociodemographic characteristics of the respondents were compiled using percentages and frequency counts. The relationship between sociodemographic characteristics, respondents' knowledge and awareness were examined using the chi square test. P value was set as 0.05, hence, p -value < 0.05 indicates a significant relationship.

Results

Sociodemographic characteristics of participants

In total, eighty-one (81) copies of questionnaires were administered, 67 copies were appropriately filled and returned. Table 1 displays the participants' sociodemographic characteristics, 12 (17.91%) were between the ages of 18-24 years, 35 (52.24%) were between the ages 25-34 years, 16 (23.88%) were between the age of 35-44 years, and 4 (5.97%) were between the ages 45 and older. The age distribution indicates that majority of the participants fall within the ages of 25-34 years. A total of 67 respondents that participated in the study, comprising 24 (32.82%) male and 43 (64.18%) female. Additionally, out of the study's participants, 67 (100.00%) are married and 0 (0.00%) are single. There are 0 (0.00%) widows and 0 (0.00%) divorced people. Furthermore, majority of respondents 50 (74.63%) had university education, 12 respondents (17.91%) had secondary education, and 5 respondents (7.46%) had only primary education. Lastly, Table 1 below also shows, 5 (7.46%) of respondents were junior technicians, 13 (19.40%) senior technicians, 22 (32.84%) junior non-technicians, while 27 (40.30%) were senior non-technicians.

Figure 1 display the overall respondents' knowledge of hypertension, 58.2% and 23.9% of the participants showed the highest and lowest level of knowledge of hypertension, respectively.

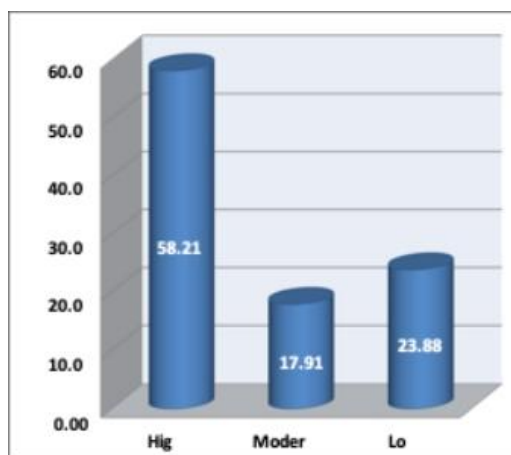


Figure 1. Overall level of knowledge of hypertension among the respondents

Furthermore, Table 1 present the relationship between sociodemographic characteristics of the respondents and their awareness of hypertension. The result shows that there is significant relationship between socio-demographic variables and participants' awareness of hypertension. The results are as follows Age, gender, marital status, educational background and working categories of the respondents/participants, <0.0001, 0.0203, <0.0001 and <0.0001, were all significant at alpha level 0.05.

Table 1. The relationship between Socio-Demographic Characteristics and the Knowledge of Hypertension

Variables	Frequency (n)	Percentage (%)	df	Chi square χ^2	P-value
Age group (year)			3	30.97	<0.0001
18-24	12	17.91			
25-34	35	52.24			
35-44	16	23.88			
45 and above	4	5.97			
Gender			1	5.39	0.0203
Male	24	35.82			
Female	43	64.18			
Marital status			2	201.0	<0.0001
Single	-	0.00			
Married	67	100			
Divorced	-	0.00			
Academic qualification			3	92.343	<0.0001
No formal education	-	0.00			
Primary	5	7.48			
Secondary	12	17.91			
Tertiary	50	74.63			
Working categories			3	17.00	<0.0007
Junior Technicians	5	7.46			
Senior Technicians	13	19.4			
Junior non-Technicians	22	32.8			
Senior non-Technicians	27	40.3			

Knowledge and awareness of hypertension risk factors

As shown in Figure 2, participants' degree of understanding of risk factors indicates that majority (82%) of the respondents' demonstrated adequate knowledge while only 13.5% exhibited low level of knowledge. Regarding the responses of the participants on knowledge of risk factors for hypertension, 53 respondents (79.10%) reported that excessive salt intake can lead to high blood pressure, 2 respondents (2.99%) agreed that excessive salt consumption causes blood pressure to fall, 1 respondent (1.49%) thought that excessive salt intake causes blood pressure to stay the same, and 11 respondents (16.42%) were unsure. Additionally, it was found that 62 respondents (92.54%) agreed that tension is one of the risk factors for hypertension, 1 respondent (1.49%) disagreed that tension is not a risk factor for hypertension, and 4 respondents (5.97%) were unsure. Additionally, 59 respondents (88.06%) agreed that excessive intake of fat can lead to hypertension, 2 respondents (2.96%) disagreed, and 6 respondents (8.98%) were unsure. Table 2 shows the summary of overall level of awareness of risk factors for hypertension among participants.

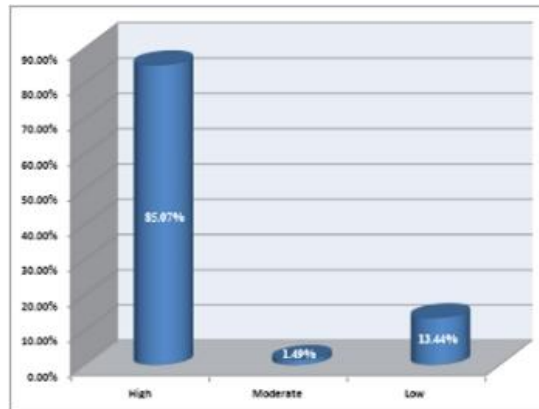


Figure 2. Overall level of awareness of risk factors of hypertension among the respondents

Table 2. Level of Knowledge of Hypertension among Respondents

S/N	Level of knowledge of hypertension among non-teaching staff	Responses n(%)			
1.	How often do you check your blood pressure	Daily 0(0.0%)	Weekly 11(16.42%)	Monthly 45(67.16%)	Not at all 11(16.42%)
2.	If someone's blood pressure is 115/75, it is...?	High 8(11.98%)	Low 12(17.91%)	Normal 32(47.76%)	Do not know 15(22.39%)
3.	If someone's blood pressure is 160/100, it is...?	High 46(68.66%)	Low 2(2.99%)	Normal 9(13.43%)	Do not know 10(14.92%)
4.	Once someone has high blood pressure, it usually lasts for ?	A few years	5-10 years 1(1.49%)	The rest of their life 36 (53.73%)	Do not know 22(32.84%)
5	High blood pressure cannot be cured	Yes 17(25.37%)	No	Don't know 5(7.47%)	

Knowledge of the Respondents on methods of Controlling Hypertension

Participants' knowledge on various methods of controlling hypertension is presented in Figure 3. Results of response to knowledge of controlling hypertension, show that 54 respondents (80.60%) agreed that individuals with elevated blood pressure should take their medication daily, 6 respondents (8.96%) agreed that they should take their medication at least a few times a week, 0 respondents (0.00%) agreed that individuals with high blood pressure should only take their prescription when they are unwell, and 7 respondents (10.44%) did not know the precise time that individual with elevated blood pressure should take their medication. Seven respondents (10.45%) indicated that losing weight typically causes blood pressure to rise, forty respondents (59.70%) thought that blood pressure typically decreases, six respondents (8.96%) reported that blood pressure typically remains unchanged, and fourteen respondents (20.89%) did not know or have idea about it. Additionally, 51 respondents (76.12%) acknowledged that moderate to vigorous exercise for 30 minutes a day, three to five times a week, lowers blood pressure, 2 respondents (2.99%) agreed, and 14 respondents (20.89%) were unsure. Additionally, 26 respondents (38.81%) stated that motivational interviewing methods are ineffective in changing patients lifestyles, 29

respondents (43.28%) disagreed, and 12 respondents (17.91%) had no idea at all. Additionally, in this study, 56 respondents (83.58%) reported that a hypertensive person should aim for a normal blood pressure of 120/80, 1 respondent (1.49%) disagreed, and 10 respondents (14.93%) didn't know about the question. Additionally, 56 respondents (83.58%) stated that persons with elevated blood pressure should consume less fat, 2 respondents (2.99%) disagreed, and 9 respondents (13.43%) were unsure. Additionally, 64 respondents (95.52%) indicated that persons with elevated blood pressure should consume more fruits and vegetables, whereas 3 respondents (4.48%) were unsure.

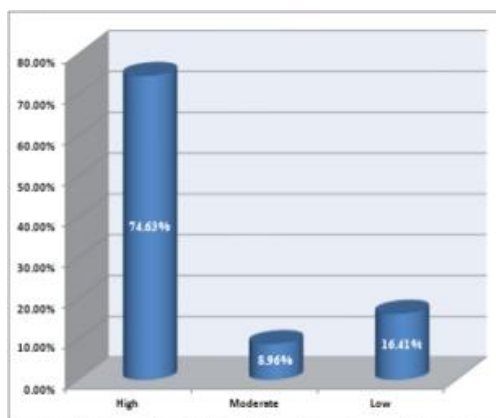


Figure 3. Overall level of knowledge of respondents on ways of reducing hypertension

Knowledge of Hypertension's Complication among the Respondents

Response regarding knowledge of complications related to hypertension is presented in Figure 4. Regarding heart attack, 60 respondents (89.55%) agreed that hypertension can result to heart attacks, whereas 7 respondents (10.45%) were unsure. Additionally, 18 respondents (26.87%) agreed that hypertension can cause heart cancer, 11 respondents (16.41%) agreed that hypertension cannot cause heart cancer, and 38 respondents (56.72%) were unsure. Regarding hypertension and renal problem, 30 respondents (44.78%) agreed that hypertension can lead to renal problems, 10 respondents (14.93%) disagreed, and 27 respondents (40.29%) had no idea. For diabetes, 41 respondents (61.19%) agreed that hypertension may lead to diabetes, 8 respondents (11.94%) disagreed, and 18 respondents (26.87%) were unsure. As for stroke, 60 respondents (89.55%) agreed that stroke can result from hypertension, whilst 7 respondents (10.45%) were unsure.

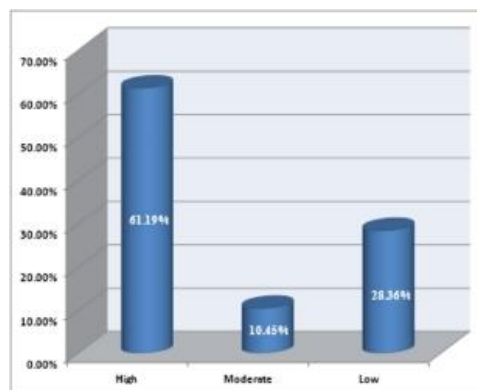


Figure 4. Overall level of knowledge of respondents on hypertension's complication

Discussion

This study assessed participants' knowledge and awareness of risk factors associated with hypertension, complications and methods of controlling it. The findings of this study indicate that the participants have reasonable high knowledge of the hypertension. This, indicates that a substantial level of awareness about hypertension exist among professionals of this category within the sample population. This finding may be linked to the educational qualification of the participants as majority (74.63%) of the participants had a tertiary/university degree. This is comparable to the results of a study that reported knowledge and awareness of hypertension to increase with educational qualification of participants [13]. On the contrary, another study found that knowledge and awareness of hypertension and its consequences were inadequate despite it being a significant health problem [14]. Another possible reason for the high-level of participants' knowledge and awareness could be ascribed to their domiciliation in the college. Of health sciences where a large proportion of them may have participated in the organizing of seminars, lectures or conferences and research on hypertension and related subjects. This might have improved their understanding and awareness thereby influencing result of the study.

The result of this study shows that participants demonstrated high knowledge of risk factors for hypertension. Therefore knowledge of hypertension risk factors has a significant effect on its prevention, management, and control by enabling individuals to adopt healthy lifestyle changes, improving adherence to treatment, and ultimately leading to better blood pressure outcomes [10,15]. Better knowledge and awareness encourage positive behaviours like healthy diet, exercise, and blood pressure monitoring, while poor knowledge may be associated with lack of high blood pressure control, highlighting the necessity of education and awareness[16]

Findings of this study reveals that, participants showed good degree of understanding towards the management and control of hypertension. Knowledge plays an important role in the management of hypertension as it helps prevent severe life-threatening complications such as stroke, renal problems, and diabetics [17]. Knowledge of methods of controlling hypertension also encourages consistent monitoring, lifestyle modification, adherence to medication and helps reduce the disease's burden and improve health outcome [18,19].

Most of the participants demonstrate high awareness of hypertension. This result is contrary to the finding of a study that found awareness and knowledge about hypertension and its consequences were inadequate despite being a serious health problem [13]. Also, the result does not align with the findings of a study that reported knowledge of hypertension among patients, was low [14].

Majority of non-teaching staff of College of Health Science, declared high knowledge of risk factor associated with hypertension. This finding is similar a study [17] that reported over 75% of Americans are aware of the risk factors linked to hypertension. However, the result is contrary to the finding of a study which reported that the knowledge of patients on the risk factor linked to high blood pressure were low [14]. Large percentage of the non-teaching staff of College of Health Science have high knowledge of methods of reducing hypertension. This result is contrary to the findings of a study that reported inadequate knowledge of hypertension, its complications and management among hypertensive patients [19].

Majority of the participants demonstrated a high level of Knowledge regarding complication of hypertension. This is similar to the finding of a study [12], who reported high level of knowledge about complications related to hypertension. Also, the result is similar to the findings of a study that revealed a greater awareness of major risk factors and complications, but unaware of minor clinical consequence 20, 20].

Conclusion

This study aimed to improve the overall health and burden of hypertension among the study population and by extension contribute to reducing global burden of the disease through efforts aimed at lowering its prevalence. It focuses on the relevance of knowledge and awareness of risk factors, complication and control measures of hypertension. It is essential to implement strategies aimed at further improve the population's knowledge and awareness of hypertension. Majority of non-teaching staff of the college have adequate awareness of risk factors, complication and control measures of hypertension. However, continuous awareness and knowledge will considerably reduce the prevalence and burden of the disease.

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Modelled Simulation of Epidemiological Differential Equations Using New Block Method

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Abstract

This study presents the development and application of a New Block Method (NBM) for the numerical simulation of first-order differential equations arising in epidemiological and real-life dynamical systems. The method is formulated using a power series polynomial approach combined with interpolation and collocation techniques to generate a high-order implicit block scheme. The resulting method is applied to three test problems, namely the susceptible-infected-recovered (SIR) model, a mixture (tank) model, and a logistic growth model. These models represent key applications in epidemiology, chemical engineering, and population dynamics, respectively. The performance of the proposed method is evaluated by comparing the computed solutions with exact analytical solutions across selected evaluation points. The results demonstrate that the NBM produces highly accurate approximations that closely match the exact solutions, as also illustrated by the graphical comparisons. The study further confirms that the method is stable, convergent, and efficient for solving nonlinear and linear differential equations. The NBM is shown to be a reliable and effective computational tool for modelling complex dynamical systems.

Keywords: New Block Method (NBM), epidemiological models, SIR model, logistic growth model, mixture model, differential equations, power series method.

Introduction

Numerical methods are essential for applied domains, which include scientific research, social science studies and engineering applications and medical practice, because exact solutions to their problems cannot be obtained. The methods enable various practical uses, which include spacecraft trajectory prediction and product safety testing and insurance risk assessment, through their combination with simulation methods that depend on solving PDEs (Adesanya, Onsachi and Odekunle, 2017). Numerical analysts develop methods to find approximate solutions, which they use to create solutions that approximate precise results, but they must overcome difficulties which need either extensive computation or limited time to achieve precise outcomes (Khalsaraei and Shokri, 2020; Kyagya, Raymond and Sabo, 2021; Ojo and Samuel, 2025). Numerical methods find applications in various fields, which include multidimensional root-finding and industry forecasts and computing and profit-loss calculations, while their theoretical and practical applications extend to engineering and

medical and business fields (Akinfenwa et al. 2020; Kwanamu et al. 2021; Ishaq et al. 2024).

The 1927 SIR model developed by Kermack and McKendrick serves as a key example for epidemiologists who use differential equations to study infectious disease transmission. The model divides populations into three groups which include susceptible (S) individuals, infective (I) individuals, and recovered (R) individuals while using oscillatory differential equations to show how diseases progress (Raymond and Sabo, 2023). Researchers create new numerical methods to study those models because they need better solutions than what existing methods can provide. The block hybrid methods use power series polynomials through interpolation and collocation to improve simulation accuracy and efficiency in epidemiological models which present difficulties because of their intricate disease spread patterns (Rizky, Mochammad and Sri, 2021; Raymond and Sabo, 2023; Bello et al. 2023; Udo and Awoyemi, 2017).

The Predictor-Corrector Method has several limitations that affect the numerical methods' efficiency and applicability in numerical computations. The method requires an initial prediction step which creates errors that spread through the entire computation process. The corrector needs multiple iterations to reach an accurate solution when the predictor delivers an incorrect result which results in higher computational expenses and longer processing durations. The problem occurs during stiff equation solution because inaccurate predictions lead to gradual convergence or complete method failure which reduces overall method stability (Adesanya, Odekunle and Alkali, 2012). The correction process uses implicit methods which require iterative methods like Newton's method. This requirement increases computational complexity especially in cases of nonlinear systems and systems that require high-dimensional processing (Tafida, 2015).

The Predictor-Corrector Method faces its main difficulty because it requires multiple function evaluations during large-scale computations. The method needs to solve equations during both prediction and correction steps which results in higher processing requirements than fully implicit methods that reduce extra work. The system loses its operational value because it cannot operate effectively to meet real-time requirements because its calculation process needs to be completed quicker (Kumleng et al., 2017). The process of selecting an appropriate step size proves essential for achieving both stability and accuracy while selecting an unsuitable step size results in unnecessary computation expenses and diminished accuracy. The implicit Runge-Kutta and Rosenbrock methods provide better stability and efficiency for solving stiff and large-scale problems than other numerical methods according to Tafida 2015. The method's accuracy and effectiveness face limitations because some implementations depend on lower-order predictors according to Adewale and Sabo 2024. The existing numerical techniques require additional development to achieve better performance across different disciplines according to Abdulrahman and Fatima 2016.

The Block Method was created as a new method to solve the problems that exist in the traditional Predictor-Corrector Method (Raymond and Sabo, 2023). The numerical method divides the time period into smaller sections which allow it to solve different schemes at various off-grid locations throughout each time section. The Block Method uses implicit integration schemes which include backward Euler and Rosenbrock methods to handle stiff and highly nonlinear systems through balanced computational distribution (Omar and Adeyeye, 2016). The primary advantages of block method is to enhanced efficiency as it divide into smaller partitions to reduces the computational burden (Khalsaraei and Shokri, 2020). The Block Method solves both the computational and stability problems of the Predictor-Corrector Method while providing research opportunities to develop better numerical methods which can effectively solve complex differential equations.

This research considers the simulation of first order oscillatory differential equation of the form

$$\frac{dy}{dx} = y, \quad y(x_0) = y(0), \quad x \in [a, b] \quad (1.1)$$

where $f : \mathfrak{R} \times \mathfrak{R}^m \rightarrow \mathfrak{R}^m$, $y, y_0 \in \mathfrak{R}^m$, f satisfy the Lipchitz condition.

Materials and Methodology

The New Block Method (NBM) derivation process in this section uses power series polynomial approximations to create a one-step solution method for the specific epidemic models that equation (1.1) defines. The method requires single-point power series interpolation together with multiple-point series collocation through alignment to partition points present in one interval.

The power series polynomial as defined by

$$y(x) = \sum_{i=0}^{\Psi} a_i \left(\frac{x - x_n}{h} \right)^i \quad (2.1)$$

The power series polynomial (2.1) is consider to simulate equation (1.1), for $x \in [x_n, x_{n+1}]$ where $\Psi = \nu + \nu - 1$, that is ν the number of interpolation and ν is the number of collocation, $n = 0, 1, 2, \dots, N - 1$, a 's the determination of the real coefficients is integral to this process. Additionally, a constant step size $h = x_n - x_{n-1}$ for partitioning the interval $[a, b]$ is prescribed by the partition itself $\pi_N : a = x_0 < x_1 < x_2 < \dots < x_n < x_{n+1} < \dots < x_N = b$

The one-step block hybrid method with ten partitions (nine grid points) were derived by using partitioned points $\left\{0, \frac{1}{10}, \frac{1}{5}, \frac{3}{10}, \frac{2}{5}, \frac{1}{2}, \frac{3}{5}, \frac{7}{10}, \frac{4}{5}, \frac{9}{10}, 1\right\}$.

Using equation (3.1) with $\nu = 1$, $\nu = 11$, we have a polynomial as

$$y(x) = \sum_{i=0}^{11} a_i \left(\frac{x - x_n}{h} \right)^i \quad (2.2)$$

Differentiating (2.2) once, to get

$$y'(x) = \sum_{i=0}^{11} i a_i \left(\frac{x - x_n}{h} \right)^{i-1} \quad (2.3)$$

Substituting (2.3) and (2.2) into (1.1) gives

$$\sum_{i=0}^{11} a_i \left(\frac{x - x_n}{h} \right)^i = f(x) \quad (2.4)$$

$$\sum_{i=0}^{11} i a_i \left(\frac{x - x_n}{h} \right)^{i-1} = f(x, y) \quad (2.5)$$

Now interpolating (2.4) at $\nu = 1$ and collocating (2.5) at

$$\nu = 0, \frac{1}{10}, \frac{1}{5}, \frac{3}{10}, \frac{2}{5}, \frac{1}{2}, \frac{3}{5}, \frac{7}{10}, \frac{4}{5}, \frac{9}{10}, 1.$$

This leads to a system of equations expressed in matrix form as

$$\begin{bmatrix} 1 & x_{n+1} & x_{n+1}^2 & x_{n+1}^3 & x_{n+1}^4 & x_{n+1}^5 & x_{n+1}^6 & x_{n+1}^7 & x_{n+1}^8 & x_{n+1}^9 & x_{n+1}^{10} & x_{n+1}^{11} \\ 0 & 1 & 2x_n & 3x_{n+1}^2 & 4x_{n+1}^3 & 5x_{n+1}^4 & 6x_{n+1}^5 & 7x_{n+1}^6 & 8x_{n+1}^7 & 9x_{n+1}^8 & 10x_{n+1}^9 & 11x_{n+1}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{10}} & 3x_{\frac{n+1}{10}}^2 & 4x_{\frac{n+1}{10}}^3 & 5x_{\frac{n+1}{10}}^4 & 6x_{\frac{n+1}{10}}^5 & 7x_{\frac{n+1}{10}}^6 & 8x_{\frac{n+1}{10}}^7 & 9x_{\frac{n+1}{10}}^8 & 10x_{\frac{n+1}{10}}^9 & 11x_{\frac{n+1}{10}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{5}} & 3x_{\frac{n+1}{5}}^2 & 4x_{\frac{n+1}{5}}^3 & 5x_{\frac{n+1}{5}}^4 & 6x_{\frac{n+1}{5}}^5 & 7x_{\frac{n+1}{5}}^6 & 8x_{\frac{n+1}{5}}^7 & 9x_{\frac{n+1}{5}}^8 & 10x_{\frac{n+1}{5}}^9 & 11x_{\frac{n+1}{5}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{10}} & 3x_{\frac{n+1}{10}}^2 & 4x_{\frac{n+1}{10}}^3 & 5x_{\frac{n+1}{10}}^4 & 6x_{\frac{n+1}{10}}^5 & 7x_{\frac{n+1}{10}}^6 & 8x_{\frac{n+1}{10}}^7 & 9x_{\frac{n+1}{10}}^8 & 10x_{\frac{n+1}{10}}^9 & 11x_{\frac{n+1}{10}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{5}} & 3x_{\frac{n+1}{5}}^2 & 4x_{\frac{n+1}{5}}^3 & 5x_{\frac{n+1}{5}}^4 & 6x_{\frac{n+1}{5}}^5 & 7x_{\frac{n+1}{5}}^6 & 8x_{\frac{n+1}{5}}^7 & 9x_{\frac{n+1}{5}}^8 & 10x_{\frac{n+1}{5}}^9 & 11x_{\frac{n+1}{5}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{2}} & 3x_{\frac{n+1}{2}}^2 & 4x_{\frac{n+1}{2}}^3 & 5x_{\frac{n+1}{2}}^4 & 6x_{\frac{n+1}{2}}^5 & 7x_{\frac{n+1}{2}}^6 & 8x_{\frac{n+1}{2}}^7 & 9x_{\frac{n+1}{2}}^8 & 10x_{\frac{n+1}{2}}^9 & 11x_{\frac{n+1}{2}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{5}} & 3x_{\frac{n+1}{5}}^2 & 4x_{\frac{n+1}{5}}^3 & 5x_{\frac{n+1}{5}}^4 & 6x_{\frac{n+1}{5}}^5 & 7x_{\frac{n+1}{5}}^6 & 8x_{\frac{n+1}{5}}^7 & 9x_{\frac{n+1}{5}}^8 & 10x_{\frac{n+1}{5}}^9 & 11x_{\frac{n+1}{5}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{10}} & 3x_{\frac{n+1}{10}}^2 & 4x_{\frac{n+1}{10}}^3 & 5x_{\frac{n+1}{10}}^4 & 6x_{\frac{n+1}{10}}^5 & 7x_{\frac{n+1}{10}}^6 & 8x_{\frac{n+1}{10}}^7 & 9x_{\frac{n+1}{10}}^8 & 10x_{\frac{n+1}{10}}^9 & 11x_{\frac{n+1}{10}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{5}} & 3x_{\frac{n+1}{5}}^2 & 4x_{\frac{n+1}{5}}^3 & 5x_{\frac{n+1}{5}}^4 & 6x_{\frac{n+1}{5}}^5 & 7x_{\frac{n+1}{5}}^6 & 8x_{\frac{n+1}{5}}^7 & 9x_{\frac{n+1}{5}}^8 & 10x_{\frac{n+1}{5}}^9 & 11x_{\frac{n+1}{5}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{10}} & 3x_{\frac{n+1}{10}}^2 & 4x_{\frac{n+1}{10}}^3 & 5x_{\frac{n+1}{10}}^4 & 6x_{\frac{n+1}{10}}^5 & 7x_{\frac{n+1}{10}}^6 & 8x_{\frac{n+1}{10}}^7 & 9x_{\frac{n+1}{10}}^8 & 10x_{\frac{n+1}{10}}^9 & 11x_{\frac{n+1}{10}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{5}} & 3x_{\frac{n+1}{5}}^2 & 4x_{\frac{n+1}{5}}^3 & 5x_{\frac{n+1}{5}}^4 & 6x_{\frac{n+1}{5}}^5 & 7x_{\frac{n+1}{5}}^6 & 8x_{\frac{n+1}{5}}^7 & 9x_{\frac{n+1}{5}}^8 & 10x_{\frac{n+1}{5}}^9 & 11x_{\frac{n+1}{5}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{10}} & 3x_{\frac{n+1}{10}}^2 & 4x_{\frac{n+1}{10}}^3 & 5x_{\frac{n+1}{10}}^4 & 6x_{\frac{n+1}{10}}^5 & 7x_{\frac{n+1}{10}}^6 & 8x_{\frac{n+1}{10}}^7 & 9x_{\frac{n+1}{10}}^8 & 10x_{\frac{n+1}{10}}^9 & 11x_{\frac{n+1}{10}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{5}} & 3x_{\frac{n+1}{5}}^2 & 4x_{\frac{n+1}{5}}^3 & 5x_{\frac{n+1}{5}}^4 & 6x_{\frac{n+1}{5}}^5 & 7x_{\frac{n+1}{5}}^6 & 8x_{\frac{n+1}{5}}^7 & 9x_{\frac{n+1}{5}}^8 & 10x_{\frac{n+1}{5}}^9 & 11x_{\frac{n+1}{5}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{10}} & 3x_{\frac{n+1}{10}}^2 & 4x_{\frac{n+1}{10}}^3 & 5x_{\frac{n+1}{10}}^4 & 6x_{\frac{n+1}{10}}^5 & 7x_{\frac{n+1}{10}}^6 & 8x_{\frac{n+1}{10}}^7 & 9x_{\frac{n+1}{10}}^8 & 10x_{\frac{n+1}{10}}^9 & 11x_{\frac{n+1}{10}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{5}} & 3x_{\frac{n+1}{5}}^2 & 4x_{\frac{n+1}{5}}^3 & 5x_{\frac{n+1}{5}}^4 & 6x_{\frac{n+1}{5}}^5 & 7x_{\frac{n+1}{5}}^6 & 8x_{\frac{n+1}{5}}^7 & 9x_{\frac{n+1}{5}}^8 & 10x_{\frac{n+1}{5}}^9 & 11x_{\frac{n+1}{5}}^{10} \\ 0 & 1 & 2x_{\frac{n+1}{10}} & 3x_{\frac{n+1}{10}}^2 & 4x_{\frac{n+1}{10}}^3 & 5x_{\frac{n+1}{10}}^4 & 6x_{\frac{n+1}{10}}^5 & 7x_{\frac{n+1}{10}}^6 & 8x_{\frac{n+1}{10}}^7 & 9x_{\frac{n+1}{10}}^8 & 10x_{\frac{n+1}{10}}^9 & 11x_{\frac{n+1}{10}}^{10} \end{bmatrix} \begin{bmatrix} a_0 \\ a_1 \\ a_2 \\ a_3 \\ a_4 \\ a_5 \\ a_6 \\ a_7 \\ a_8 \\ a_9 \\ a_{10} \\ a_{11} \end{bmatrix} = \begin{bmatrix} y_{n+1} \\ f_n \\ f_{\frac{n+1}{10}} \\ f_{\frac{n+1}{5}} \\ f_{\frac{n+1}{10}} \\ f_{\frac{n+1}{5}} \\ f_{\frac{n+1}{2}} \\ f_{\frac{n+1}{5}} \\ f_{\frac{n+1}{10}} \\ f_{\frac{n+1}{5}} \\ f_{\frac{n+1}{10}} \\ f_{\frac{n+1}{5}} \\ f_{\frac{n+1}{10}} \\ f_{\frac{n+1}{5}} \\ f_{\frac{n+1}{10}} \\ f_{\frac{n+1}{5}} \\ f_{\frac{n+1}{10}} \\ f_{\frac{n+1}{5}} \\ f_{\frac{n+1}{10}} \\ f_{\frac{n+1}{5}} \\ f_{\frac{n+1}{10}} \end{bmatrix} \quad (2.6)$$

Employing the Gaussian elimination method on equation (2.6) yields the coefficients. These coefficients are subsequently substituted into equation (1.1) to obtain the implicit continuous method in the form:

$$y(x) = a_1 y_{n+1} + h \left(\beta_0 f_{n+0} + \beta_1 f_{\frac{n+1}{10}} + \beta_2 f_{\frac{n+1}{5}} + \beta_3 f_{\frac{n+1}{10}} + \beta_4 f_{\frac{n+1}{5}} + \beta_5 f_{\frac{n+1}{2}} + \beta_6 f_{\frac{n+1}{5}} + \beta_7 f_{\frac{n+1}{10}} + \beta_8 f_{\frac{n+1}{5}} + \beta_9 f_{\frac{n+1}{10}} + \beta_{10} f_{\frac{n+1}{5}} + \beta_{11} f_{n+1} \right) \quad (2.7)$$

$$\begin{aligned} \alpha_1 &= 1 \\ \beta_0 &= -\frac{16067}{598752} + t - \frac{7381}{504}t^2 + \frac{177133}{1512}t^3 - \frac{10511875}{18144}t^4 + \frac{8542325}{4536}t^5 - \frac{5369375}{1296}t^6 + \frac{4695625}{756}t^7 - \frac{9453125}{1512}t^8 + \frac{6875000}{1701}t^9 + \frac{859375}{567}t^{10} + \frac{1562500}{6232}t^{11} \\ \beta_1 &= -\frac{26575}{149688} + 50t^2 - \frac{149688}{189}t^3 + \frac{1997825}{504}t^4 - \frac{16598525}{1134}t^5 + \frac{3775625}{108}t^6 - \frac{20965625}{378}t^7 + \frac{1046875}{18}t^8 - \frac{65937500}{1701}t^9 + \frac{312500}{21}t^{10} + \frac{15625000}{6232}t^{11} \\ \beta_2 &= \frac{16175}{199584} - \frac{225}{2}t^2 + \frac{153025}{84}t^3 - \frac{2898075}{224}t^4 + \frac{6619175}{128}t^5 - \frac{57981875}{432}t^6 + \frac{56396875}{252}t^7 - \frac{17546875}{72}t^8 + \frac{94843750}{567}t^9 - \frac{4140625}{63}t^{10} + \frac{7812500}{693}t^{11} \\ \beta_3 &= -\frac{5675}{12474} + 200t^2 - \frac{12474}{189}t^3 + \frac{10014475}{378}t^4 - \frac{21686950}{189}t^5 + \frac{8343125}{27}t^6 - \frac{33868750}{63}t^7 + \frac{5453125}{9}t^8 - \frac{242500000}{567}t^9 + \frac{32500000}{189}t^{10} + \frac{62500000}{2079}t^{11} \\ \beta_4 &= \frac{4825}{11088} - \frac{525}{2}t^2 + \frac{168775}{36}t^3 - \frac{1792225}{48}t^4 + \frac{18214675}{108}t^5 - \frac{102070625}{216}t^6 + \frac{102070625}{126}t^7 - \frac{35640625}{36}t^8 + \frac{19375000}{27}t^9 - \frac{19375000}{9}t^{10} + \frac{15625000}{297}t^{11} \\ \beta_5 &= -\frac{17807}{24948} + 252t^2 - \frac{252}{3}t^3 + \frac{149625}{4}t^4 - \frac{1563725}{9}t^5 + \frac{27074375}{54}t^6 - \frac{58608125}{63}t^7 + \frac{10000000}{9}t^8 - \frac{66875000}{81}t^9 + \frac{3125000}{9}t^{10} - \frac{6250000}{99}t^{11} \\ \beta_6 &= \frac{4825}{561108800} - 175t^2 + \frac{174025}{54}t^3 - \frac{11544725}{432}t^4 + \frac{6835775}{54}t^5 - \frac{80666875}{216}t^6 + \frac{89384375}{126}t^7 - \frac{31234375}{36}t^8 + \frac{5937500}{9}t^9 - \frac{7656250}{27}t^{10} + \frac{15625000}{297}t^{11} \\ \beta_7 &= -\frac{5675}{12474} + \frac{600}{7}t^2 - \frac{600}{63}t^3 + \frac{600}{42}t^4 - \frac{12131950}{189}t^5 + \frac{5200625}{27}t^6 - \frac{1118750}{3}t^7 + \frac{29328125}{63}t^8 - \frac{29328125}{567}t^9 + \frac{10000000}{63}t^{10} - \frac{62500000}{2079}t^{11} \\ \beta_8 &= \frac{1562500}{199584} - \frac{225}{8}t^2 + \frac{88325}{168}t^3 - \frac{996675}{224}t^4 + \frac{996675}{504}t^5 - \frac{28405625}{432}t^6 + \frac{28405625}{252}t^7 - \frac{11828125}{72}t^8 + \frac{73750000}{567}t^9 - \frac{73750000}{63}t^{10} + \frac{7812500}{693}t^{11} \\ \beta_9 &= -\frac{26575}{149688} + \frac{50}{9}t^2 - \frac{6575}{63}t^3 + \frac{4033825}{4536}t^4 - \frac{4943525}{1134}t^5 + \frac{4341875}{324}t^6 - \frac{10090625}{378}t^7 + \frac{1859375}{54}t^8 + \frac{1859375}{1701}t^9 - \frac{7187500}{567}t^{10} + \frac{15625000}{6232}t^{11} \\ \beta_{10} &= -\frac{16067}{598752} + \frac{1}{2}t^2 - \frac{7129}{756}t^3 + \frac{162875}{2016}t^4 - \frac{226150}{567}t^5 + \frac{59375}{48}t^6 - \frac{1883125}{756}t^7 + \frac{78125}{24}t^8 - \frac{4531250}{1701}t^9 + \frac{78125}{63}t^{10} + \frac{1562500}{6232}t^{11} \end{aligned} \quad (2.8)$$

and

$$t = \frac{x - x_n}{h} \quad (2.9)$$

Simplify equation (2.7) at all the grid points to obtain the new Block Method (NBM) as

$$\left. \begin{aligned} y_{n+\frac{1}{10}} &= y_n + \frac{26842253}{958003200} h f_n + \frac{164046413}{1197504000} h f_{n+\frac{1}{10}} - \frac{296725183}{1596672000} h f_{n+\frac{1}{5}} + \frac{12051709}{39916800} h f_{n+\frac{3}{10}} - \frac{33765029}{88704000} h f_{n+\frac{2}{5}} \\ &+ \frac{2227571}{6237000} h f_{n+\frac{1}{2}} - \frac{21677723}{88704000} h f_{n+\frac{3}{5}} + \frac{23643791}{199584000} h f_{n+\frac{7}{10}} - \frac{12318413}{319334400} h f_{n+\frac{4}{5}} + \frac{9071219}{1197504000} h f_{n+\frac{9}{10}} - \frac{3250433}{4790016000} h f_{n+1} \\ y_{n+\frac{1}{5}} &= y_n + \frac{2046263}{74844000} h f_n + \frac{645431}{3742200} h f_{n+\frac{1}{10}} - \frac{2149811}{24948000} h f_{n+\frac{1}{5}} + \frac{355583}{1559250} h f_{n+\frac{3}{10}} - \frac{1258463}{4158000} h f_{n+\frac{2}{5}} + \frac{904403}{3118500} h f_{n+\frac{1}{2}} \\ &- \frac{166931}{831600} h f_{n+\frac{3}{5}} + \frac{21833}{222750} h f_{n+\frac{7}{10}} - \frac{800243}{24948000} h f_{n+\frac{4}{5}} + \frac{118291}{18711000} h f_{n+\frac{9}{10}} - \frac{8501}{14968800} h f_{n+1} \\ y_{n+\frac{3}{10}} &= y_n + \frac{108223}{3942400} h f_n + \frac{840607}{4928000} h f_{n+\frac{1}{10}} - \frac{879183}{19712000} h f_{n+\frac{1}{5}} + \frac{762497}{2464000} h f_{n+\frac{3}{10}} - \frac{670233}{1971200} h f_{n+\frac{2}{5}} + \frac{24399}{77000} h f_{n+\frac{1}{2}} \\ &- \frac{2136259}{9856000} h f_{n+\frac{3}{5}} + \frac{259071}{2464000} h f_{n+\frac{7}{10}} - \frac{675441}{19712000} h f_{n+\frac{4}{5}} + \frac{6637}{985600} h f_{n+\frac{9}{10}} - \frac{11899}{19712000} h f_{n+1} \\ y_{n+\frac{2}{5}} &= y_n + \frac{64121}{2338875} h f_n + \frac{400138}{2338875} h f_{n+\frac{1}{10}} - \frac{2159}{44550} h f_{n+\frac{1}{5}} + \frac{39752}{111375} h f_{n+\frac{3}{10}} - \frac{23423}{86625} h f_{n+\frac{2}{5}} + \frac{230788}{779625} h f_{n+\frac{1}{2}} \\ &- \frac{17879}{86625} h f_{n+\frac{3}{5}} + \frac{2248}{22275} h f_{n+\frac{7}{10}} - \frac{25754}{779625} h f_{n+\frac{4}{5}} + \frac{15226}{2338875} h f_{n+\frac{9}{10}} - \frac{547}{935550} h f_{n+1} \\ y_{n+\frac{1}{2}} &= y_n + \frac{1051285}{38320128} h f_n + \frac{1636625}{9580032} h f_{n+\frac{1}{10}} - \frac{599275}{12773376} h f_{n+\frac{1}{5}} + \frac{558725}{1596672} h f_{n+\frac{3}{10}} - \frac{461275}{2128896} h f_{n+\frac{2}{5}} + \frac{17807}{49896} h f_{n+\frac{1}{2}} \\ &- \frac{465125}{2128896} h f_{n+\frac{3}{5}} + \frac{167675}{1596672} h f_{n+\frac{7}{10}} - \frac{62275}{1824768} h f_{n+\frac{4}{5}} + \frac{64175}{9580032} h f_{n+\frac{9}{10}} - \frac{22997}{38320128} h f_{n+1} \\ y_{n+\frac{3}{5}} &= y_n + \frac{1689}{61600} h f_n + \frac{13169}{77000} h f_{n+\frac{1}{10}} - \frac{14787}{308000} h f_{n+\frac{1}{5}} + \frac{1363}{3850} h f_{n+\frac{3}{10}} - \frac{35229}{154000} h f_{n+\frac{2}{5}} + \frac{16083}{38500} h f_{n+\frac{1}{2}} \\ &- \frac{25373}{154000} h f_{n+\frac{3}{5}} + \frac{1887}{19250} h f_{n+\frac{7}{10}} - \frac{2007}{61600} h f_{n+\frac{4}{5}} + \frac{71}{11000} h f_{n+\frac{9}{10}} - \frac{179}{308000} h f_{n+1} \\ y_{n+\frac{7}{10}} &= y_n + \frac{18775351}{684288000} h f_n + \frac{5843887}{34214400} h f_{n+\frac{1}{10}} - \frac{10669897}{228096000} h f_{n+\frac{1}{5}} + \frac{9973607}{28512000} h f_{n+\frac{3}{10}} - \frac{2767667}{12672000} h f_{n+\frac{2}{5}} \\ &+ \frac{353633}{891000} h f_{n+\frac{1}{2}} - \frac{241129}{2534400} h f_{n+\frac{3}{5}} + \frac{4148249}{28512000} h f_{n+\frac{7}{10}} - \frac{8312311}{228096000} h f_{n+\frac{4}{5}} + \frac{1190357}{171072000} h f_{n+\frac{9}{10}} - \frac{84427}{136857600} h f_{n+1} \\ y_{n+\frac{4}{5}} &= y_n + \frac{12818}{467775} h f_n + \frac{400448}{2338875} h f_{n+\frac{1}{10}} - \frac{38176}{779625} h f_{n+\frac{1}{5}} + \frac{278272}{779625} h f_{n+\frac{3}{10}} - \frac{12184}{51975} h f_{n+\frac{2}{5}} + \frac{330368}{779625} h f_{n+\frac{1}{2}} \\ &- \frac{34432}{259875} h f_{n+\frac{3}{5}} + \frac{176896}{779625} h f_{n+\frac{7}{10}} - \frac{3998}{779625} h f_{n+\frac{4}{5}} + \frac{2368}{467775} h f_{n+\frac{9}{10}} - \frac{1184}{2338875} h f_{n+1} \\ y_{n+\frac{9}{10}} &= y_n + \frac{542331}{19712000} h f_n + \frac{837567}{4928000} h f_{n+\frac{1}{10}} - \frac{167427}{3942400} h f_{n+\frac{1}{5}} + \frac{829089}{2464000} h f_{n+\frac{3}{10}} - \frac{1880253}{9856000} h f_{n+\frac{2}{5}} + \frac{27459}{77000} h f_{n+\frac{1}{2}} \\ &- \frac{537219}{9856000} h f_{n+\frac{3}{5}} + \frac{10773}{70400} h f_{n+\frac{7}{10}} - \frac{2065743}{19712000} h f_{n+\frac{4}{5}} + \frac{199809}{4928000} h f_{n+\frac{9}{10}} - \frac{4671}{3942400} h f_{n+1} \\ y_{n+1} &= y_n + \frac{16067}{598752} h f_n + \frac{26575}{149688} h f_{n+\frac{1}{10}} - \frac{16175}{199584} h f_{n+\frac{1}{5}} + \frac{5675}{12474} h f_{n+\frac{3}{10}} - \frac{4825}{11088} h f_{n+\frac{2}{5}} + \frac{17807}{24948} h f_{n+\frac{1}{2}} \\ &- \frac{4825}{11088} h f_{n+\frac{3}{5}} + \frac{5675}{12474} h f_{n+\frac{7}{10}} + \frac{16175}{199584} h f_{n+\frac{4}{5}} + \frac{26575}{149688} h f_{n+\frac{9}{10}} + \frac{16067}{598752} h f_{n+1} \end{aligned} \right\} \quad (2.10)$$

Numerical Properties of New Block Method (NBM)

The analysis of the basic properties of NBM are analyzed numerically, such as order and error constant, consistency, zero-stability and the region of absolute stability.

Order and Error Constant of NBM

The order and error constant of NBM was carryout according to Kumleng et al. (2017), by using.

$$\left. \begin{aligned}
 & \sum_{j=0}^{\infty} \frac{\left(\frac{1}{10}\right)^j}{j!} - y_n - \sum_{j=0}^{\infty} \frac{h^{j+1}}{j!} y_n^{j+1} \left[\frac{164046413\left(\frac{1}{10}\right)^j}{1197504000} - \frac{296725183\left(\frac{1}{10}\right)^j}{1596672000} + \frac{12051709\left(\frac{3}{10}\right)^j}{39916800} - \frac{33765029\left(\frac{2}{5}\right)^j}{88704000} + \frac{2227571\left(\frac{1}{2}\right)^j}{6237000} \right. \\
 & \left. - \frac{21677723}{88704000} h f_{n+\frac{3}{5}} + \frac{23643791}{199584000} h f_{n+\frac{7}{10}} - \frac{12318413}{319334400} h f_{n+\frac{4}{5}} + \frac{9071219}{1197504000} h f_{n+\frac{9}{10}} - \frac{3250433}{4790016000} h f_{n+1} \right] \\
 & \sum_{j=0}^{\infty} \frac{\left(\frac{1}{5}\right)^j}{j!} - y_n - \sum_{j=0}^{\infty} \frac{h^{j+1}}{j!} y_n^{j+1} \left[\frac{645431\left(\frac{1}{10}\right)^j}{3742200} - \frac{2149811\left(\frac{1}{10}\right)^j}{24948000} + \frac{355583\left(\frac{3}{10}\right)^j}{1559250} - \frac{1258463\left(\frac{2}{5}\right)^j}{4158000} + \frac{904403\left(\frac{1}{2}\right)^j}{3118500} \right. \\
 & \left. - \frac{166931}{831600} h f_{n+\frac{3}{5}} + \frac{21833}{222750} h f_{n+\frac{7}{10}} - \frac{800243}{24948000} h f_{n+\frac{4}{5}} + \frac{118291}{18711000} h f_{n+\frac{9}{10}} - \frac{8501}{14968800} h f_{n+1} \right] \\
 & \sum_{j=0}^{\infty} \frac{\left(\frac{3}{10}\right)^j}{j!} - y_n - \sum_{j=0}^{\infty} \frac{h^{j+1}}{j!} y_n^{j+1} \left[\frac{840607\left(\frac{1}{10}\right)^j}{4928000} - \frac{879183\left(\frac{1}{10}\right)^j}{19712000} + \frac{762497\left(\frac{3}{10}\right)^j}{2464000} - \frac{670233\left(\frac{2}{5}\right)^j}{1971200} + \frac{24399\left(\frac{1}{2}\right)^j}{77000} \right. \\
 & \left. - \frac{2136259}{9856000} h f_{n+\frac{3}{5}} + \frac{259071}{2464000} h f_{n+\frac{7}{10}} - \frac{675441}{19712000} h f_{n+\frac{4}{5}} + \frac{6637}{985600} h f_{n+\frac{9}{10}} - \frac{11899}{19712000} h f_{n+1} \right] \\
 & \sum_{j=0}^{\infty} \frac{\left(\frac{2}{5}\right)^j}{j!} - y_n - \sum_{j=0}^{\infty} \frac{h^{j+1}}{j!} y_n^{j+1} \left[\frac{400138\left(\frac{1}{10}\right)^j}{2338875} - \frac{2159\left(\frac{1}{5}\right)^j}{44550} + \frac{39752\left(\frac{3}{10}\right)^j}{111375} - \frac{23423\left(\frac{2}{5}\right)^j}{86625} + \frac{230788\left(\frac{1}{2}\right)^j}{779625} \right. \\
 & \left. - \frac{17879}{86625} h f_{n+\frac{3}{5}} + \frac{2248}{22275} h f_{n+\frac{7}{10}} - \frac{25754}{779625} h f_{n+\frac{4}{5}} + \frac{15226}{2338875} h f_{n+\frac{9}{10}} - \frac{547}{935550} h f_{n+1} \right] \\
 & \sum_{j=0}^{\infty} \frac{\left(\frac{1}{2}\right)^j}{j!} - y_n - \sum_{j=0}^{\infty} \frac{h^{j+1}}{j!} y_n^{j+1} \left[\frac{1636625\left(\frac{1}{10}\right)^j}{9580032} - \frac{599275\left(\frac{1}{10}\right)^j}{12773376} + \frac{558725\left(\frac{3}{10}\right)^j}{1596672} - \frac{461275\left(\frac{2}{5}\right)^j}{2128896} + \frac{17807\left(\frac{1}{2}\right)^j}{49896} \right. \\
 & \left. - \frac{465125}{2128896} h f_{n+\frac{3}{5}} + \frac{167675}{1596672} h f_{n+\frac{7}{10}} - \frac{62275}{1824768} h f_{n+\frac{4}{5}} + \frac{64175}{9580032} h f_{n+\frac{9}{10}} - \frac{22997}{38320128} h f_{n+1} \right] \\
 & \sum_{j=0}^{\infty} \frac{\left(\frac{3}{5}\right)^j}{j!} - y_n - \sum_{j=0}^{\infty} \frac{h^{j+1}}{j!} y_n^{j+1} \left[\frac{13169\left(\frac{1}{10}\right)^j}{77000} - \frac{14787\left(\frac{1}{5}\right)^j}{308000} + \frac{1363\left(\frac{3}{10}\right)^j}{3850} - \frac{35229\left(\frac{2}{5}\right)^j}{154000} + \frac{16083\left(\frac{1}{2}\right)^j}{38500} \right. \\
 & \left. - \frac{25373\left(\frac{3}{5}\right)^j}{154000} + \frac{1887\left(\frac{7}{10}\right)^j}{19250} - \frac{2007\left(\frac{4}{5}\right)^j}{61600} + \frac{71\left(\frac{9}{10}\right)^j}{11000} - \frac{179}{308000} (1)^j \right] \\
 & \sum_{j=0}^{\infty} \frac{\left(\frac{7}{10}\right)^j}{j!} - y_n - \sum_{j=0}^{\infty} \frac{h^{j+1}}{j!} y_n^{j+1} \left[\frac{5843887\left(\frac{1}{10}\right)^j}{34214400} - \frac{10669897\left(\frac{1}{10}\right)^j}{228096000} + \frac{9973607\left(\frac{3}{10}\right)^j}{28512000} - \frac{2767667\left(\frac{2}{5}\right)^j}{12672000} + \frac{353633\left(\frac{1}{2}\right)^j}{891000} \right. \\
 & \left. - \frac{241129\left(\frac{3}{5}\right)^j}{2534400} + \frac{4148249\left(\frac{7}{10}\right)^j}{28512000} - \frac{8312311\left(\frac{4}{5}\right)^j}{228096000} + \frac{1190357\left(\frac{9}{10}\right)^j}{171072000} - \frac{84427}{136857600} (1)^j \right] \\
 & \sum_{j=0}^{\infty} \frac{\left(\frac{4}{5}\right)^j}{j!} - y_n - \sum_{j=0}^{\infty} \frac{h^{j+1}}{j!} y_n^{j+1} \left[\frac{400448\left(\frac{1}{10}\right)^j}{2338875} - \frac{38176\left(\frac{1}{5}\right)^j}{779625} + \frac{278272\left(\frac{3}{10}\right)^j}{779625} - \frac{12184\left(\frac{2}{5}\right)^j}{51975} + \frac{330368\left(\frac{1}{2}\right)^j}{779625} \right. \\
 & \left. - \frac{34432\left(\frac{3}{5}\right)^j}{259875} + \frac{176896\left(\frac{7}{10}\right)^j}{779625} - \frac{3998\left(\frac{4}{5}\right)^j}{779625} + \frac{2368\left(\frac{9}{10}\right)^j}{467775} - \frac{1184}{2338875} (1)^j \right] \\
 & \sum_{j=0}^{\infty} \frac{\left(\frac{9}{10}\right)^j}{j!} - y_n - \sum_{j=0}^{\infty} \frac{h^{j+1}}{j!} y_n^{j+1} \left[\frac{837567\left(\frac{1}{10}\right)^j}{4928000} - \frac{167427\left(\frac{1}{10}\right)^j}{3942400} + \frac{829089\left(\frac{3}{10}\right)^j}{2464000} - \frac{1880253\left(\frac{2}{5}\right)^j}{9856000} + \frac{27459\left(\frac{1}{2}\right)^j}{77000} \right. \\
 & \left. - \frac{537219\left(\frac{3}{5}\right)^j}{9856000} + \frac{10773\left(\frac{7}{10}\right)^j}{70400} - \frac{2065743\left(\frac{4}{5}\right)^j}{19712000} + \frac{199809\left(\frac{9}{10}\right)^j}{4928000} - \frac{4671}{3942400} (1)^j \right] \\
 & \sum_{j=0}^{\infty} \frac{(1)^j}{j!} - y_n - \sum_{j=0}^{\infty} \frac{h^{j+1}}{j!} y_n^{j+1} \left[\frac{26575\left(\frac{1}{10}\right)^j}{149688} - \frac{16175\left(\frac{1}{5}\right)^j}{199584} + \frac{5675\left(\frac{3}{10}\right)^j}{12474} - \frac{4825\left(\frac{2}{5}\right)^j}{11088} + \frac{17807\left(\frac{1}{2}\right)^j}{24948} \right. \\
 & \left. - \frac{4825\left(\frac{3}{5}\right)^j}{11088} + \frac{5675\left(\frac{7}{10}\right)^j}{12474} + \frac{16175\left(\frac{4}{5}\right)^j}{199584} + \frac{26575\left(\frac{9}{10}\right)^j}{149688} + \frac{16067}{598752} (1)^j \right]
 \end{aligned} \right\} = 0 \quad (3.1)$$

Thus, $\bar{C}_0 = 0, \bar{C}_1 = 0, \bar{C}_2 = 0, \bar{C}_3 = 0, \dots = 0, \bar{C}_p = 0$; implying that the order of NBM is 11. That is, NBM is of order eleven, with the error constant given as

$$C_{12} = \begin{bmatrix} 1.2343 \times 10^{-14} & 1.7861 \times 10^{-14} & 1.8763 \times 10^{-14} & 1.8821 \times 10^{-14} & 1.2301 \times 10^{-14} \\ 1.2226 \times 10^{-14} & 1.0752 \times 10^{-14} & 1.9328 \times 10^{-14} & 1.5213 \times 10^{-14} & 1.4439 \times 10^{-14} \end{bmatrix}^T$$

Consistency of NBM

According to numerical analyst, NBM is consistent, since the order is greater than one (Kwanamu, 2023).

Zero Stability of NBM

According to Kwanamu, (2023), the NBM is said to be zero-stable if as $h \rightarrow 0$, the root $z_j, j = 1(1)k$ of the first characteristic polynomial $\rho(z) = 0$ that is

$$\rho(z) = \det \left[\sum_{j=0}^k A^{(j)} z^{k-j} \right] = 0 \quad (3.2)$$

satisfies $|z_j| \leq 1$ and for those roots with $|z_j| = 1$, multiplicity must not exceed two. The NBM is expressed in the form

$$\rho(z) = z \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix} - \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix} = z^9(z-1) \quad (3.3)$$

Thus, solving for z in

$$\rho(z) = z^9(z-1) = 0 \quad (3.4)$$

Gives $z_1 = 0, z_2 = 0, z_3 = 0, z_4 = 0, z_5 = 0, z_6 = 0, z_7 = 0, z_8 = 0, z_9 = 0$ and $z_{10} = 1$

Therefore, NBM is zero-stable.

Convergence of NBM

Based on Dahlquist's Theorem which state that, the requisite and complete prerequisites for a NBM to achieve convergence entail both consistency and zero-stability (Yakubu & Markus, 2016).

Therefore, the NBMis convergence since it is consistency and zero-stability.

Absolute Stability Region of NBM

The absolute stability region discrib the region of complex z -plane. The boundary locus method was used on the NBM using Udo&Awoyemi, (2017) to obtain the stability polynomial as

$$\bar{h}(w) = h^{10} \left(\frac{12753803513931}{744943288200000} w^9 - \frac{58777}{4204890} w^{10} \right) - h^9 \left(- \frac{102184206471587}{633719116000000000000} w^9 + \frac{183517}{4752} w^{10} \right) + \left. \begin{aligned} & h^8 \left(- \frac{124078709442223}{405580234520000000000} w^9 + \frac{66528}{1181437} w^{10} \right) + h^7 \left(\frac{1338797113347}{14696043107843840000} w^9 - \frac{1303}{3214155168} w^{10} \right) + \\ & h^6 \left(- \frac{267567585251287}{9797362069562560000} w^9 + \frac{502211745}{4523} w^{10} \right) + h^5 \left(\frac{2174780359142223}{1216740702560000000000} w^9 - \frac{985}{5629856} w^{10} \right) + \\ & h^4 \left(- \frac{15542282539737}{5431878144000000000000} w^9 - \frac{986}{74640} w^{10} \right) + h^3 \left(\frac{16180995779906749}{391095226380000000000} w^9 - \frac{867}{90875} w^{10} \right) + \\ & h^2 \left(\frac{1441155772}{189684633000000000000} w^9 + \frac{332}{8524} w^{10} \right) + h \left(\frac{2400000}{10886400} w^9 - \frac{1}{8} w^{10} \right) - w^8 + w^9 \end{aligned} \right\} \quad (3.5)$$

The stability polynomial (3.5) is used to plot the absolute stability region as

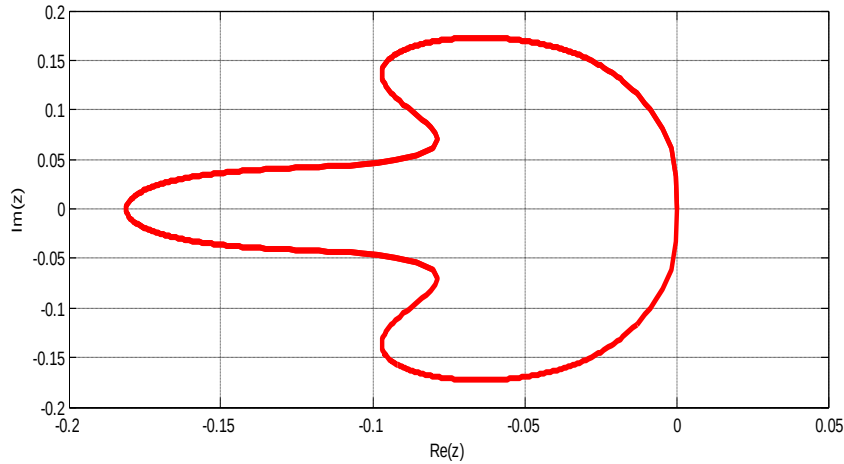


Figure 3.3: An A_α – Stability region of NBM.

Numerical Experiments

The NBM was numerically simulated on several epidemiological modelled differential equations of the form (1.1) as outlined below. The following notations shall be used in Tables and figure below.

t means Point of Evaluation

OA16 means Error in Omar and Adeyeye, (2016)

SOA13 means Error in Sunday, *et al.* (2013)

SYA16 means Error in Sunday et al. (2016)

Problem 4.1 (SIR Model):

The susceptible-infected-recovered (SIR) model tracks the progression of contagious diseases through its measurement of infected individuals in a population across different time points. The models are based on coupled equations which define three different population groups: $S(t)$ shows the number of people who still have not caught the disease, $I(t)$ displays the current count of infected cases and $R(t)$ shows the total number of people who have recovered from the illness. The model is coupled as:

$$\frac{dS}{dt} = \mu - \beta SI - \mu S \quad (4.1)$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I \quad (4.2)$$

$$\frac{dR}{dt} = \gamma I - \mu R \quad (4.3)$$

for μ, γ and β are positive parameters. y is given as,

$$y = S + I + R \quad (4.4)$$

Summing (4.1), (4.2) and (4.3) to get

$$y' = \mu(1 - y) \quad (4.5)$$

let $\mu = 0.5$ with initial condition as $y(0) = -0.5$, we get,

$$\frac{dy}{dt} = \frac{1}{2}(1-y), y(0) = \frac{1}{2}, h = 0.1 \quad (4.6)$$

with exact solution:

$$y(t) = 1 - \frac{1}{2}e^{-t} \quad (4.7)$$

Source: [Omar and Adeyeye, (2016) and Sunday *et al.* (2013)]

Problem 4.2 (Mixture Model)

The study observed a storage tank in the oil industry which 2000gal a certain amount of gasoline that originally has 100lb of an additive dissolved in it. When preparing for winter, gasoline containing 2lb of additive per gallon, which is pumped into the tank at a rate of 140gal/min. The well-mixed solution is pumped out at a rate of 45gal/min. Now, by numerical application, calculate the additive in the tank 0.1 min, 0.5 min and 1 min after the pumping process begins? operation starts.

We assumed y to be the amount (in pounds) of additive in the tank at time t . Taking $y = 100$, when the time $t = 0$. Therefore, after modeling the problem we have,

$$\frac{dy}{dt} = 80 - \frac{45}{(2000-5t)}y, y(0) = 100 \quad (4.8)$$

with the exact solution

$$2(2000-5t) - \frac{3900}{2000} \left(\frac{2000}{5t} \right) \quad (4.9)$$

Source: [Omar and Adeyeye, (2016) and Sunday *et al.* (2013)]

Problem 4.3 (Logistic Model)

The logistic model which extends the growth model functions as a law which accurately predicts population growth rates based on time. The model is established on Assuming the population grows within a confined environment where resources are limited and there is no migration in or out, the change in population over time can be described mathematically. Let $x(t)$ be the population size at time t be represented as x , which allows us to express its growth pattern through differential equation.

$$\frac{dx}{dT} = rx \left(1 - \frac{x}{k} \right) \quad (4.10)$$

Where $k > 0$ is the carrying capacity of the system/environment, $r > 0$ is a parameter called intrinsic growth rate ($r = b - d$, where b is the instantaneous birth rate and d the instantaneous death rate).

We will begin the process of non-dimensionalization through the scaling method for equation (4.10). The model in equation (4.10) needs its four parameters to be reduced through the application of an appropriate scaling method. We will establish the transformation through the following definition:

$$T = \frac{t}{r} \text{ and } x - ky \quad (4.11)$$

Substitute equation (4.11) in (4.10), we get

$$\frac{d(ky)}{d\frac{t}{r}} = rky \left(1 - \frac{ky}{k} \right) \quad (4.12)$$

Which reduce to

$$\frac{dy}{dt} = y(1 - y) \quad (4.13)$$

Equation (4.13) is called the logistic model. The equation can be solved by the method of separation of variable to give the exact solution,

$$y(t) = \frac{y_0}{y_0 + (1 - y_0)} \quad (4.14)$$

where $y(t_0) = y_0$.

It is significant to state that this equation possess a very simple asymptotic dynamics, all solutions with positive initial condition ($y_0 > 0$) will eventually approach the carrying capacity k .

Therefore, population size will eventually be stabilized to k in the long run if population dynamics initially either overshoot or undershoot the carrying capacity.

Here, we shall consider a special case of equation (4.14) given by substituting $r = k = 1$ in Equation (4.12). This gives,

$$\frac{dy}{dt} = y(1 - y), y(0) = 0.5 \quad (4.15)$$

With theoretical solution,

$$y(t) = \frac{0.5}{0.5(1 + e^{-t})} \quad (4.16)$$

Source: [Sunday et al. 2016].

Table 4.1: Showing the results of Problem 4.1

x	Exact Solution	Computed Solution	ENBM	EOA16	ESOA13
0.1	0.52438528774964299546	0.52438528774964299541	5.0000E-20	4.9562E-06	5.5744E-12
0.2	0.54758129098202021342	0.54758129098202021330	1.2000E-19	4.7260E-06	3.9462E-12
0.3	0.56964601178747109638	0.56964601178747109621	1.7000E-19	8.9799E-06	8.1832E-12
0.4	0.59063462346100907066	0.59063462346100907046	2.0000E-19	8.5524E-06	3.4361E-11
0.5	0.61059960846429756588	0.61059960846429756563	2.5000E-19	1.2193E-05	1.9294E-10
0.6	0.62959088965914106696	0.62959088965914106666	3.0000E-19	1.1608E-05	1.8790E-10
0.7	0.64765595514064328282	0.64765595514064328247	3.5000E-19	1.4713E-05	1.7768E-10
0.8	0.66483997698218034963	0.66483997698218034925	3.8000E-19	1.4004E-05	1.7247E-10
0.9	0.68118592418911335343	0.68118592418911335301	4.2000E-19	1.6643E-05	1.8476E-10
1.0	0.69673467014368328820	0.69673467014368328774	4.6000E-19	1.5839E-05	3.0058E-10

Table 4.2: Showing the results of Problem 4.2

x	Exact Solution	Computed Solution	ENBM	EOA16	ESOA13
0.1	107.7662301168309486000	107.76623011683094854000	1.0000E-17	2.5540E-06	2.7001E-13
0.2	115.5149409193028511000	115.51494091930285114800	1.0000E-17	2.5490E-06	1.2790E-13
0.3	123.2461630508845220000	123.24616305088452201000	1.0000E-17	5.0900E-06	1.4211E-13
0.4	130.9599271090910725000	130.95992710909107258000	4.0000E-17	5.0790E-06	4.2633E-13
0.5	138.6562636455413535000	138.65626364554135354000	2.0000E-17	7.6070E-06	1.1367E-13
0.6	146.3352031660153396000	146.33520316601533959000	2.0000E-17	7.5900E-06	1.7053E-13
0.7	153.9967761305114566000	153.99677613051145661000	1.0000E-17	1.0100E-05	8.5265E-14
0.8	161.6410129533038516000	161.64101295330385157700	1.0000E-17	1.0080E-05	8.5265E-14
0.9	169.2679440029996050000	169.26794400299960502000	1.0000E-17	1.2580E-05	8.5265E-14
1.0	176.8775996025958864000	176.87759960259588644000	4.0000E-17	1.2560E-05	2.2737E-13

Table 4.3: Showing the results of Problem 4.3

x	Exact Solution	Computed Solution	ENBM	SYA16
0.1	0.52497918747893998610	0.52497918747893998612	2.0000E-20	1.1102E-16
0.2	0.54983399731247790855	0.54983399731247790860	5.0000E-20	1.1102E-16
0.3	0.57444251681165898715	0.57444251681165898722	7.0000E-20	4.4489E-16
0.4	0.59868766011245200035	0.59868766011245200045	1.0000E-20	7.7716E-16
0.5	0.62245933120185456465	0.62245933120185456474	9.0000E-20	1.2213E-15
0.6	0.64565630622579545290	0.64565630622579545298	8.0000E-20	1.5543E-15
0.7	0.66818777216816610655	0.66818777216816610660	5.0000E-20	2.1094E-15
0.8	0.68997448112761244265	0.68997448112761244270	5.0000E-20	2.6645E-15
0.9	0.71094950262500396345	0.71094950262500396350	5.0000E-20	3.2197E-15
1.0	0.73105857863000487925	0.73105857863000487933	8.0000E-20	3.7748E-15

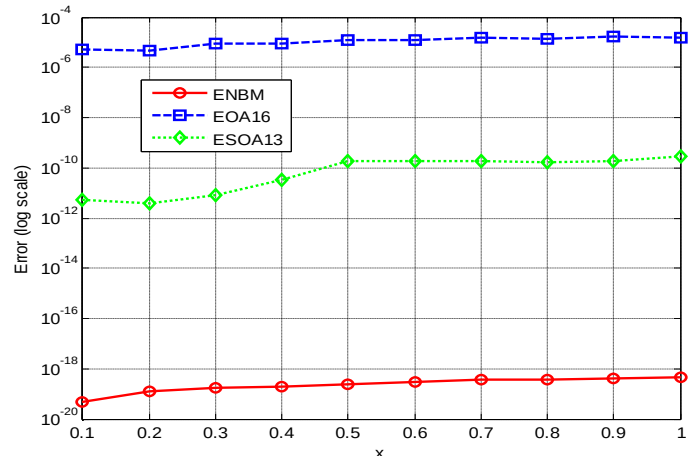


Figure 4.1: Textual curve of table 4.1

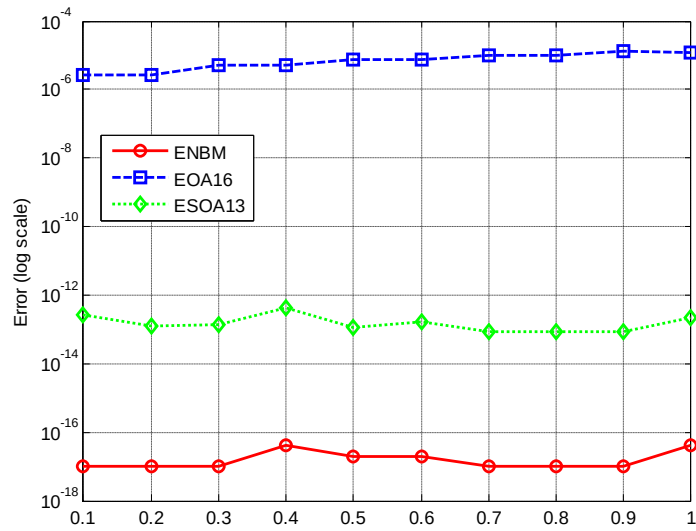


Figure 4.2: Textual curve of table 4.2

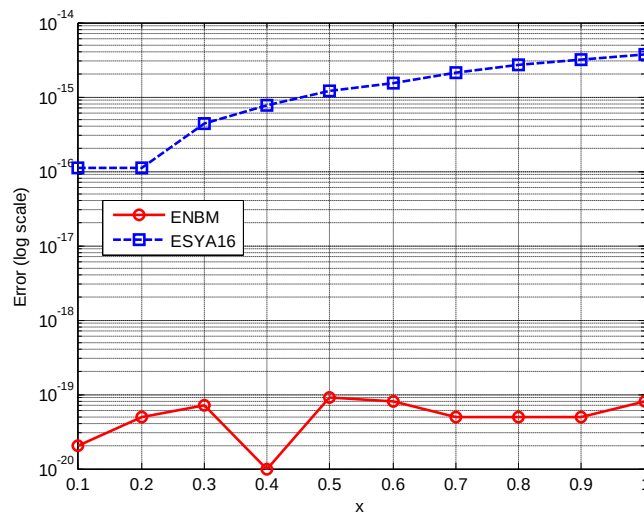


Figure 4.3: Textual curve of table 4.3

Discussion of Results

The numerical results of the Nonlinear Block Method (NBM) applied to the SIR model show an excellent agreement between the computed and exact solutions across all evaluation points. As presented in Table 4.1, the computed values closely follow the analytical solutions with no visible deviation at the displayed precision level. This indicates that the method is highly reliable for capturing the dynamics of infectious disease spread governed by the SIR system. The corresponding Figure 4.1 further illustrates this strong agreement, as both curves essentially overlap, confirming the accuracy and stability of the proposed method.

For the mixture model in Problem 4.2, the results in Table 4.2 also show a strong correspondence between the computed and exact solutions over the entire time interval considered. The numerical values increase steadily with time, reflecting the continuous accumulation of additive in the tank as dictated by the inflow and outflow rates. The NBM successfully reproduces this physical behaviour without noticeable deviation from the analytical solution. Figure 4.2 similarly shows that the computed curve follows the exact trajectory very closely, indicating that the method is well-suited for solving first-order linear transport and mixing problems in engineering applications.

In Problem 4.3, the logistic growth model results in Table 4.3 demonstrate that the computed solutions match the exact solutions throughout the evaluation points. The numerical results capture the characteristic sigmoidal growth pattern, where the solution increases rapidly at early times and gradually levels off as it approaches the carrying capacity. This behaviour is also clearly reflected in Figure 4.3, where the computed and exact curves are indistinguishable. The agreement confirms that the NBM accurately preserves the nonlinear dynamics inherent in population growth models.

The results across the three test problems demonstrate that the NBM is highly accurate and consistently reliable for solving nonlinear and linear differential equations arising in epidemiology and population dynamics. The close agreement between computed and exact solutions, as well as the overlapping nature of the graphical results, confirms the effectiveness of the method in capturing both transient and long-term system behaviour. This establishes the NBM as a robust computational technique for real-world mathematical modelling problems.

Summary and Conclusion

This study presents the development of a New Block Method (NBM) for solving first-order ordinary differential equations arising from real-life and epidemiological models. The method was constructed using a power series polynomial framework combined with interpolation and collocation techniques to generate a high-order implicit block scheme. It was applied to three benchmark problems: the SIR epidemiological model, a mixture (tank) model, and the logistic population growth model. These applications demonstrate the method's capability in handling both linear and nonlinear problems without reducing them to first-order systems.

The numerical experiments show that the NBM produces solutions that are in very close agreement with exact analytical solutions across all tested models. The results confirm that the method accurately captures the dynamic behaviour of each system, including disease transmission patterns in the SIR model, concentration variation in the tank model, and sigmoidal growth in the logistic model. Graphical and computational comparisons further indicate that the method performs with high precision and maintains the essential qualitative features of the models.

The study establishes that the New Block Method is stable, consistent, and convergent. It demonstrates strong reliability in solving initial value problems with high accuracy and computational efficiency. The method therefore serves as a robust

alternative to existing numerical techniques and is recommended for extension to more complex nonlinear systems and advanced epidemiological models requiring precise numerical approximation.

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Computing Numerical Scheme for Approximate Solutions of Second-Order Oscillatory Initial Value Problems

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Abstract

This study presents a Computational Numerical Scheme (CNS) for the direct solution of second-order oscillatory initial value problems without reducing them to systems of first-order differential equations. The motivation for this approach stems from the need to improve computational efficiency, reduce numerical complexity, and preserve the inherent structure of oscillatory physical models such as Bessel-type equations and mass-spring systems. The CNS was derived using a power series approximation combined with interpolation and collocation techniques, leading to a continuous hybrid linear multistep formulation. The method was analyzed for consistency, order, zero-stability, convergence, and absolute stability, where it was shown to be A-stable. The CNS was applied to three test problems, and its performance was evaluated through comparisons of absolute errors with existing methods in the literature. Numerical results demonstrated that the CNS consistently produced highly accurate approximations with significantly reduced errors, outperforming competing methods. Graphical illustrations further confirmed the superiority and stability of the method across all tested intervals. The findings indicate that the CNS is an efficient, reliable, and accurate approach for solving second-order oscillatory differential equations arising in real-life physical systems.

Keywords: Computational Numerical Scheme (CNS); second-order differential equations; oscillatory systems; initial value problems; stability analysis; mass-spring system; Bessel equation; numerical simulation.

Introduction

Scientists and engineers use second-order oscillatory problems to study mechanical vibrations, electrical circuits, quantum mechanics, and wave propagation phenomena. The solutions to these problems are described by second-order ordinary differential equations that produce oscillatory patterns throughout the solution process. Traditional numerical methods for solving such equations often convert them into systems of first-order equations, which leads to higher computational costs and reduced efficiency. Consequently, researchers now focus on developing numerical methods that allow the direct computation of second-order equations, as this approach preserves the structure of the original problem and improves solution accuracy (Omoile and Ogunware, 2018; Lydia *et al.* 2021).

Computational numerical methods for solving second-order oscillatory problems directly exploit the inherent oscillatory behavior of the solution. These methods include direct integration schemes, trigonometrically fitted methods, and block methods, all of which are designed to enhance accuracy, stability, and efficiency by minimizing phase-

lag and numerical damping errors. By solving periodic problems directly, these approaches eliminate the need to transform the equations into systems of first-order equations, thereby reducing computational cost. As a result, they have become essential tools for researchers dealing with practical oscillatory models that require both high accuracy and fast computational performance (Adewale and Sabo, 2024).

Mathematical modeling has become a powerful tool in recent years for advancing our understanding of processes in biology, physics, and medicine, particularly in areas such as dynamical systems, body cooling, and simple harmonic motion. The mathematical models developed by Sabo *et al.* (2021) and Elazzouzi *et al.* (2019) have proven effective in supporting both mathematical research and advances in the biosciences. The application of mathematics in these fields has led to the development of new methods that promote interdisciplinary collaboration among scientists. This study further demonstrates how mathematical techniques enhance understanding and improve the accuracy of predictions in dynamical systems and thermal process investigations (Kubuye and Omar, 2015a).

The numerical solution of higher-order ordinary differential equations is typically achieved by converting the equations into first-order differential equation systems which require a suitable numerical method designed for first-order equations. The method calculates solutions by processing one point at a time to complete the solution. The method suffers from multiple problems because it requires extensive computational resources, it has complicated execution requirements, its accuracy suffers from error problems, the method needs complex programming work, and it consumes excessive time (Kubuye and Omar, 2015b).

This research examines direct solutions for initial value problems which involve second-order ordinary differential equations of the form

$$\gamma''(t) = f(t, \gamma, \gamma'), \gamma(t_0) = \gamma_0, \gamma'(t_0) = \gamma'_0, \quad t \in [t_0, t_n] \quad (1.1)$$

where t_0 represents initial value/point, γ_0 denotes the solution at time t_0 , f remains continuous over the integration interval. We operate under the assumption that equation (1.1) adheres to the existence and uniqueness theorem of differential equations. Furthermore, we presume that solutions to equations akin to (1.1) remain bounded. It is crucial to clarify that a solution $\gamma(t)$ to equation (1.1) is deemed bounded if,

$$\sup_{t \in \mathbb{R}} \|\gamma(t)\| < \infty \quad (1.2)$$

Researchers have dedicated significant efforts to developing numerical solutions for equations of the form (1.1), as reported in studies by Kubuye and Omar (2015b), Adeyeye and Omar (2017), and Skwame *et al.* (2019).

The development of numerical methods for solving second-order ordinary differential equations has attracted considerable attention in recent years. Various researchers have made significant contributions to this field through the formulation of improved computational techniques, among others are Adeniran and Ogundare (2015), Kwanamu *et al.* (2021), Donald *et al.* (2021), Ayinde *et al.* (2023), and Ishaq *et al.* (2024) all contributed to advancing solution strategies for differential equations, particularly in enhancing efficiency and accuracy.

Ibrahim (2017) introduced a hybrid multistep method for solving second-order initial value problems, which avoids the transformation of such problems into systems of first-order equations. The study demonstrated that the incorporation of hybridization and off-step points improves the accuracy and stability of the method.

Similarly, Kamo *et al.* (2018) developed a block method with continuous coefficients based on shifted Legendre polynomials. This approach provided a simple and

efficient framework for solving second-order ordinary differential equations by expressing the solution in continuous functional form.

In addition, Raymond *et al.* (2018) developed an implicit two-step hybrid block method that utilizes off-grid points and incorporates third-derivative information. Their method showed improved performance in handling both stiff and oscillatory problems, achieving higher accuracy through the use of higher-order derivative information.

Current research efforts focus on addressing the limitations of existing numerical methods for solving oscillatory differential equations, particularly the wastage of human efforts, time constraints and the computational burden that affect the accuracy of the method in terms of errors.

To overcome these challenges, Sabo *et al.* (2024) introduced a two-step block method specifically designed for oscillatory systems. Their study demonstrated that the method improves accuracy while reducing computational requirements, although its performance may still depend on step-size selection in highly stiff or complex oscillatory cases. Similarly, Adewale and Sabo (2024) showed that while classical approaches may struggle with long-term simulation of oscillatory motion in mass-spring systems, direct computational techniques provide better stability and efficiency in modelling dynamic behaviour.

Furthermore, Aloko *et al.* (2024) developed a direct method for solving third and fourth-order oscillatory differential equations. Although the method improves computational efficiency and reduces transformation errors associated with converting higher-order problems into first-order systems, challenges may still arise in handling highly nonlinear or strongly stiff oscillations.

Derivation of Computational Numerical Scheme (CNS)

The computational numerical scheme (CNS) is derived in this section, where an approximate solution is expressed in the form of a power series polynomial as

$$\gamma(t) = \sum_{j=0}^{\varphi} \alpha_j t^j \quad (2.1)$$

is considered as the basis function for the direct solution of the second initial value problems of the form (1.1). Where $t \in [a, b]$, the α 's are real unknown parameters to be determined, $\varphi = \omega + \varpi - 1$ and $\omega + \varpi$ is the sum of the number of interpolation and collocation points.

Differentiate equation (2.1) two times to obtain

$$\gamma''(t) = \sum_{j=0}^{\varphi} j(j-1)\alpha_j t^{j-2} \quad (2.2)$$

Now, interpolating (2.1) at point $u = \frac{1}{2}, 1$ and collocating (2.2) at $v = 0, \frac{1}{6}, \frac{1}{4}, \frac{1}{3}, \frac{1}{2}, \frac{2}{3}, \frac{3}{4}, 1$

which lead to a system of equation in a matrix form as

$$\begin{bmatrix} 1 & t_{n+\frac{1}{2}} & t_{n+\frac{1}{2}}^2 & t_{n+\frac{1}{2}}^3 & t_{n+\frac{1}{2}}^4 & t_{n+\frac{1}{2}}^5 & t_{n+\frac{1}{2}}^6 & t_{n+\frac{1}{2}}^7 & t_{n+\frac{1}{2}}^8 & t_{n+\frac{1}{2}}^9 \\ 1 & t_{n+1} & t_{n+1}^2 & t_{n+1}^3 & t_{n+1}^4 & t_{n+1}^5 & t_{n+1}^6 & t_{n+1}^7 & t_{n+1}^8 & t_{n+1}^9 \\ 0 & 0 & 2 & 6t_n & 12t_n^2 & 20t_n^3 & 30t_n^4 & 42t_n^5 & 56t_n^6 & 72t_n^7 \\ 0 & 0 & 2 & 6t_{n+\frac{1}{6}} & 12t_{n+\frac{1}{6}}^2 & 20t_{n+\frac{1}{6}}^3 & 30t_{n+\frac{1}{6}}^4 & 42t_{n+\frac{1}{6}}^5 & 56t_{n+\frac{1}{6}}^6 & 72t_{n+\frac{1}{6}}^7 \\ 0 & 0 & 2 & 6t_{n+\frac{1}{4}} & 12t_{n+\frac{1}{4}}^2 & 20t_{n+\frac{1}{4}}^3 & 30t_{n+\frac{1}{4}}^4 & 42t_{n+\frac{1}{4}}^5 & 56t_{n+\frac{1}{4}}^6 & 72t_{n+\frac{1}{4}}^7 \\ 0 & 0 & 2 & 6t_{n+\frac{1}{3}} & 12t_{n+\frac{1}{3}}^2 & 20t_{n+\frac{1}{3}}^3 & 30t_{n+\frac{1}{3}}^4 & 42t_{n+\frac{1}{3}}^5 & 56t_{n+\frac{1}{3}}^6 & 72t_{n+\frac{1}{3}}^7 \\ 0 & 0 & 2 & 6t_{n+\frac{1}{2}} & 12t_{n+\frac{1}{2}}^2 & 20t_{n+\frac{1}{2}}^3 & 30t_{n+\frac{1}{2}}^4 & 42t_{n+\frac{1}{2}}^5 & 56t_{n+\frac{1}{2}}^6 & 72t_{n+\frac{1}{2}}^7 \\ 0 & 0 & 2 & 6t_{n+\frac{2}{3}} & 12t_{n+\frac{2}{3}}^2 & 20t_{n+\frac{2}{3}}^3 & 30t_{n+\frac{2}{3}}^4 & 42t_{n+\frac{2}{3}}^5 & 56t_{n+\frac{2}{3}}^6 & 72t_{n+\frac{2}{3}}^7 \\ 0 & 0 & 2 & 6t_{n+\frac{3}{4}} & 12t_{n+\frac{3}{4}}^2 & 20t_{n+\frac{3}{4}}^3 & 30t_{n+\frac{3}{4}}^4 & 42t_{n+\frac{3}{4}}^5 & 56t_{n+\frac{3}{4}}^6 & 72t_{n+\frac{3}{4}}^7 \\ 0 & 0 & 2 & 6t_{n+1} & 12t_{n+1}^2 & 20t_{n+1}^3 & 30t_{n+1}^4 & 42t_{n+1}^5 & 56t_{n+1}^6 & 72t_{n+1}^7 \end{bmatrix} \begin{bmatrix} a_0 \\ a_1 \\ a_2 \\ a_3 \\ a_4 \\ a_5 \\ a_6 \\ a_7 \\ a_8 \\ a_9 \end{bmatrix} = \begin{bmatrix} \gamma_{n+\frac{1}{2}} \\ \gamma_{n+2} \\ f_n \\ f_{n+\frac{1}{6}} \\ f_{n+\frac{1}{4}} \\ f_{n+\frac{1}{3}} \\ f_{n+\frac{1}{2}} \\ f_{n+\frac{2}{3}} \\ f_{n+\frac{3}{4}} \\ f_{n+1} \end{bmatrix} \quad (2.3)$$

using Gaussian elimination method on maple 2024.2 software package, (2.3) is solved for the a_j 's. The values of the a_j 's obtained are then substituted into (1.1), after some manipulations, this gives a continuous hybrid linear multistep method of the form;

$$\gamma(t) = \alpha_{\frac{1}{2}}(t)\gamma_{n+\frac{1}{2}} + \alpha_1(t)\gamma_{n+1} + h^2 \left[\sum_{j=0}^1 \beta_j(t)f_{n+j} + \beta_{v_i}(t)f_{n+v_i} \right], v_i = 0, \frac{1}{6}, \frac{1}{4}, \frac{1}{3}, \frac{1}{2}, \frac{2}{3}, \frac{3}{4} \quad (2.4)$$

the coefficient $\alpha_{\frac{1}{2}}, \alpha_1, \beta_0, \beta_{\frac{1}{6}}, \beta_{\frac{1}{4}}, \beta_{\frac{1}{3}}, \beta_{\frac{1}{2}}, \beta_{\frac{2}{3}}, \beta_{\frac{3}{4}}$ are given by;

$$\alpha_{\frac{1}{2}} = 2 - 2t$$

$$\alpha_1 = 2t - 1$$

$$\beta_0 = \frac{41}{13440} - \frac{1867}{40320}t + \frac{1}{2}t^2 - \frac{113}{36}t^3 + \frac{427}{36}t^4 - \frac{3367}{120}t^5 + \frac{1876}{45}t^6 - \frac{113}{3}t^7 + \frac{132}{7}t^8 - 4t^9$$

$$\beta_{\frac{1}{6}} = -\frac{54}{175}t + \frac{648}{35}t^3 - \frac{594}{5}t^4 + \frac{9072}{25}t^5 - \frac{3132}{5}t^6 + \frac{21816}{35}t^7 - \frac{11664}{35}t^8 + \frac{2592}{35}t^9$$

$$\beta_{\frac{1}{4}} = \frac{2}{21} + \frac{26}{175}t - \frac{512}{15}t^3 + \frac{11392}{45}t^4 - \frac{21248}{25}t^5 + \frac{14080}{9}t^6 - \frac{171008}{105}t^7 + \frac{31488}{35}t^8 - \frac{1024}{5}t^9$$

$$\beta_{\frac{1}{3}} = -\frac{243}{4480} - \frac{567}{3200}t + \frac{243}{10}t^3 - \frac{1539}{8}t^4 + \frac{138267}{200}t^5 - \frac{6723}{5}t^6 + \frac{51111}{35}t^7 - \frac{5832}{7}t^8 + \frac{972}{5}t^9$$

$$\beta_{\frac{1}{2}} = \frac{17}{105} - \frac{9}{35}t - 8t^3 + \frac{202}{3}t^4 - \frac{1304}{5}t^5 + \frac{8252}{15}t^6 - \frac{4504}{7}t^7 + \frac{2736}{7}t^8 - 96t^9$$

$$\beta_{\frac{2}{3}} = -\frac{243}{4480} - \frac{1647}{22400}t + \frac{81}{20}t^3 - \frac{351}{10}t^4 + \frac{28269}{200}t^5 - \frac{1566}{5}t^6 + \frac{13581}{35}t^7 - \frac{8748}{35}t^8 + \frac{324}{5}t^9$$

$$\beta_{\frac{3}{4}} = \frac{2}{21} - \frac{278}{1575}t - \frac{512}{315}t^3 + \frac{128}{9}t^4 - \frac{4352}{75}t^5 + \frac{5888}{45}t^6 - \frac{17408}{105}t^7 + \frac{768}{7}t^8 - \frac{1024}{35}t^9$$

$$\beta_1 = \frac{41}{13440} - \frac{143}{22400}t + \frac{1}{30}t^3 - \frac{107}{360}t^4 + \frac{249}{200}t^5 - \frac{131}{45}t^6 + \frac{407}{405}t^7 - \frac{96}{35}t^8 + \frac{4}{5}t^9$$

evaluating (2.4) at non interpolating points to obtain the continuous form as,

$$\begin{aligned}
 \gamma_n - 2\gamma_{n+\frac{1}{2}} - \gamma_{n+1} &= \frac{41}{13440} h^2 f_n + \frac{2}{21} h^2 f_{n+\frac{1}{4}} - \frac{243}{4480} h^2 f_{n+\frac{1}{2}} + \frac{17}{105} h^2 f_{n+\frac{3}{4}} - \frac{243}{4480} h^2 f_{n+1} + \frac{2}{21} f_{n+\frac{1}{2}} + \frac{41}{13440} f_{n+1} \\
 \gamma_{n+\frac{1}{6}} - \frac{5}{3} \gamma_{n+\frac{1}{2}} + \frac{2}{3} \gamma_{n+1} &= \frac{8851}{8817984} h^2 f_n + \frac{188}{8505} h^2 f_{n+\frac{1}{6}} + \frac{52522}{688905} h^2 f_{n+\frac{1}{4}} - \frac{4979}{90720} h^2 f_{n+\frac{1}{2}} + \frac{10117}{91854} h^2 f_{n+\frac{3}{4}} - \frac{20473}{544320} h^2 f_{n+1} + \frac{44146}{688905} h^2 f_{n+\frac{3}{4}} + \frac{22283}{11022480} h^2 f_{n+1} \\
 \gamma_{n+\frac{1}{4}} - \frac{3}{2} \gamma_{n+\frac{1}{2}} + \frac{1}{2} \gamma_{n+1} &= \frac{1749}{2293760} h^2 f_n - \frac{4941}{286720} h^2 f_{n+\frac{1}{4}} + \frac{359}{6720} h^2 f_{n+\frac{1}{2}} - \frac{106191}{2293760} h^2 f_{n+\frac{3}{4}} + \frac{23393}{286720} h^2 f_{n+1} - \frac{64233}{2293760} h^2 f_{n+\frac{3}{4}} + \frac{43}{896} h^2 f_{n+\frac{3}{4}} + \frac{10441}{6881280} h^2 f_{n+1} \\
 \gamma_{n+\frac{1}{3}} - \frac{4}{3} \gamma_{n+\frac{1}{2}} + \frac{1}{3} \gamma_{n+1} &= \frac{22819}{44089920} h^2 f_n - \frac{20}{1701} h^2 f_{n+\frac{1}{6}} + \frac{25052}{688905} h^2 f_{n+\frac{1}{4}} - \frac{6737}{181440} h^2 f_{n+\frac{1}{2}} + \frac{12182}{229635} h^2 f_{n+\frac{1}{2}} - \frac{10019}{544320} h^2 f_{n+\frac{3}{4}} + \frac{628}{19683} h^2 f_{n+\frac{3}{4}} + \frac{44659}{44089920} h^2 f_{n+1} \\
 \gamma_{n+\frac{2}{3}} - \frac{2}{3} \gamma_{n+\frac{1}{2}} - \frac{1}{3} \gamma_{n+1} &= -\frac{45149}{88179840} h^2 f_n + \frac{20}{1701} h^2 f_{n+\frac{1}{6}} - \frac{5098}{137781} h^2 f_{n+\frac{1}{4}} + \frac{2963}{72576} h^2 f_{n+\frac{1}{2}} - \frac{7211}{229635} h^2 f_{n+\frac{1}{2}} + \frac{24061}{1088640} h^2 f_{n+\frac{3}{4}} - \frac{22418}{688905} h^2 f_{n+\frac{3}{4}} - \frac{88829}{88179840} h^2 f_{n+1} \\
 \gamma_{n+\frac{3}{4}} - \frac{1}{2} \gamma_{n+\frac{1}{2}} - \frac{1}{2} \gamma_{n+1} &= -\frac{5179}{6881280} h^2 f_n + \frac{4941}{286720} h^2 f_{n+\frac{1}{4}} - \frac{727}{13440} h^2 f_{n+\frac{1}{2}} + \frac{136323}{2293760} h^2 f_{n+\frac{1}{2}} - \frac{37883}{860160} h^2 f_{n+\frac{1}{2}} + \frac{18873}{458752} h^2 f_{n+\frac{3}{4}} - \frac{109}{2240} h^2 f_{n+\frac{3}{4}} - \frac{10373}{6881280} h^2 f_{n+1}
 \end{aligned} \quad (2.5)$$

differentiating (2.4) once, yields

$$\gamma'(t) = \sigma_{\frac{1}{2}}(t) \gamma_{n+\frac{1}{2}} + \sigma_1(t) \gamma_{n+1} + h^2 \left[\sum_{j=0}^1 \beta'_j(t) f_{n+j} + \beta'_{v_i}(t) f_{n+v_i} \right], \quad v_i = 0, \frac{1}{6}, \frac{1}{4}, \frac{1}{3}, \frac{1}{2}, \frac{2}{3}, \frac{3}{4} \quad (2.6)$$

the coefficient $\sigma_{\frac{1}{2}}, \sigma_1, \beta'_0, \beta'_{\frac{1}{6}}, \beta'_{\frac{1}{4}}, \beta'_{\frac{1}{3}}, \beta'_{\frac{1}{2}}, \beta'_{\frac{2}{3}}, \beta'_{\frac{3}{4}}$ are given by;

$$\begin{aligned}
 \alpha'_{\frac{1}{2}} &= -2 \\
 \alpha'_1 &= 2 \\
 \beta'_0 &= -\frac{1867}{40320} + t - \frac{113}{12} t^2 + \frac{427}{9} t^3 - \frac{3367}{24} t^4 + \frac{3752}{15} t^5 - \frac{791}{3} t^6 + \frac{1056}{7} t^7 - 36t^8 \\
 \beta'_{\frac{1}{6}} &= -\frac{54}{175} + \frac{1944}{35} t^2 - \frac{2376}{5} t^3 + \frac{9072}{5} t^4 - \frac{18792}{5} t^5 + \frac{21816}{5} t^6 - \frac{93312}{35} t^7 + \frac{23328}{35} t^8 \\
 \beta'_{\frac{1}{4}} &= \frac{26}{175} - \frac{512}{5} t^2 + \frac{45568}{45} t^3 - \frac{21248}{5} t^4 + \frac{28160}{3} t^5 - \frac{171008}{15} t^6 + \frac{251904}{35} t^7 - \frac{9216}{5} t^8 \\
 \beta'_{\frac{1}{3}} &= -\frac{567}{3200} + \frac{729}{10} t^2 - \frac{1539}{2} t^3 + \frac{138267}{40} t^4 - \frac{40338}{5} t^5 + \frac{51111}{5} t^6 - \frac{46656}{7} t^7 + \frac{8748}{5} t^8 \\
 \beta'_{\frac{1}{2}} &= -\frac{9}{35} - 24t^2 + \frac{808}{3} t^3 - 1304t^4 + \frac{16504}{5} t^5 - 4504t^6 + \frac{21888}{7} t^7 - 864t^8 \\
 \beta'_{\frac{2}{3}} &= +\frac{1647}{22400} + \frac{243}{20} t^2 - \frac{702}{5} t^3 + \frac{28269}{40} t^4 - \frac{9396}{5} t^5 + \frac{13581}{5} t^6 - \frac{69984}{35} t^7 + \frac{2916}{5} t^8 \\
 \beta'_{\frac{3}{4}} &= -\frac{278}{1575} - \frac{512}{105} t^2 + \frac{512}{9} t^3 - \frac{4352}{15} t^4 + \frac{11776}{15} t^5 - \frac{17408}{15} t^6 + \frac{6144}{7} t^7 - \frac{9216}{35} t^8 \\
 \beta'_1 &= -\frac{143}{22400} + \frac{1}{10} t^2 - \frac{107}{90} t^3 + \frac{249}{40} t^4 - \frac{626}{15} t^5 + \frac{407}{15} t^6 - \frac{768}{35} t^7 + \frac{36}{5} t^8
 \end{aligned}$$

Evaluate equation (2.6) at all point, to yields the following discrete (CNS)as

$$\begin{aligned}
 h\gamma'_n + 2\gamma_{n+\frac{1}{2}} - 2\gamma_{n+1} &= -\frac{1867}{40320} hf_n - \frac{54}{175} hf_{n+\frac{1}{6}} + \frac{26}{175} hf_{n+\frac{1}{4}} - \frac{567}{3200} hf_{n+\frac{1}{3}} - \frac{9}{35} hf_{n+\frac{1}{2}} + \frac{1647}{22400} hf_{n+\frac{2}{3}} - \frac{278}{1575} hf_{n+\frac{3}{4}} - \frac{143}{22400} hf_{n+1} \\
 h\gamma'_{n+\frac{1}{6}} + 2\gamma_{n+\frac{1}{2}} - 2\gamma_{n+1} &= -\frac{13609}{4898880} hf_n + \frac{167}{4725} hf_{n+\frac{1}{6}} - \frac{114598}{382725} hf_{n+\frac{1}{4}} + \frac{5611}{50400} hf_{n+\frac{1}{3}} - \frac{8789}{25515} hf_{n+\frac{1}{2}} - \frac{35107}{302400} hf_{n+\frac{2}{3}} - \frac{74014}{382725} hf_{n+\frac{3}{4}} - \frac{18511}{3061800} hf_{n+1} \\
 h\gamma'_{n+\frac{1}{4}} + 2\gamma_{n+\frac{1}{2}} - 2\gamma_{n+1} &= -\frac{1711}{573440} hf_n + \frac{24273}{358400} hf_{n+\frac{1}{6}} - \frac{23659}{100800} hf_{n+\frac{1}{4}} + \frac{273213}{2867200} hf_{n+\frac{1}{3}} - \frac{73343}{215040} hf_{n+\frac{1}{2}} + \frac{46953}{409600} hf_{n+\frac{2}{3}} - \frac{6479}{33600} hf_{n+\frac{3}{4}} - \frac{156281}{25804800} hf_{n+1} \\
 h\gamma'_{n+\frac{1}{3}} + 2\gamma_{n+\frac{1}{2}} - 2\gamma_{n+1} &= -\frac{811}{279936} hf_n + \frac{302}{4725} hf_{n+\frac{1}{6}} - \frac{72418}{382725} hf_{n+\frac{1}{4}} + \frac{28039}{201600} hf_{n+\frac{1}{3}} - \frac{8777}{25515} hf_{n+\frac{1}{2}} + \frac{69989}{604800} hf_{n+\frac{2}{3}} - \frac{73954}{382725} hf_{n+\frac{3}{4}} - \frac{296341}{48988800} hf_{n+1} \\
 h\gamma'_{n+\frac{1}{2}} + 2\gamma_{n+\frac{1}{2}} - 2\gamma_{n+1} &= \frac{67}{20160} hf_n + \frac{27}{350} hf_{n+\frac{1}{6}} - \frac{386}{1575} hf_{n+\frac{1}{4}} + \frac{1539}{5600} hf_{n+\frac{1}{3}} - \frac{11}{42} hf_{n+\frac{1}{2}} + \frac{1161}{11200} hf_{n+\frac{2}{3}} - \frac{298}{1575} hf_{n+\frac{3}{4}} - \frac{11}{1800} hf_{n+1} \\
 h\gamma'_{n+\frac{2}{3}} + 2\gamma_{n+\frac{1}{2}} - 2\gamma_{n+1} &= -\frac{27569}{9797760} hf_n + \frac{302}{4725} hf_{n+\frac{1}{6}} - \frac{10894}{54675} hf_{n+\frac{1}{4}} + \frac{436339}{201600} hf_{n+\frac{1}{3}} - \frac{3713}{25515} hf_{n+\frac{1}{2}} + \frac{116789}{604800} hf_{n+\frac{2}{3}} - \frac{77794}{382725} hf_{n+\frac{3}{4}} - \frac{292261}{48988800} hf_{n+1} \\
 h\gamma'_{n+\frac{3}{4}} + 2\gamma_{n+\frac{1}{2}} - 2\gamma_{n+1} &= -\frac{3067}{1032192} hf_n + \frac{24273}{358400} hf_{n+\frac{1}{6}} - \frac{2371}{11200} hf_{n+\frac{1}{4}} + \frac{662013}{2867200} hf_{n+\frac{1}{3}} - \frac{11349}{71680} hf_{n+\frac{1}{2}} + \frac{717471}{2867200} hf_{n+\frac{2}{3}} - \frac{17117}{100800} hf_{n+\frac{3}{4}} - \frac{17329}{2867200} hf_{n+1} \\
 h\gamma'_{n+1} + 2\gamma_{n+\frac{1}{2}} - 2\gamma_{n+1} &= -\frac{61}{4480} hf_n - \frac{54}{175} hf_{n+\frac{1}{6}} + \frac{1514}{1575} hf_{n+\frac{1}{4}} - \frac{23409}{22400} hf_{n+\frac{1}{3}} + \frac{11}{15} hf_{n+\frac{1}{2}} - \frac{17793}{22400} hf_{n+\frac{2}{3}} + \frac{334}{525} hf_{n+\frac{3}{4}} + \frac{10793}{201600} hf_{n+1}
 \end{aligned} \quad (2.7)$$

Now, combining equation (2.5) and (2.7) together to obtain the CNS, which can be written as

$$\begin{aligned}
 \gamma_{n+1/6} &= \gamma_n + \frac{1}{6} h \gamma'_n + \frac{1000061}{176359680} h^2 f_n + \frac{1247}{42525} h^2 f_{n+1/6} - \frac{150733}{3444525} h^2 f_{n+1/4} + \frac{104833}{3628800} h^2 f_{n+1/3} - \frac{409}{45927} h^2 f_{n+1/2} + \frac{47623}{10886400} h^2 f_{n+2/3} - \frac{5989}{3444525} h^2 f_{n+3/4} + \frac{30853}{881798400} h^2 f_{n+1} \\
 \gamma_{n+1/4} &= \gamma_n + \frac{1}{4} h \gamma'_n + \frac{191741}{20643840} h^2 f_n + \frac{85887}{1433600} h^2 f_{n+1/4} - \frac{379}{4800} h^2 f_{n+1/2} + \frac{599157}{11468800} h^2 f_{n+2/3} - \frac{13789}{860160} h^2 f_{n+3/4} + \frac{90099}{11468800} h^2 f_{n+1} - \frac{629}{201600} h^2 f_{n+1/4} + \frac{719}{11468800} h^2 f_{n+1} \\
 \gamma_{n+1/3} &= \gamma_n + \frac{1}{3} h \gamma'_n + \frac{14221}{1102248} h^2 f_n + \frac{3874}{42525} h^2 f_{n+1/3} - \frac{373376}{3444525} h^2 f_{n+1/4} + \frac{617}{8100} h^2 f_{n+1/2} + \frac{5314}{229635} h^2 f_{n+2/3} + \frac{3853}{340200} h^2 f_{n+3/4} - \frac{15488}{3444525} h^2 f_{n+1} + \frac{311}{3444525} h^2 f_{n+1} \\
 \gamma_{n+1/2} &= \gamma_n + \frac{1}{2} h \gamma'_n + \frac{1621}{80640} h^2 f_n + \frac{27}{175} h^2 f_{n+1/2} - \frac{89}{525} h^2 f_{n+1/4} + \frac{6399}{44800} h^2 f_{n+1/3} - \frac{1}{30} h^2 f_{n+2/3} + \frac{783}{44800} h^2 f_{n+3/4} - \frac{11}{1575} h^2 f_{n+1} + \frac{19}{134400} h^2 f_{n+1} \\
 \gamma_{n+2/3} &= \gamma_n + \frac{2}{3} h \gamma'_n + \frac{18812}{688905} h^2 f_n + \frac{9248}{42525} h^2 f_{n+2/3} - \frac{796672}{3444525} h^2 f_{n+1/4} + \frac{3022}{14175} h^2 f_{n+1/2} - \frac{5024}{229635} h^2 f_{n+2/3} + \frac{166}{6075} h^2 f_{n+3/4} - \frac{34816}{3444525} h^2 f_{n+1} + \frac{682}{3444525} h^2 f_{n+1} \\
 \gamma_{n+3/4} &= \gamma_n + \frac{3}{4} h \gamma'_n + \frac{14187}{458752} h^2 f_n + \frac{356481}{1433600} h^2 f_{n+3/4} - \frac{5841}{22400} h^2 f_{n+1/4} + \frac{2827791}{11468800} h^2 f_{n+1/3} - \frac{3753}{286720} h^2 f_{n+1/2} + \frac{461457}{11468800} h^2 f_{n+2/3} - \frac{129}{11200} h^2 f_{n+3/4} + \frac{2637}{11468800} h^2 f_{n+1} \\
 \gamma_{n+1} &= \gamma_n + h \gamma'_n + \frac{109}{2520} h^2 f_n + \frac{54}{175} h^2 f_{n+1} - \frac{128}{525} h^2 f_{n+1/4} + \frac{81}{350} h^2 f_{n+1/3} - \frac{2}{21} h^2 f_{n+1/2} + \frac{27}{1400} h^2 f_{n+2/3} - \frac{128}{1575} h^2 f_{n+3/4} + \frac{1}{300} h^2 f_{n+1} \\
 \gamma'_{n+1/6} &= \gamma'_n + \frac{426463}{9797760} h f_n + \frac{65}{189} h f_{n+1/6} - \frac{34292}{76545} h f_{n+1/4} + \frac{11633}{40320} h f_{n+1/3} - \frac{2228}{25515} h f_{n+1/2} + \frac{5149}{120960} h f_{n+2/3} - \frac{1292}{76545} h f_{n+3/4} + \frac{3313}{9797760} h f_{n+1} \\
 \gamma'_{n+1/4} &= \gamma'_n + \frac{223577}{5160960} h f_n + \frac{26973}{71680} h f_{n+1/4} - \frac{7727}{20160} h f_{n+1/2} + \frac{156249}{573440} h f_{n+1/3} - \frac{18047}{215040} h f_{n+1/2} + \frac{23571}{573440} h f_{n+2/3} - \frac{47}{2880} h f_{n+3/4} + \frac{1691}{5160960} h f_{n+1} \\
 \gamma'_{n+1/3} &= \gamma'_n + \frac{26581}{612360} h f_n + \frac{352}{945} h f_{n+1/3} - \frac{25856}{76545} h f_{n+1/4} + \frac{797}{2520} h f_{n+1/2} - \frac{2216}{25515} h f_{n+2/3} + \frac{319}{7560} h f_{n+2/3} - \frac{256}{15309} h f_{n+3/4} + \frac{41}{122472} h f_{n+1} \\
 \gamma'_{n+1/2} &= \gamma'_n + \frac{1733}{40320} h f_n + \frac{27}{70} h f_{n+1/2} - \frac{124}{315} h f_{n+1/4} + \frac{405}{896} h f_{n+1/3} - \frac{1}{210} h f_{n+1/2} + \frac{27}{896} h f_{n+2/3} - \frac{4}{315} h f_{n+2/3} + \frac{11}{40320} h f_{n+1} \\
 \gamma'_{n+2/3} &= \gamma'_n + \frac{3329}{76545} h f_n + \frac{352}{945} h f_{n+2/3} - \frac{26624}{76545} h f_{n+1/4} + \frac{124}{315} h f_{n+1/3} - \frac{2848}{25515} h f_{n+1/2} + \frac{113}{945} h f_{n+2/3} - \frac{2048}{76545} h f_{n+3/4} + \frac{32}{76545} h f_{n+1} \\
 \gamma'_{n+3/4} &= \gamma'_n + \frac{24849}{573440} h f_n + \frac{26973}{71680} h f_{n+3/4} - \frac{807}{2240} h f_{n+1/4} + \frac{234009}{573440} h f_{n+1/3} - \frac{7083}{71680} h f_{n+1/2} + \frac{101331}{573440} h f_{n+2/3} - \frac{3}{448} h f_{n+3/4} + \frac{39}{114688} h f_{n+1} \\
 \gamma'_{n+1} &= \gamma'_n + \frac{151}{2520} h f_n + \frac{256}{315} h f_{n+1} - \frac{243}{280} h f_{n+1/4} + \frac{104}{105} h f_{n+1/3} - \frac{243}{280} h f_{n+1/2} + \frac{256}{315} h f_{n+2/3} + \frac{151}{2520} h f_{n+1}
 \end{aligned}$$

(2.8)

Analysis of Computational Numerical Scheme(CNS)

In this section, the analysis of the basic properties of the CNS are analyzed. These properties are order, error constant, consistency, zero-stability and region of absolute stability.

Order and Error Constant of CNS

Let the linear operator defined on the method be $[\gamma(t); h]$, where,

$$\Lambda\{\gamma(t); h\} = A^{(0)} Y_m^{(i)} - \sum_{i=0}^k \frac{j h^{(i)}}{i!} \gamma_n^{(i)} - h^{(2-1)} [\delta_i f(\gamma_n) + b_i Z(Y_m)] \quad (3.1)$$

Expanding Y_m and $Z(Y_m)$ in Taylor series and comparing the coefficients of h gives

$$\Lambda\{\gamma(t); h\} = C_0 \gamma(t) + C_1 \gamma'(t) + \Lambda + C_p h^p \gamma^p(t) + C_{p+1} h^{p+1} \gamma^{p+1}(t) + C_{p+2} h^{p+2} \gamma^{p+2}(t) + \Lambda \quad (3.2)$$

Definition 3.1: The linear operator Λ and the CNS are said to be of order p if $C_0 = C_1 = \dots = C_p = C_{p+1} = 0$, $C_{p+2} \neq 0$. C_{p+2} is called the error constant and implies that the truncation error is given by $t_{n+k} = C_{p+2} h^{p+2} \gamma^{p+3}(t) + O(h^{p+3})$

$$\Lambda\{\gamma(t); h\} = C_0 \gamma(t) + C_1 \gamma'(t) + \Lambda + C_p h^p \gamma^p(t) + C_{p+1} h^{p+1} \gamma^{p+1}(t) + C_{p+2} h^{p+2} \gamma^{p+2}(t) + \Lambda \quad (3.3)$$

Apply definition 3.1 alongside with equation (3.3) to obtain

$$\begin{bmatrix} \sum_{j=0}^{\infty} \frac{\left(\frac{1}{6}\right)^j}{j!} - \gamma_n - \frac{1}{6} h \gamma'_n - \frac{1000061}{176359680} h \gamma''_n - \sum_{j=0}^{\infty} \frac{h^{j+2}}{j!} \gamma_n^{(j+2)} \left[\frac{1247}{42525} \left(\frac{1}{6}\right) - \frac{150733}{3444525} \left(\frac{1}{4}\right) + \frac{104833}{3628800} \left(\frac{1}{3}\right) - \frac{409}{45927} \left(\frac{1}{2}\right) + \frac{47623}{10886400} \left(\frac{2}{3}\right) - \frac{5989}{3444525} \left(\frac{3}{4}\right) + \frac{30853}{881798400} (1) \right] \\ \sum_{j=0}^{\infty} \frac{\left(\frac{1}{4}\right)^j}{j!} - \gamma_n - \frac{1}{4} h \gamma'_n - \frac{191741}{20643840} h \gamma''_n - \sum_{j=0}^{\infty} \frac{h^{j+2}}{j!} \gamma_n^{(j+2)} \left[\frac{85887}{1433600} \left(\frac{1}{6}\right) - \frac{379}{4800} \left(\frac{1}{4}\right) + \frac{599157}{11468800} \left(\frac{1}{3}\right) - \frac{13789}{860160} \left(\frac{1}{2}\right) + \frac{90099}{11468800} \left(\frac{2}{3}\right) - \frac{629}{201600} \left(\frac{3}{4}\right) + \frac{719}{11468800} (1) \right] \\ \sum_{j=0}^{\infty} \frac{\left(\frac{1}{3}\right)^j}{j!} - \gamma_n - \frac{1}{3} h \gamma'_n - \frac{14221}{1102248} h \gamma''_n - \sum_{j=0}^{\infty} \frac{h^{j+2}}{j!} \gamma_n^{(j+2)} \left[\frac{3874}{42525} \left(\frac{1}{6}\right) - \frac{373376}{3444525} \left(\frac{1}{4}\right) + \frac{617}{8100} \left(\frac{1}{3}\right) - \frac{5314}{229635} \left(\frac{1}{2}\right) + \frac{3853}{340200} \left(\frac{2}{3}\right) - \frac{15488}{3444525} \left(\frac{3}{4}\right) + \frac{311}{3444525} (1) \right] \\ \sum_{j=0}^{\infty} \frac{\left(\frac{1}{2}\right)^j}{j!} - \gamma_n - \frac{1}{2} h \gamma'_n - \frac{1621}{80640} h \gamma''_n - \sum_{j=0}^{\infty} \frac{h^{j+2}}{j!} \gamma_n^{(j+2)} \left[\frac{27}{175} \left(\frac{1}{6}\right) - \frac{89}{525} \left(\frac{1}{4}\right) + \frac{6399}{44800} \left(\frac{1}{3}\right) - \frac{1}{30} \left(\frac{1}{2}\right) + \frac{783}{44800} \left(\frac{2}{3}\right) - \frac{11}{1575} \left(\frac{3}{4}\right) + \frac{19}{134400} h^2 f_{n+1} \right] \\ \sum_{j=0}^{\infty} \frac{\left(\frac{2}{3}\right)^j}{j!} - \gamma_n - \frac{2}{3} h \gamma'_n - \frac{18812}{688905} h \gamma''_n - \sum_{j=0}^{\infty} \frac{h^{j+2}}{j!} \gamma_n^{(j+2)} \left[\frac{9248}{42525} \left(\frac{1}{6}\right) - \frac{796672}{3444525} \left(\frac{1}{4}\right) + \frac{3022}{14175} \left(\frac{1}{3}\right) - \frac{5024}{229635} \left(\frac{1}{2}\right) + \frac{166}{6075} \left(\frac{2}{3}\right) - \frac{34816}{3444525} \left(\frac{3}{4}\right) + \frac{682}{3444525} (1) \right] \\ \sum_{j=0}^{\infty} \frac{\left(\frac{3}{4}\right)^j}{j!} - \gamma_n - \frac{3}{4} h \gamma'_n - \frac{14187}{458752} h \gamma''_n - \sum_{j=0}^{\infty} \frac{h^{j+2}}{j!} \gamma_n^{(j+2)} \left[\frac{356481}{1433600} \left(\frac{1}{6}\right) - \frac{5841}{22400} \left(\frac{1}{4}\right) + \frac{2827791}{11468800} \left(\frac{1}{3}\right) - \frac{3753}{286720} \left(\frac{1}{2}\right) + \frac{461457}{11468800} \left(\frac{2}{3}\right) - \frac{129}{11200} \left(\frac{3}{4}\right) + \frac{2637}{11468800} (1) \right] \\ \sum_{j=0}^{\infty} \frac{(1)^j}{j!} - \gamma_n - h \gamma'_n - \frac{109}{2520} h \gamma''_n - \sum_{j=0}^{\infty} \frac{h^{j+2}}{j!} \gamma_n^{(j+2)} \left[\frac{54}{175} \left(\frac{1}{6}\right) - \frac{128}{525} \left(\frac{1}{4}\right) + \frac{81}{350} \left(\frac{1}{3}\right) - \frac{2}{21} \left(\frac{1}{2}\right) + \frac{27}{1400} \left(\frac{2}{3}\right) - \frac{128}{1575} \left(\frac{3}{4}\right) + \frac{1}{300} (1) \right] \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

Comparing the coefficient of h , according to Skwameet *al.* (2019), the CNS is of uniform order $p = [7 \ 7 \ 7 \ 7 \ 7 \ 7 \ 7]^T$. The error constant are $C_{p+2} = [-1.3640 \times 10^{-10} \ -1.3281 \times 10^{-10} \ -1.3497 \times 10^{-10} \ -1.1961 \times 10^{-10} \ -1.4526 \times 10^{-10} \ -1.3561 \times 10^{-10} \ -1.4353 \times 10^{-09}]$

Consistency of the CNS

A CNS is consistent since the order of CNS p is greater than or equal to zero to one i.e. $p \geq 1$ (Skwameet *al.* 2019).

Zero Stability of the CNS

Definition 3.2: The CNS is said to be zero-stable, if the roots z_s , $s = 1, 2, \dots, L, k$ of the first characteristics polynomial $\rho(z)$ defined by $\rho(z) = \det(zA^{(0)} - E)$ satisfies $|z_s| \leq 1$ and every root satisfies $|z_s| = 1$, Sunday (2018). The first characteristic polynomial is given by,

$$\rho(z) = z \begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{bmatrix} - \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{bmatrix} = z^6(z-1) = z^6(z-1)$$

Thus, solving for z in

$$z^6(z-1) \quad (3.3)$$

gives $z = 0, 0, 0, 0, 0, 0, 1$. Hence the CNS is said to be zero-stable.

Convergence of the CNS

Theorem 3.1: The necessary and sufficient conditions for linear multistep method to be convergent are that it must be consistent and zero-stable (Skwameet *al.* (2019). Hence the CNS formulated is consistent.

Region of Absolute Stability of CNS

Definition 3.3: The region of absolute stability is the region of the complex plane Z , where $z = \lambda h$ for which the CNS is absolute stable. To determine the region of absolute

stability of the CNS, the method that compare neither the computation of roots of a polynomial nor solving of simultaneous inequalities is adopted. Thus, the method according to Sunday, (2018) is called the boundary locus method. Applying the boundary locus method on the CNS, we obtain the stability polynomial of the CNSas

$$\bar{h}(w) = \left(-\frac{1}{11612160}w^6 - \frac{163}{1463132160}w^7 \right)h^{14} + \left(-\frac{810757}{8778792960}w^6 + \frac{113}{34836480}w^7 \right)h^{12} + \left(-\frac{4473503}{3762339840}w^6 - \frac{61}{829440}w^7 \right)h^{10} + \left(-\frac{1235057}{522547200}w^6 + \frac{481}{414720}w^7 \right)h^8 + \left(-\frac{128809}{6967296}w^6 - \frac{67}{5184}w^7 \right)h^6 + \left(-\frac{817}{5040}w^6 + \frac{113}{1152}w^7 \right)h^4 + \left(-\frac{17}{30}w^6 - \frac{11}{24}w^7 \right)h^2 - 2w^6 + w^7 \right) \quad (3.4)$$

Using the stability polynomial in equation (3.4), we obtain the region of absolute stability in figure below as

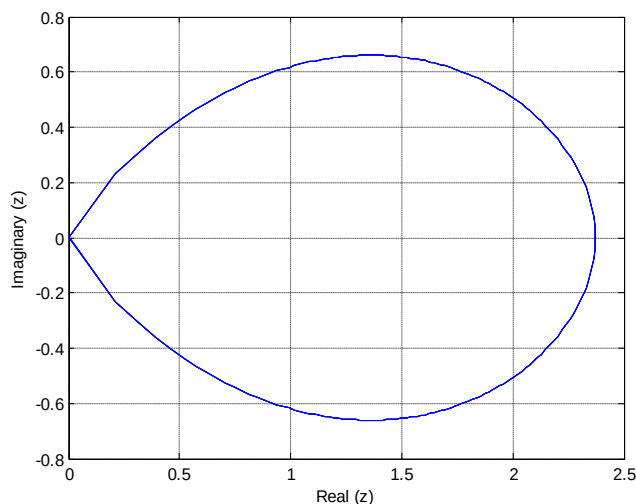


Figure 3.1: Stability region of CNS.

The stability region obtained in Figure 3.1 is A-stable, since its region of absolute stability include the whole left half of the complex z-plane.

Numerical Applications of CNS

The Computational Numerical Scheme (CNS) was implemented on second order initial value problems of ordinary differential equation of the form (1.1) without the need to reduce it to an equivalent system of first order ODEs. The study conducted a comparison of absolute errors which were calculated from approximate solutions against results obtained through established methods for each application.

Example 4.1:

Consider the second-order Bessel differential equation describing an oscillatory physical problem, given as follows:

$$\frac{d^2y}{dt^2} + \frac{dy}{dt} = 0.001\cos(t), \quad y(0)=1, \quad \frac{dy}{dt} = 0 \quad (4.1)$$

with analytical solution of (4.1) as

$$y(t) = \cos(t) + 0.0005t\sin(t) \quad (4.2)$$

Source: [Olabode and Momoh (2016); Lydia *et al.* (2021)]

Table 4.1 and Figure 4.1 present numerical results which compare the CNS method against the methods developed by Olabode and Momoh (2016) and Lydia *et al.* (2021). The notation AEOM16 refers to the work of Olabode and Momoh (2016) while AELe21 stands for the research of Lydia *et al.* (2021).

Example 4.2:

Consider the second-order oscillatory dynamic problem modeled as follows:

$$\frac{d^2y}{dt^2} + 9\frac{dy}{dt} + 14y(t) = \frac{1}{2}\sin(t), y(0) = 0, y'(0) = -1 \quad (4.3)$$

with the analytic solution of (4.3) is given by,

$$y(t) = \frac{1}{500}(-90e^{-2t} + 99e^{-7t} + 13\sin t - 9\cos t) \quad (4.4)$$

Source: [Skwameet *al.* (2017); Skwameet *al.* (2020)].

The absolute error of the CNS method, denoted as AECNS, was compared with the absolute errors of the existing methods of Skwameet *al.* (2017), denoted as AESe17; Skwameet *al.* (2020), denoted as AESe20; and Sabo *et al.* (2021), denoted as AESe21. The results are presented in Table 4.2 and are shown graphically in Figure 4.2.

Example 4.3:

The mass-spring system of the second-order oscillatory problem is modeled as follows:

$$\frac{d^2y}{dt^2} + 16y = 2\sin 4t, y(0) = -\frac{1}{2}, y'(0) = 0 \quad (4.5)$$

is consider with the analytic solution of (4.5) is given by,

$$y(t) = -\frac{1}{2}\cos 4t + \frac{1}{16}\sin 4t - \frac{1}{4}t\cos 4t \quad (4.6)$$

Source: [Skwameet *al.* (2017); Sabo *et al.* (2021)].

Table 4.3 presents a comparison of the absolute errors of the CNS method with those of Skwameet *al.* (2017) and Sabo *et al.* (2021). The error curves are displayed in Figure 4.3 as a visual representation of the data while AESe17 represents Skwameet *al.* (2017) and AESe21 represents Sabo *et al.* (2021).

Table 4.1: Numerical Comparison of Example 4.1

t	Analytic Solution	Approximate Solution	AECNS	AELe21	AEOM16
0.1	0.09978366643856425102	0.09978366643856425102	0.0000e00	1.2567e-12	1.0170e-12
0.2	0.19857132413727709130	0.19857132413727709132	2.0000e-20	2.1140e-12	1.4285e-11
0.3	0.29537690618797073421	0.29537690618797073422	1.0000e-20	2.3764e-12	4.9557e-11
0.4	0.38923413010984991465	0.38923413010984991467	2.0000e-20	3.4242e-12	1.0161e-10
0.5	0.47920614296373040709	0.47920614296373040711	2.0000e-20	3.3944e-12	1.7416e-10
0.6	0.56439487271056245371	0.56439487271056245372	1.0000e-20	3.3436e-12	2.6425e-10
0.7	0.64394999247214148272	0.64394999247214148273	1.0000e-20	4.2949e-12	3.7579e-10
0.8	0.71707740821578389546	0.71707740821578389546	0.0000e00	4.2574e-12	5.0602e-10
0.9	0.78304718514176158945	0.78304718514176158945	0.0000e00	5.2344e-12	6.5904e-10
1.0	0.84120083365496243679	0.84120083365496243678	1.0000e-20	6.2265e-12	8.3225e-10

Table 4.2: Numerical Comparison of Example 4.2

t	Analytic Solution	Approximate Solution	AECNS	AESe17	AESe21	AESe20
0.1	-0.06436205154552458248	-0.06436205154550692713	1.7655e-14	1.2744e-08	2.0453e-10	4.4268e-09
0.2	-0.08430720522644774945	-0.08430720522643379455	1.3955e-14	3.0442e-08	4.8485e-10	2.2383e-08
0.3	-0.08405225313390041905	-0.08405225313389384414	6.5749e-15	4.1501e-08	6.6174e-10	3.5865e-08
0.4	-0.07529304213333374810	-0.07529304213333305897	6.8913e-16	4.5385e-08	7.2649e-10	4.2157e-08
0.5	-0.06357063960355798563	-0.06357063960356088723	2.9016e-15	4.4298e-08	7.1295e-10	4.2895e-08
0.6	-0.05142117069384508163	-0.05142117069384974188	4.6603e-15	4.0466e-08	6.5550e-10	4.0288e-08
0.7	-0.03993052956438697070	-0.03993052956439220056	5.2299e-15	3.5475e-08	5.7884e-10	3.6051e-08
0.8	-0.02949865862803573900	-0.02949865862804086216	5.1232e-15	3.0285e-08	4.9808e-10	3.1287e-08
0.9	-0.02021269131259124546	-0.02021269131259569819	4.6813e-15	2.5408e-08	4.2140e-10	2.6618e-08
1.0	-0.01202699425403169607	-0.01202699425403580479	4.1087e-15	2.1071e-08	3.5257e-10	2.2352e-08

Table 4.3: Numerical Comparison of Example 4.3

t	Analytic Solution	Approximate Solution	AECNS	AESe17	AESe21
0.01	-0.49959872021047678004	-0.49959872021047678005	1.0000e-20	1.6621e-09	1.0000e-19
0.02	-0.49839019330974949646	-0.49839019330974949645	1.0000e-20	1.1586e-08	4.1000e-19
0.03	-0.49636836974027966301	-0.49636836974027966301	0.0000e00	2.9743e-08	9.1000e-19
0.04	-0.49352852660817937130	-0.49352852660817937131	1.0000e-20	5.6076e-08	1.6600e-18
0.05	-0.48986728796894500998	-0.48986728796894500999	1.0000e-20	9.0504e-08	2.6200e-18
0.06	-0.48538264289709933476	-0.48538264289709933476	0.0000e00	1.3291e-07	3.8000e-18
0.07	-0.48007396129056685722	-0.48007396129056685721	1.0000e-20	1.8317e-07	5.2000e-18
0.08	-0.47394200736436189072	-0.47394200736436189071	1.0000e-20	2.4110e-07	6.8500e-18
0.09	-0.46698895079202783994	-0.46698895079202783995	1.0000e-20	3.0653e-07	8.7500e-18
0.1	-0.45921837545722401274	-0.45921837545722401275	1.0000e-20	1.6621e-09	1.0850e-17

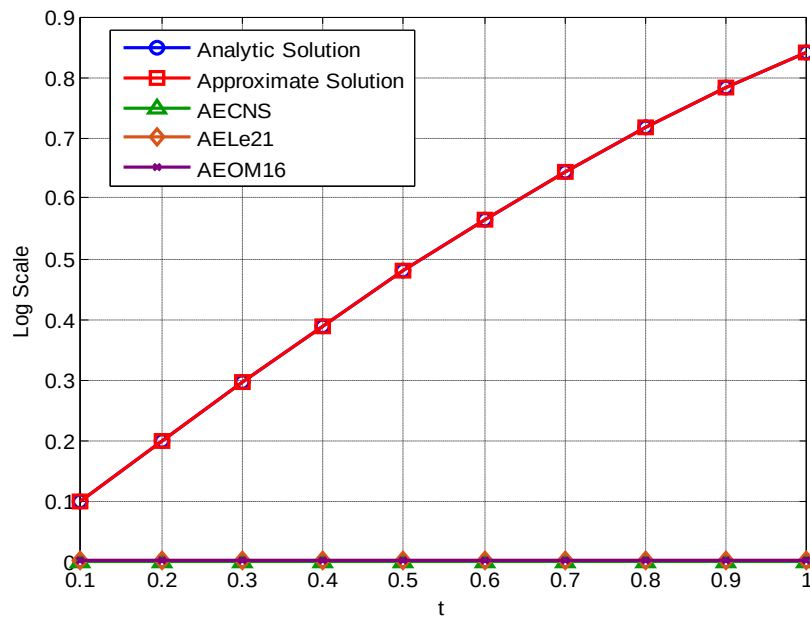


Figure 4.1: Graphical Curve of Table 4.1

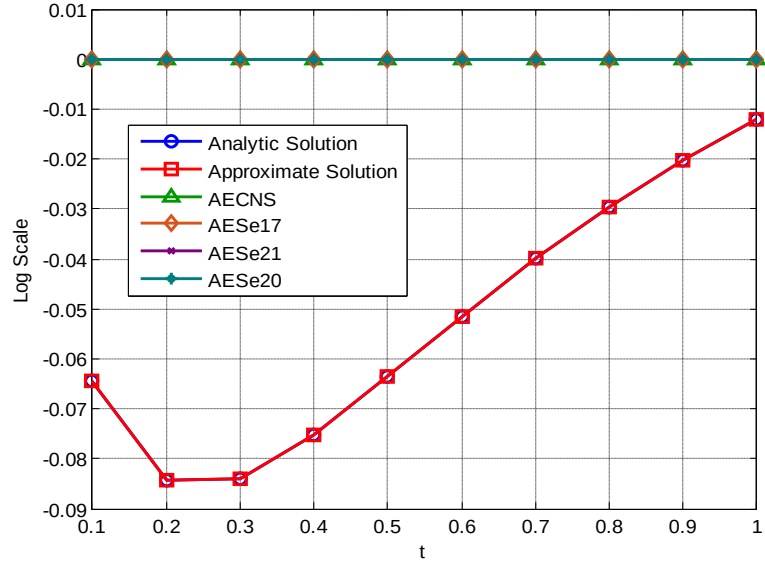


Figure 4.2: Graphical Curve of Table 4.2

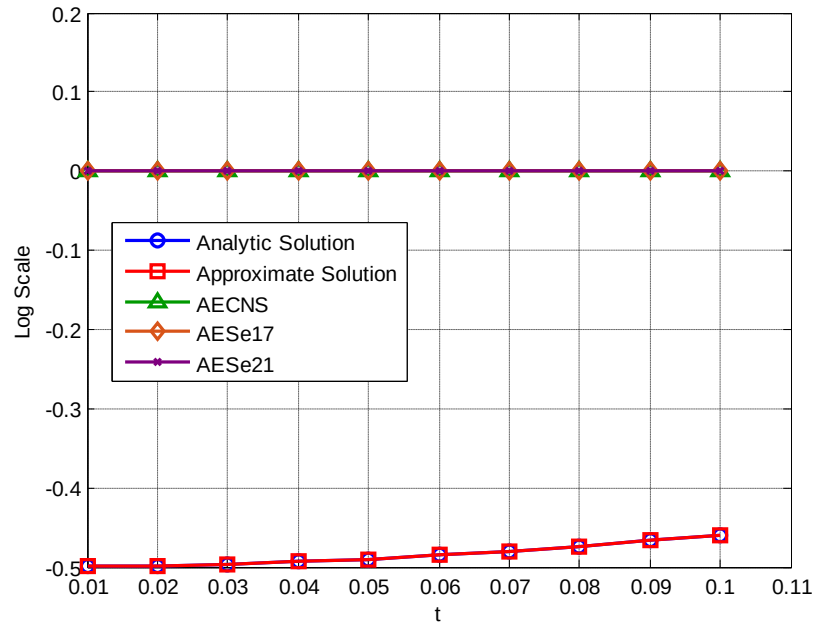


Figure 4.3: Graphical Curve of Table 4.3

Discussion of Results

The Computational Numerical Scheme (CNS) was applied to selected second-order initial value problems of ordinary differential equations of the form (1.1) without reducing them to equivalent systems of first-order equations. This direct implementation preserves the original mathematical structure of the problems and simplifies the computational procedure. For each example considered, the numerical solutions obtained using the CNS method were compared with the corresponding analytical solutions and with results from established methods in the literature. The comparisons were presented in tables and illustrated graphically to demonstrate the effectiveness of the scheme.

In Example 4.1, the CNS method was implemented on a second-order Bessel differential equation that models an oscillatory physical phenomenon. The numerical results displayed in Table 4.1 and Figure 4.1 show a very close agreement between the analytical and approximate solutions. When compared with the methods developed by Olabode and Momoh (2016) and Lydia *et al.* (2021), the CNS method demonstrates excellent consistency and reliability across the integration interval. The graphical representation confirms that the solution curve produced by the CNS method closely follows the exact solution, reflecting its strong performance in handling oscillatory problems.

Example 4.2 considers another second-order oscillatory dynamic problem with a known analytical solution. The results presented in Table 4.2 and Figure 4.2 reveal that the CNS method produces solutions that align closely with the exact values. In comparison with the approaches of Skwame *et al.* (2017), Skwame *et al.* (2020), and Sabo *et al.* (2021), the CNS method maintains a high level of computational accuracy and stability. The graphical curves further illustrate the effectiveness of the scheme, as the numerical solution nearly overlaps with the analytical solution throughout the domain.

In Example 4.3, the CNS method was applied to a classical mass-spring system modeled by a second-order oscillatory differential equation. The results shown in Table 4.3 and Figure 4.3 indicate that the approximate solutions generated by the CNS method are in strong agreement with the analytical solution. When compared with existing methods, the CNS approach demonstrates comparable or improved performance. The graphical illustration supports this observation, highlighting the smoothness and stability of the solution profile. Overall, the numerical applications confirm that the CNS method is efficient, accurate, and well-suited for solving second-order oscillatory initial value problems directly.

Summary and Conclusions

The researchers created and executed a Computational Numerical Scheme (CNS) which solves second-order oscillatory initial value problems without requiring first-order system transformations. The method was derived through power series approximation which combined interpolation with collocation techniques to produce a continuous hybrid linear multistep system. The analysis of CNS showed that it possesses key numerical properties wastested, that is the order and consistency and zero-stability and convergence and absolute stability which established A-stability for the system. The method was tested through three test problems which used oscillatory systems that included Bessel-type equations and mass-spring models. The numerical results which appear in Tables 4.1 to 4.3 and Figures 4.1 to 4.3 demonstrated that CNS achieved highly accurate solutions which resulted in minimal absolute errors when compared to methods found in existing literature.

The Computational Numerical Scheme achieved better results than all other methods considered because it provided better accuracy, stability and efficiency when solving second-order oscillatory differential equations. The CNS produced errors that were lower than those found in established numerical methods considered. The method proved reliable through graphical results, which showed that the CNS error curves stayed below those of all competing schemes. The method provides scientists and engineers with an effective solution to real-world oscillatory problems that require second-order ordinary differential equation modeling because it delivers both high precision and efficient computing capabilities.

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