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Parametrization of Algebraic Points of Low Degrees on the Schaeffer Curve

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Abstract

In this paper, we give a parametrization of algebraic points of degree at most 4 over $\mathbb{Q}$ on the schaeffer curve $\mathscr{C}$ of affine equation : $y^2 = x^5 + 1$. The result extends our previous result which describes in [5] (Afr. Mat 29:1151-1157, 2018) the set of algebraic points of degree at most 3 over $\mathbb{Q}$ on this curve.

1. Introduction

Let $\mathscr{C}$ be a smooth projective plane curve defined over K. For all algebraic extension field K of $\mathbb{Q}$, we denote by $\mathscr{C}(K)$ the set of K-rational points of $\mathscr{C}$ over K and $\mathscr{C}^{(d)}(\mathbb{Q})$ the set of algebraic points of $\leq d$ over $\mathbb{Q}$. The degree of an algebraic point R is the degree of its field of definition on $\mathbb{Q}$: $deg(R) = [\mathbb{Q}(R) : \mathbb{Q}]$.
A famous theorem of Fatling show that if $\mathscr{C}$ is a smooth projective plane curve defined over K of genus $g \geq 2$, then $\mathscr{C}(K)$ is finite. Fatling's proof is still ineffective in the sense that it does not provide an algorithm for computing $\mathscr{C}(K)$. A most precise theorem of Debarre and Klasen [4] show that if $\mathscr{C}$ be a smooth projective plane curve defined by an equation of degree $d \geq 7$ with rational coefficients then $\mathscr{C}^{(d-2)}(\mathbb{Q})$ is finite. This theorem often us to characterize the set $\mathscr{C}^{(2)}(\mathbb{Q})$ of all algebraic points of degree ≤ 2 over $\mathbb{Q}$.
Currently for curve $\mathscr{C}$ defined over a numbers field K of genus $g \geq 2$, there is no known algorithm for computing the set $\mathscr{C}(K)$ or for deciding if $\mathscr{C}(K)$ is empty. But there is a bag of strikes that can be used to show that $\mathscr{C}(K)$ is empty, or to determine $\mathscr{C}(K)$ if it is not empty. Among these methods, we can cite the local method, Chabauty method [2], Descent method [7], mordell-weil sieves method [1]. These methods often succeed with less than full knowledge of the jacobian $J(\mathbb{Q})$ of the curve . If $J(\mathbb{Q})$ is finite then it is no hard to determine $\mathscr{C}(\mathbb{Q})$ and to generalize for all number field K.
Previous works ([3] and [5]) have studied the algebraic points of degree at most 3 on the schaeffer curve of affine equation $y^2 = x^5 + 1$ denoted $\mathscr{C}$. The curve $\mathscr{C}$ is hyperelliptic of genus $g = 2$ and of rank null by [3].
Let's denote $P_0 = (-1, 0)$, $P_1 = (0, 1)$, $\overline{P_1} = (0, -1)$, $Q_1 = (1+i, 1-2i)$, $Q_2 = (1-i, 1+2i)$, $\overline{Q_1} = (1+i, -1+2i)$, $\overline{Q_2} = (1-i, -1-2i)$ and ∞ the point at infinity.
The purpose of this note is to determine the algebraic parametrization of all algebraic points of degree at most four on the curve $\mathscr{C}_s$ over the rationnal numbers field $\mathbb{Q}$ using ideas in [5] (Afr. Mat 29:1151-1157, 2018).

2. Auxiliary results

Lemma 2.1. *Let x and y be the rational functions defined on $\mathscr{C}_s$ by $x(X,Y,Z) = \frac{X}{Z}$ and $y(X,Y,Z) = \frac{Y}{Z}$:*

- $div(y-1) = 5P_1 - 5\infty$; $\quad div(y+1) = 5\overline{P}_1 - 5\infty$;
- $div(x) = P_1 + \overline{P}_1 - 2\infty$; $\quad div(x+1) = 2P_0 - 2\infty$
- $div(y) = A_0 + A_1 + A_2 + A_3 + A_4 - 5\infty$ *where* $A_i = exp(i(2k+1)\frac{\pi}{5})$.

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Denote by $\mathscr{L}(m\infty)$ the $\overline{\mathbb{Q}}-$vector space of rational functions defined by $\mathscr{L}(m\infty)=\left\{f\in\overline{\mathbb{Q}}(\mathscr{C}_s)^* \mid div(f)\geq -m\infty\right\}\cup\{0\}$:

- $\mathscr{L}(\infty)=\langle\,1\,\rangle$
- $\mathscr{L}(2\infty)=\mathscr{L}(3\infty)=\langle\,1,\,x\,\rangle$
- $\mathscr{L}(4\infty)=\langle\,1,\,x,\,x^2\,\rangle$
- $\mathscr{L}(5\infty)=\langle\,1,\,x,\,x^2,\,y\,\rangle$
- $\mathscr{L}(6\infty)=\langle\,1,x,x^2,y,x^3\,\rangle$

Proof. See [5]

Lemma 2.2. *We consider the divisor D on the curve $\mathscr{C}_s$:*

- $D=[(-1,\,0)+(0,\,1)-2\infty]=[P_0+P_1-2\infty]$
- $2D=[2\,(0,\,1)-2\infty]=[2P_1-2\infty]$
- $3D=[(1+i,\,1-2i)+(1-i,\,1+2i)-2\infty]=[Q_1+Q_2-2\infty]$
- $4D=[(0,\,-1)-\infty]=\left[\overline{P}_1-\infty\right]$
- $5D=[(-1,\,0)-\infty]=[P_0-\infty]$
- $6D=[(0,\,1)-\infty]=[P_1-\infty]$
- $7D=[(1+i,\,-1+2i)+(1-i,\,-1-2i)-2\infty]=\left[\overline{Q}_1+\overline{Q}_2-2\infty\right]$
- $8D=[2\,(0,\,-1)-2\infty]=\left[2\overline{P}_1-2\infty\right]$
- $9D=[(-1,\,0)+(0,\,-1)-2\infty]=\left[P_0+\overline{P}_1-2\infty\right]$
- $10D=0.$

The Mordell-weil groupe of the curve $\mathscr{C}_s$ is $J(\mathbb{Q})\cong(\mathbb{Z}/10\mathbb{Z})\cong\langle\,D\rangle=\{mD\mid 0\leq m\leq 9\}$.

Proof. See [3].

3. Main result

Our main result is the following theorem

Theorem 3.1. *The algebraic points of degree 4 over $\mathbb{Q}$ on the curve $\mathscr{C}_s$ are given by the union of the following sets : $\mathscr{G}_0\cup\mathscr{G}_1\cup\mathscr{G}_2\cup\mathscr{G}_3\cup\mathscr{G}_4\cup\mathscr{G}_5$ with*

- $\mathscr{G}_0=\left\{\left(x,\,\pm\sqrt{x^2+1}\right)\right) \mid [\mathbb{Q}(x):\mathbb{Q}]=2\,,x^2-2x+2\neq 0\Big\}$;
- $\mathscr{G}_1=\left\{\begin{array}{l}\left(x,\,\pm(-1+(-1-a+c)x-ax^2-cx^3\right) \mid a,c\in\mathbb{Q},c\neq 0\,et\,a\neq c-1,\,x\ root\ of\\ B_1(x)=c^2x^4+\left(2ac-c^2-1\right)x^3+\left(a^2-c^2+2c+1\right)x^2+\left(a^2+2a-2ac+c^2-1\right)x+2a-2c+2=0\end{array}\right\}$;
- $\mathscr{G}_2=\left\{\begin{array}{l}\left(x,\,\pm\left(cx^3+ax^2-1\right)\right) \mid a,c\in\mathbb{Q}^*,a\neq c+1,\,x\ root\ of\ B_2(x)=c^2x^4+2acx^3-x^3+a^2x^2-2cx-2a=0\end{array}\right\}$;
- $\mathscr{G}_3=\left\{\begin{array}{l}\left(x,\,\pm(-3-2a-4c+(2+2a+2c)x-ax^2-cx^3)\right) \mid a,c\in\mathbb{Q},a\neq -1-2c,c\neq 0,\,x\ root\ of\ B_3(x)=c^2x^4+\\ \left(2c^2+2ac-1\right)x^3+\left(-2c^2-4c+a^2-2\right)x^2+\left(-4ac-2c-2a^2-4a-2\right)x+8c^2+8ac+12c+2a^2+6a+4\end{array}\right\}$;
- $\mathscr{G}_4=\left\{\begin{array}{l}\left(x,\,\pm(1+ax+cx^2)\right) \mid a,c\in\mathbb{Q},a\neq 0,\,x\ root\ of\ B_4(x)=-x^4+c^2x^3+2acx^2+\left(2c+a^2\right)x+2\end{array}\right\}$;
- $\mathscr{G}_5=\left\{\begin{array}{l}\left(x,\,\pm(-a+(-a-c)x-cx^2)\right) \mid,a,c\in\mathbb{Q},\,a\neq\pm 1,\,x\ root\ of B_5(x)=-x^4+\left(c^2+1\right)x^3+\left(c^2+2ac-1\right)x^2\\ +\left(2ac+a^2+1\right)x+a^2-1\end{array}\right\}$.

Proof of theoreme.
Let $R\in\mathscr{C}_s\left(\overline{\mathbb{Q}}\right)$ with $[\mathbb{Q}(R):\mathbb{Q}]=4$. Let R_1,R_2,R_3,R_4 be the Galois conjugates of R. We have

$$[R_1+R_2+R_3+R_4-4\infty]\in J(\mathbb{Q})$$

from lemma (2.2) , we get

$$[R_1+R_2+R_3+R_4-4\infty]=mD,\quad 0\leq m\leq 9$$

Now for any integer m such that $0\leq m\leq 9$, we have $mD=(m-10)D$, so

$$[R_1+R_2+R_3+R_4-4\infty]=(m-10)\,D\,,\quad 0\leq m\leq 9.\qquad(\star)$$

Our proof is divided in five cases

$$\underline{\text{Case } m=0}$$

Formula $(\star)$ becomes

$$[R_1+R_2+R_3+R_4-4\infty]=0.$$

The Abel Jacobi theorem involves the existence of a function F such that

$$div(F)=R_1+R_2+R_3+R_4-4\infty$$

so $F\in\mathscr{L}(4\infty)$, and lemma (2.1) gives $F(x,y)=a+bx+cx^2$; x must be in the $\overline{\mathbb{Q}}$ such as $[\mathbb{Q}(x):\mathbb{Q}]=2$ and $x^2-2x+2\neq 0$. We get a family of quartic points

$$\mathscr{G}_0=\left\{\left(x,\,\pm\sqrt{x^5+1}\right)\right) \mid x\in[\mathbb{Q}(x):\mathbb{Q}]=2,\,x^2-2x+2\neq 0\Big\}.$$

$$\underline{\text{Cases } m=1 \text{ and } m=9}$$

For $m=1$: The formula $(\star)$ and lemma (2.2) give

$$[R_1+R_2+R_3+R_4-4\infty]=-9D=-\left[P_0+\overline{P}_1-2\infty\right].$$

This means

$$\left[R_1+R_2+R_3+R_4+P_0+\overline{P}_1-6\infty\right]=0$$

The Abel Jacobi theorem involves the existence of a function F such that

$$div(F)=R_1+R_2+R_3+R_4+P_0+\overline{P}_1-6\infty.$$

So $F\in\mathscr{L}(6\infty)$, then $F(x,y)=u+vx+wx^2+dx^3+ey$ $(e\neq 0)$. We have $F\left(\overline{P}_1\right)=F(P_0)=0$, so $u-e=0$ and $u-v+w-d=0$, thus

$$F(x,y)=u+(u+w-d)x+wx^2+dx^3+uy \quad u\neq 0.$$

At the points R_i, we have $y=-1+(-1-a+c)x-ax^2-cx^3$ with $a=\frac{w}{u}$ and $c=\frac{d}{u}$. By substituting y in $y^2-x^5-1=0$ and simplifying by $x(x+1)$ we obtain

$$B_1(x)=c^2x^4+\left(2ac-c^2-1\right)x^3+\left(a^2-c^2+2c+1\right)x^2+\left(a^2+(2-2c)a+c^2-1\right)x+2a-2c+2=0$$

We must have $B_1(0)\neq 0$ and $B_1(-1)\neq 0$ which involves $a\neq c-1$ and $c\neq 0$. We have a family of quartic points

$$\mathscr{G}_{1,1}=\left\{\ \left(x,\ +(-1+(-1-a+c)x-ax^2-cx^3)\right)\mid a,c\in\mathbb{Q},\ a\neq c-1,c\neq 0,x \text{ root of } B_1(x)=0\ \right\}$$

For $m=9$: The formula $(\star)$ and lemma (2.2) give

$$[R_1+R_2+R_3+R_4-4\infty]=-D=-[P_0+P_1-2\infty]$$

This means

$$[R_1+R_2+R_3+R_4+P_0+P_1-6\infty]=0$$

The Abel Jacobi theorem involves the existence of a function F such that

$$div(F)=R_1+R_2+R_3+R_4+P_0+P_1-6\infty.$$

So $F\in\mathscr{L}(6\infty)$, hence $F(x,y)=u+vx+wx^2+dx^3+ey$ $(e\neq 0)$. We have $F(P_1)=F(P_0)=0$, so $u-e=0$ et $u-v+w-d=0$, then

$$F(x,y)=u+(u+w-d)x+wx^2+dx^3+uy \quad u\neq 0.$$

At the points R_i, we have $y=1+(1+a-c)x+ax^2+cx^3$ with $a=-\frac{w}{u}$ and $c=-\frac{d}{u}$. By substituting y in $y^2-x^5-1=0$ and simplifying by $x(x+1)$, we have

$$B_1(x)=c^2x^4+\left(2ac-c^2-1\right)x^3+\left(a^2-c^2+2c+1\right)x^2+\left(a^2+(2-2c)a+c^2-1\right)x+2a-2c+2=0$$

We must have $B_1(0)\neq 0$ and $B_1(-1)\neq 0$ involving $a\neq c-1$ and $c\neq 0$. We get a family of quartic points

$$\mathscr{G}_{1,2}=\left\{\ \left(x,\ -(-1+(-1-a+c)x-ax^2-cx^3)\right)\mid a,c\in\mathbb{Q},a\neq c-1,c\neq 0,x \text{ root of } B_1(x)=0\ \right\}.$$

Finally, we get a second family of quartic points $\mathscr{G}_1=\mathscr{G}_{1,1}\cup\mathscr{G}_{1,2}$.

<u>Cases $m=2$ and $m=8$</u>

For $m=2$: the formula $(\star)$ becomes

$$[R_1+R_2+R_3+R_4-4\infty]=-8D=-\left[2\overline{P_1}-2\infty\right]$$

This means

$$\left[R_1+R_2+R_3+R_4+2\overline{P_1}-6\infty\right]=0$$

The Abel Jacobi theorem involves the existence of a function F such that

$$div(F)=R_1+R_2+R_3+R_4+2\overline{P_1}-6\infty$$

So $F\in\mathscr{L}(6\infty)$, hence $F(x,y)=a+bx+cx^2+dx^3+ey$ $(e\neq 0)$. The point $\overline{P}_1$ is order 2, so $u-e=0$ and $v=0$, thus

$$F(x,y)=u+wx^2+dx^3+uy$$

At the points R_i, we have $-uy=u+wx^2+dx^3$ $(u\neq 0)$, so $y=-1+ax^2+cx^3$ with $a=-\frac{w}{u}$ and $k=-\frac{d}{u}$. Substuting y to $y^2=x^5+1$, we have

$$x^2\left(a^2x^4+2acx^3-x^3+a^2x^2-2cx-2a\right)=0.$$

Simplifying by x^2, we have

$$B_2(x)=c^2x^4+2acx^3-x^3+a^2x^2-2cx-2a.$$

We must have $ac\neq 0$ and $a\neq c+1$. We obtain a family of quartic points :

$$\mathscr{G}_{2,1}=\left\{\left(x,\ \left(cx^3+ax^2-1\right)\right)\ \mid\ a,c\in\mathbb{Q}^*,a\neq c+1,x \text{ root of } B_2(x)=0\right\}.$$

For $m=8$: by a similar argument as in case $m=2$, we have

$$\mathscr{G}_{2,2}=\left\{\left(x,\ -\left(cx^3+ax^2-1\right)\right)\ \mid\ a,c\in\mathbb{Q}^*,a\neq c+1,x \text{ root of } B_2(x)=0\right\}.$$

Finally, we have the third family $\mathscr{G}_2=\mathscr{G}_{2,1}\cup\mathscr{G}_{2,2}$.

Cases $m=3$ and $m=7$

For $m=3$: the formula $(\star)$ becomes

$$[R_1+R_2+R_3+R_4-4\infty]=-7D=-\left[\overline{Q_1}+\overline{Q_2}-2\infty\right]$$

This means

$$\left[R_1+R_2+R_3+R_4+\overline{Q_1}+\overline{Q_2}-6\infty\right]=0$$

The Abel Jacobi theorem involves the existence of a function F such that

$$div(F)=R_1+R_2+R_3+R_4+\overline{Q_1}+\overline{Q_2}-6\infty.$$

Then $F(x,y)=u+vx+wx^2+dx^3+ey$ $(e\neq 0)$. We have $F\left(\overline{Q_1}\right)=F\left(\overline{Q_2}\right)=0$, so $u+v-2d-e=0$ and $v+2w+2d+2e=0$, hence

$$F(x,y)=2w+4d+3e+(-2w-2d-2e)x+wx^2+dx^3+ey\ (e\neq 0).$$

At points R_i, we have $y=(-3-2a-4c)+(2+2a+2c)x-ax^2-cx^3$ with $a=\frac{w}{e}$ and $c=\frac{d}{e}$. Substuting y into $y^2=x^5+1$ and simplifying by x^2-2x+2, we have

$$B_3(x)=c^2x^4+\left(2c^2+2ac-1\right)x^3+\left(-2c^2-4c+a^2-2\right)x^2+\left((-4a-2)c-2a^2-4a-2\right)x+8c^2+(8a+12)c+2a^2+6a+4=0.$$

We must have $c\neq 0$ and $a\neq -1-2c$. We get a family of quartic points

$$\mathscr{G}_{3,1}=\left\{\ \left(x,\ (-3-2a-4c+(2+2a+2c)x-ax^2-cx^3)\right)\mid a,c\in\mathbb{Q}\, c\neq 0,\ a\neq -1-2c,\ x \text{ root of } B_3(x)=0\ \right\}$$

For $m=7$: by a similar argument as in previous case, we get a family of quartic points

$$\mathscr{G}_{3,2}=\left\{\ \left(x,\ -(-3-2a-4c+(2+2a+2c)x-ax^2-cx^3)\right)\mid a,c\in\mathbb{Q}\ c\neq 0,\ a\neq -1-2c,\ x \text{ root of } B_3(x)=0\ \right\}$$

Therefore, we have the fourth family $\mathscr{G}_3=\mathscr{G}_{3,1}\cup\mathscr{G}_{3,2}$.

Cases $m=4$ and $m=6$

For $m=4$: it exists a fonction F such that $div(F)=R_1+R_2+R_3+R_4+P_1-5\infty$, hence $F\in\mathscr{L}(5\infty)$,

$$F(x,y)=u+vx+wx^2+dy\quad(d\neq 0).$$

We have $F(P_1)=0$, therefore $u+d=0$, then $F(x,y)=u+vx+wx^2-uy$, $(u\neq 0)$. At points R_i, we have $y=1+ax+cx^2$. Substiting y to $y^2=x^5+1$, we have

$$x\left(x^4+c^2x^3+2acx^2+\left(2c+a^2\right)x+2a\right)=0.$$

Simplifiying by x, we have the minimal polynomial

$$B_4(x)=x^4+c^2x^3+2acx^2+\left(2c+a^2\right)x+2a=0.$$

We must have $a\neq 0$. We obtain a family of quartic points :

$$\mathscr{G}_{4,1}=\left\{\left(x,\ +(1+ax+cx^2)\right)\mid a,c\in\mathbb{Q},\ a\neq 0,\ x \text{ root of } B_4(x)=0\right\}.$$

For $m=6$: by a similar argument as in previous case, we get a family of quartic points :

$$\mathscr{G}_{4,2}=\left\{\left(x,\ -(1+ax+cx^2)\right)\mid a,c\in\mathbb{Q},\ a\neq 0,\ x \text{ root of } B_4(x)=0\right\}$$

Therefore, we have the firth family : $\mathscr{G}_4=\mathscr{G}_{4,1}\cup\mathscr{G}_{4,2}$.

Case $m=5$

It exists F such that $div(F)=R_1+R_2+R_3+R_4+P_0-5\infty$, so $F\in\mathscr{L}(5\infty)$, then

$$F(x,y)=u+vx+wx^2+dy\quad(d\neq 0).$$

We have $F(P_0)=0$, so $v=u+w$, therefore $F(x,y)=u+(u+w)x+wx^2+dy$. At points R_i, we have $y=-a+(-a-c)x-cx^2$ with $a=\frac{u}{d}$ and $c=\frac{w}{d}$. Substiting y to $y^2=x^5+1$, we have

$$(x+1)\left(x^4+\left(c^2+1\right)x^3+\left(c^2+2ac-1\right)x^2+\left(2ac+a^2+1\right)x+a^2-1\right)=0.$$

Simplifliying by $x+1$, we have the polynomial

$$B_5(x)=x^4+\left(c^2+1\right)x^3+\left(c^2+2ac-1\right)x^2+\left(2ac+a^2+1\right)x+a^2-1.$$

We must have $a\neq\pm 1$, therefore, we have the fifth family :

$$\mathscr{G}_5=\left\{\left(x,\ (-a+(-a-l)x-cx^2)\right)\mid a,c\in\mathbb{Q},\ a\neq\pm 1,\ x \text{ root of } B_5(x)=0\right\}.$$

References

[1] N. Bruin, M. Stoll, *The Mordell-Weil sieve : proving the nonexistence of Rational points on curves*, LMS J. Comp. Math., **13** (2010), 272 -306.
[2] R. F. Coleman, *Effective Chabauty* Duke Math. J. **52**(3) (1985), 765-770.
[3] E. F. Schaefer, *Computing a Selmer group of a Jacobian using functions on the curve*, Mathematische Annalen, **310** (1998), 447–471.
[4] M. J. Klassen, E. F. Schaefer, *Arithmetic and geometry of the curve* $x^4 = y^3 + 1$, Acta Arithmetica LXXIV.**3** (1996) 241-257.
[5] M. Fall, O. Sall, *Ponts algébriques de petit degré sur la courbe d'équation affine* $y^2 = x^5 + 1$, Afr. Mat. **29** (2018) 1151-1157.
[6] O. Sall, *Points algébriques sur certains quotients de courbes de Fermat*, C. R. Acad. Sci. Paris Sér I **336** (2003) 117-120.
[7] S. Siksek, M. Stoll, *Partial descent on hyper elliptic curves and the generalized Fermat equation* $x^3 + y^4 + z^5 = 0$, Bulletin of the LMS **44** (2012) 151 -166

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Threshold and Stability Results of a New Mathematical Model for Infectious Diseases Having Effective Preventive Vaccine

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Abstract

This paper evaluates the impact of an effective preventive vaccine on the control of some infectious diseases by using a new deterministic mathematical model. The model is based on the fact that the immunity acquired by a fully effective vaccination is permanent. Threshold $\mathscr{R}_0$, defined as the basic reproduction number, is critical indicator in the extinction or spread of any disease in any population, and so it has a very important role for the course of the disease that caused to an epidemic. In epidemic models, it is expected that the disease becomes extinct in the population if $\mathscr{R}_0 < 1$. In addition, when $\mathscr{R}_0 < 1$ it is expected that the disease-free equilibrium point of the model, and so the model, is stable in the sense of local and global. In this context, the threshold value $\mathscr{R}_0$ regarding the model is obtained. The local asymptotic stability of the disease-free equilibrium is examined with analyzing the corresponding characteristic equation. Then, by proved the global attractivity of disease-free equilibrium, it is shown that this equilibria is globally asymptotically stable.

1. Introduction

It has been seen that epidemics have had major effects and leave deep remains on human lives throughout history. To prevent and control the spread of epidemic, the examination of its' dynamics has an important role. In this context, mathematical modelling in epidemiology provides understanding and explanation of the underlying mechanisms that influenced the spread of disease, and it suggests control strategies. The COVID-19 pandemic, which emerged at the end of 2019 and is the most devastating epidemic of recent times, has seriously shaken humanity as a global threat. Modeling and analysis studies in mathematical epidemiology have focused on this ground in conjunction with this compelling and exhausting epidemic and many authors have made various contributions to this field with the help of the models they have constituted. [1–3] and references therein are among some current studies on this subject.

While dealing with mathematical modeling spread of disease, in order to formulate the transmissions of an epidemic, the population in a region is often divided into different compartments, and the models, which formulate the relations between these compartments, are called as compartmental models. In the literature, there are many compartmental models provided basic principles for the spread of a disease in a population. Kermack and McKendrick with their study [4] have pioneered in studies using compartmental mathematical models. In the model proposed by Kermack and McKendrick in 1927, the population was divided into three compartments: a susceptible compartment labelled S, in which all individuals are susceptible to the disease; an infected compartment labelled I, in which all individuals are infected by the disease and have infectivity; and a removed compartment labelled R, in which there exist the individuals consist whose infectiousness finished. This model is called as "SIR model" based on the initials of the group names.

Then, a lot of authors have tackled with various details to carry further forward this model. Adding a vaccination compartment is just one of these details. Immunization with vaccines is among the most effective methods of protection from infectious diseases which are common in the society and which have high contamination properties. Until today, many studies including the epidemic models with vaccination have been introduced. The references [5] and [6] are just two of them.

In mathematical epidemiology, the course of the disease in the population associates with whether the basic reproduction number is greater than 1, or not. It can be made the comments "If $\mathscr{R}_0 > 1$, there is an increase in the speed of the spread; if $\mathscr{R}_0 < 1$, there is a decrease in the epidemic rate and the epidemic is under control; if $\mathscr{R}_0 = 1$, speed of the spread is constant". The value $\mathscr{R}_0$ is very important since it can tell us whether the population is at risk about the disease. Therefore, calculating this value for any disease in any population is unvaluable.

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Considering the unfavorable conditions brought on by an epidemic disease in a population, studies to reduce the spread of the disease have, of course, great significance. In order that the disease dies out in the population, it needs that $\mathscr{R}_0 < 1$ and additionally the disease-free equilibrium point of the projected model is stable when $\mathscr{R}_0 < 1$. In other words, the effort required to prevent an outbreak or to eliminate an infection in a population should be directed towards ensuring that the value $\mathscr{R}_0$ is less than 1. In addition, it is expected that the disease-free equilibrium point of the model and so the model is stable in the sense of locally and globally, when $\mathscr{R}_0 < 1$.
Hence, for the past decades, many studies have been proposed on local and global stabilities of the disease-free equilibria, [7–14].
The aim of this study is to present a model that reflects the fact that the protective effect may not occur immediately after the vaccine is administered and the fact that this protection may not be fully effective even if the protective effect of the vaccine has started. The model uses a system of nonlinear ordinary integro-differential equations with delay to explain this fact. The study focuses on the basic reproduction number of this new compartmental epidemic model and on the local and global stabilities of the disease-free equilibria of model.

2. Model Description and Assumptions

In this study in which we aim to propose and analyze a new *SVEIR* epidemic model with delay, we divide a population (N), among which some individuals are known to be exposed to infectious disease, into five classes shaped like Susceptible (S), Vaccinated (V), Exposed (E), Infectious (I) and Removed (R) individuals.
The transitions between the compartments are expressed by the following system:

$$\begin{aligned}
\frac{dS}{dt} &= b-\beta_1 S(t)I(t)-qS(t)-\mu S(t),\\
\frac{dV}{dt} &= qS(t)-q\beta_2 I(t)\int_0^h e^{-\mu\tau}S(t-\tau)\,d\tau-\nu q\int_h^\infty e^{-\mu\tau}S(t-\tau)\,d\tau-(1-\nu)q\beta_3 I(t)\int_h^\infty e^{-\mu\tau}S(t-\tau)\,d\tau-\mu V(t),\\
\frac{dE}{dt} &= \beta_1 S(t)I(t)+q\beta_2 I(t)\int_0^h e^{-\mu\tau}S(t-\tau)\,d\tau+(1-\nu)q\beta_3 I(t)\int_h^\infty e^{-\mu\tau}S(t-\tau)\,d\tau-\gamma E(t)-\eta E(t)-\mu E(t),\\
\frac{dI}{dt} &= \gamma E(t)-\alpha I(t)-\delta I(t)-\mu I(t),\\
\frac{dR}{dt} &= \nu q\int_h^\infty e^{-\mu\tau}S(t-\tau)\,d\tau+\eta E(t)+\alpha I(t)-\mu R(t).
\end{aligned} \tag{2.1}$$

Where $S(t)$, $V(t)$, $E(t)$, $I(t)$ and $R(t)$ denote the numbers of susceptible, vaccinated, exposed, infectious, and removed individuals at time t, respectively. The total population size at time t is $N(t)$ and $N(t)=S(t)+V(t)+E(t)+I(t)+R(t)$ for all $t\geq 0$. Also all these functions are nonnegative.
All parameters belonging to the model are nonnegative constants and all newborn individuals get involved in the population by entering to the susceptible class with a constant rate b.
The effective contact rate between infectious individuals and susceptibles is β_1. Also β_2 is the effective contact rate between infectious and the individuals into the vaccinated group whose vaccinated at time $t-\tau$ and vaccine effect has not yet started. β_3 represents the effective contact rate between infectious and susceptibles whose vaccinated at time $t-\tau$ and vaccine effect has started, but whose their contribution rate to the protection provided by the vaccine less than 1.
We assume that q is the rate of vaccinated individuals within susceptible group and μ is natural death rate in each compartment, δ is death rate derived from pathogen causing to outbreak. γ indicates the rate at which exposed individuals become infectious. Also, α and η represent the transition rates from compartments consist of infectious and exposed individuals to the compartment R, respectively.
Active immunization, the way of activating the body's immune system through vaccination, is often applied before encountering with microorganisms, that is, before encountering contamination, in order to create antibodies / antitoxins against infectious diseases having high contagious properties and being severe consequences.
In immunization via vaccine, it requires a certain time (weeks or months) for antibody/antitoxin formation. In other words, protective effect (antibody/antitoxin) does not occur immediately after the vaccine is administered or after the first dose of the vaccine. We assume that the protective effect of the vaccine begins h time after vaccination. That is, protection of an individual who vaccinated at time t, begins at time $t+h$. So the term $q\int_0^h e^{-\mu\tau}S(t-\tau)\,d\tau$ indicates the number of individuals who have been vaccinated at time $t-\tau$ and their protection effect of vaccine has not yet started since the time threshold h does not completed, at time t. In this period that the vaccine does not yet form any effect, a vaccinated individual who have not yet any protection enters to compartment E if exposing to the infectious agent with a sufficiently effective contact with infectious individuals. This transition is expressed with the term $q\beta_2 I(t)\int_0^h e^{-\mu\tau}S(t-\tau)\,d\tau$ in the model.
It is also another fact that no vaccine has a 100% protective effect. In some individuals, the body's response to the vaccine may be weak because of various reasons. Therefore, the vaccine protection is lower for such individuals, and it can be seen that the protective efficacy in after vaccination is not fully formed. By considering this situation, the protection rate provided by the vaccine has been shown by ν in the model. As a result of effective contact between infectious individuals and the vaccinated (but are susceptible) individuals whose protection provided by the vaccine is less than 1, enter to latent compartment by exposing to the infectious agent. This transition is denoted by $(1-\nu)q\beta_3 I(t)\int_h^\infty e^{-\mu\tau}S(t-\tau)\,d\tau$.
On the other hand the individuals, whose protection level provided by the vaccine is 1 and so have full protection, include to the class R by gaining immunity. This transition is represented by the term $\nu q\int_h^\infty e^{-\mu\tau}S(t-\tau)\,d\tau$.

2.1. Qualitative Analysis of the Model

In this section, we find the feasible positive invariant region, equilibrium points and basic reproduction number belong to the model.

2.1.1. Feasible Positive Invariant Region

Theorem 2.1. *The set*

$$\Upsilon=\left\{S\in C\left([-\tau,\infty),\left[0,\frac{b}{\mu}\right]\right),\ (V,E,I,R)\in C\left(\mathbb{R}_+,\left[0,\frac{b}{\mu}\right]\right):N(t)\leq\frac{b}{\mu}\right\} \tag{2.2}$$

is positively invariant and bounded for the model.

Proof. Summing up the five equations of system (2.1), we obtain

$$\begin{aligned} N'(t) &= \frac{dS}{dt}+\frac{dV}{dt}+\frac{dE}{dt}+\frac{dI}{dt}+\frac{dR}{dt} \\ &= b-\mu\left(S(t)+V(t)+E(t)+I(t)+R(t)\right)-\delta I(t) \\ &\leq b-\mu N(t). \end{aligned} \tag{2.3}$$

For the solution of this nonlinear differential inequality, we investigate the differential equation

$$N'(t)+\mu N(t)=b.$$

From solving of this ordinary differential equation we get $N(t)=b/\mu+ce^{-\mu t}$. For the initial condition $t=0$, we find the solution as

$$N(t)=N(0)e^{-\mu t}+\frac{b}{\mu}\left(1-e^{-\mu t}\right). \tag{2.4}$$

The right side of the equality (2.4) is the maximal solution of (2.3) by Standard Comparison Theorem [15]. Hence we have the inequality

$$N(t)\leq N(0)e^{-\mu t}+\frac{b}{\mu}\left(1-e^{-\mu t}\right)$$

for every $t\geq 0$. If $N(0)\leq b/\mu$ then $N(t)\leq b/\mu$ for all $t>0$ and so the set Υ given by (2.2) is positively invariant for the system (2.1). Also, it is clearly seen that $N(t)$ is bounded above with b/μ.
This region can be considered as a feasible bounded region which is enough to study epidemiologically and mathematically. □

Since the population $V(t)$ and $R(t)$ do not feature in remainder equations of (2.1), we can study on the following reduced system (2.5):

$$\begin{aligned} S'(t) &= b-\beta_1 S(t)I(t)-(q+\mu)S(t), \\ E'(t) &= \beta_1 S(t)I(t)+q\beta_2 I(t)\int_0^h e^{-\mu\tau}S(t-\tau)\,d\tau+(1-\nu)q\beta_3 I(t)\int_h^\infty e^{-\mu\tau}S(t-\tau)\,d\tau-(\gamma+\eta+\mu)E(t), \\ I'(t) &= \gamma E(t)-(\alpha+\delta+\mu)I(t). \end{aligned} \tag{2.5}$$

2.1.2. Disease-Free Equilibrium Point

Now, let us find the disease-free equilibrium point of the model (2.5). To do this we take $S(t)=S_0$, $V(t)=V_0$, $E(t)=E_0$ and $I(t)=I_0=0$. So, for the system of algebraic equations

$$\begin{aligned} 0 &= b-\beta_1 S_0 I_0-(q+\mu)S_0, \\ 0 &= \beta_1 S_0 I_0+q\beta_2 S_0 I_0\int_0^h e^{-\mu\tau}d\tau+(1-\nu)q\beta_3 S_0 I_0\int_h^\infty e^{-\mu\tau}d\tau-(\gamma+\eta+\mu)E_0, \\ 0 &= \gamma E_0-(\alpha+\delta+\mu)I_0, \end{aligned}$$

the disease-free equilibrium point is obtained as

$$P_{DF}=(S_0,E_0,I_0)=\left(\frac{b}{q+\mu},0,0\right). \tag{2.6}$$

2.1.3. Basic Reproduction Number

As is known from mathematical epidemiology, the basic reproduction number $\mathscr{R}_0$ is used to measure the transmission potential of a disease. It is the average number of secondary infections produced by a typical case of an infection in a population in which everyone is susceptible, [16].
In the following part, we calculate this critical threshold value $\mathscr{R}_0$ for the model with the help of next generation matrix method, [17].
Let us take $X=(E,I,S)^T$ and write the system (2.5) in the form

$$\underbrace{\begin{bmatrix}\dot{E}\\ \dot{I}\\ \dot{S}\end{bmatrix}}_{\frac{dX}{dt}} = \underbrace{\begin{bmatrix}\beta_1 S(t)I(t)+q\beta_2 I(t)\int_0^h e^{-\mu\tau}S(t-\tau)\,d\tau+(1-\nu)q\beta_3 I(t)\int_h^\infty e^{-\mu\tau}S(t-\tau)\,d\tau\\ 0\\ 0\end{bmatrix}}_{\mathscr{K}(X)} - \underbrace{\begin{bmatrix}(\gamma+\eta+\mu)E(t)\\ (\alpha+\delta+\mu)I(t)-\gamma E(t)\\ \beta_1 S(t)I(t)+(q+\mu)S(t)-b\end{bmatrix}}_{\mathscr{V}(X)},$$

that is

$$\frac{dX}{dt}=\mathscr{K}(X)-\mathscr{V}(X).$$

The values at the disease-free equilibrium point P_{DF} of the derivatives of $\mathscr{K}(X)$ and $\mathscr{V}(X)$ with respect to E, I, S, respectively, come in sight with the following Jacobian matrices:

$$J_{\mathscr{K}}(P_{DF})=\begin{bmatrix}0 & J^{12}_{\mathscr{K}}(P_{DF}) & J^{13}_{\mathscr{K}}(P_{DF})\\ 0 & 0 & 0\\ 0 & 0 & 0\end{bmatrix}$$

such that

$$J^{12}_{\mathscr{K}}(P_{DF})=\beta_1 S_0+q\beta_2 S_0\int_0^h e^{-\mu\tau}d\tau+(1-\nu)q\beta_3 S_0\int_h^\infty e^{-\mu\tau}d\tau$$

and

$$J^{13}_{\mathscr{K}}(P_{DF})=\beta_1 I_0+q\beta_2 I_0\int_0^h e^{-\mu\tau}d\tau+(1-\nu)q\beta_3 I_0\int_h^\infty e^{-\mu\tau}d\tau$$

and also

$$J_{\mathscr{V}}(P_{DF})=\begin{bmatrix}\gamma+\eta+\mu & 0 & 0\\ -\gamma & \alpha+\delta+\mu & 0\\ 0 & \beta_1 S_0 & \beta_1 I_0+(q+\mu)\end{bmatrix}.$$

Taking into account that infections can only exist in compartments E and I , the block matrices K and V are formed as

$$K=\mathscr{K}_{2x2}=\begin{bmatrix}0 & K_{12}\\ 0 & 0\end{bmatrix}$$

such that

$$K_{12}=\beta_1 S_0+q\beta_2 S_0\int_0^h e^{-\mu\tau}d\tau+(1-\nu)q\beta_3 S_0\int_h^\infty e^{-\mu\tau}d\tau,$$

and

$$V=\mathscr{V}_{2x2}=\begin{bmatrix}\gamma+\eta+\mu & 0\\ -\gamma & \alpha+\delta+\mu\end{bmatrix}.$$

Thus

$$KV^{-1}=\begin{bmatrix}KV_{11}^{-1} & KV_{12}^{-1}\\ 0 & 0\end{bmatrix}$$

such that

$$KV_{11}^{-1}=\frac{\gamma\left(\beta_1 S_0+q\beta_2 S_0\int\limits_0^h e^{-\mu\tau}d\tau+(1-\nu)q\beta_3 S_0\int\limits_h^\infty e^{-\mu\tau}d\tau\right)}{(\gamma+\eta+\mu)(\alpha+\delta+\mu)}$$

and

$$KV_{12}^{-1}=\frac{\beta_1 S_0+q\beta_2 S_0\int\limits_0^h e^{-\mu\tau}d\tau+(1-\nu)q\beta_3 S_0\int\limits_h^\infty e^{-\mu\tau}d\tau}{\alpha+\delta+\mu}.$$

So the characteristic polynomial of KV^{-1} appears with

$$\lambda\left(\lambda-\frac{\gamma\left(\beta_1 S_0+q\beta_2 S_0\int\limits_0^h e^{-\mu\tau}d\tau+(1-\nu)q\beta_3 S_0\int\limits_h^\infty e^{-\mu\tau}d\tau\right)}{(\gamma+\eta+\mu)(\alpha+\delta+\mu)}\right)=0.$$

Thus, the spectral radius of the next generation matrix is

$$\rho\left(KV^{-1}\right)=\frac{\gamma\left(\beta_1 S_0+q\beta_2 S_0\int\limits_0^h e^{-\mu\tau}d\tau+(1-\nu)q\beta_3 S_0\int\limits_h^\infty e^{-\mu\tau}d\tau\right)}{(\gamma+\eta+\mu)(\alpha+\delta+\mu)}.$$

Taking into

$$S_0=\frac{b}{q+\mu},\qquad \int\limits_0^h e^{-\mu\tau}d\tau=\frac{1-e^{-\mu h}}{\mu},\qquad \int\limits_h^\infty e^{-\mu\tau}d\tau=\lim_{k\to\infty}\int\limits_h^k e^{-\mu\tau}d\tau=\frac{e^{-\mu h}}{\mu}$$

account that, the basic reproduction number of the system (2.5) is calculated in the form of

$$\begin{aligned}\mathscr{R}_0 &= \rho\left(KV^{-1}\right)=\frac{b\gamma\left(\beta_1+q\beta_2\frac{1-e^{-\mu h}}{\mu}+(1-\nu)q\beta_3\frac{e^{-\mu h}}{\mu}\right)}{(q+\mu)(\gamma+\eta+\mu)(\alpha+\delta+\mu)}\\ &= \frac{b\gamma\left(\mu\beta_1+q\beta_2\left(1-e^{-\mu h}\right)+(1-\nu)q\beta_3 e^{-\mu h}\right)}{\mu(q+\mu)(\gamma+\eta+\mu)(\alpha+\delta+\mu)}.\end{aligned}$$

2.1.4. Existence and Uniqueness of Endemic Equilibrium Point

In subsection 2.1.2, we see that the system (2.5) always has a disease-free equilibrium point. Now, we investigate the existence and uniqueness of endemic equilibrium point. If the constant solution $P_E(S^*,E^*,I^*)$ is the endemic equilibrium of (2.5), the positive constants S^*, E^* and I^* should satisfy the algebraic equations

$$\begin{aligned}0 &= b-\beta_1 S^*I^*-(q+\mu)S^*,\\ 0 &= \beta_1 S^*I^*+q\beta_2 S^*I^*\int\limits_0^h e^{-\mu\tau}d\tau+(1-\nu)q\beta_3 S^*I^*\int\limits_h^\infty e^{-\mu\tau}d\tau-(\gamma+\eta+\mu)E^*,\\ 0 &= \gamma E^*-(\alpha+\delta+\mu)I^*.\end{aligned}\tag{2.7}$$

From second equation of this system, we have

$$S^*I^*\left(\beta_1+q\beta_2\frac{1-e^{-\mu h}}{\mu}+(1-\nu)q\beta_3\frac{e^{-\mu h}}{\mu}\right)=(\gamma+\eta+\mu)E^*$$

and then

$$\frac{E^*}{I^*}=\frac{\left(\mu\beta_1+q\beta_2\left(1-e^{-\mu h}\right)+(1-\nu)q\beta_3 e^{-\mu h}\right)S^*}{\mu(\gamma+\eta+\mu)}.\tag{2.8}$$

On the other hand, from third equation of the system (2.7), we write

$$\frac{E^*}{I^*}=\frac{\alpha+\delta+\mu}{\gamma}.\tag{2.9}$$

Considering (2.8) and (2.9), we get

$$S^*=\frac{\mu(\gamma+\eta+\mu)(\alpha+\delta+\mu)}{\gamma\left(\mu\beta_1+q\beta_2\left(1-e^{-\mu h}\right)+(1-\nu)q\beta_3 e^{-\mu h}\right)}.$$

By the way, let us express the S^* in terms of $\mathscr{R}_0$.

$$S^* = \frac{b}{(q+\mu)\mathscr{R}_0}.$$

Also, from first equation of the system (2.7)

$$\begin{aligned} I^* &= \frac{b-(q+\mu)S^*}{\beta_1 S^*} \\ &= \frac{(q+\mu)(\mathscr{R}_0-1)}{\beta_1} \end{aligned}$$

and taking into account (2.9) we obtain

$$\begin{aligned} E^* &= \frac{(\alpha+\delta+\mu)I^*}{\gamma} \\ &= \frac{(q+\mu)(\alpha+\delta+\mu)(\mathscr{R}_0-1)}{\gamma\beta_1}. \end{aligned}$$

Therefore, we say that the system (2.5) has a unique endemic equilibrium point

$$\begin{aligned} P_E &= (S^*, E^*, I^*) \\ &= \left(\frac{b}{(q+\mu)\mathscr{R}_0}, \frac{(q+\mu)(\alpha+\delta+\mu)(\mathscr{R}_0-1)}{\gamma\beta_1}, \frac{(q+\mu)(\mathscr{R}_0-1)}{\beta_1}\right) \end{aligned}$$

for $\mathscr{R}_0 > 1$.

2.2. Local and Global Stabilities of Disease-free Equilibria

In this part, local and global stability of disease-free equilibrium point P_{DF} of the model (2.5) are discussed.

Theorem 2.2. *The disease-free equilibrium point P_{DF} is locally asymptotically stable in Υ for $\mathscr{R}_0 < 1$.*

Proof. The Jacobian matrix at $P_{DF} = (S_0, E_0, I_0)$ of the system (2.5) is

$$J(P_{DF}) = \begin{bmatrix} -\beta_1 I_0 - (q+\mu) & 0 & -\beta_1 S_0 \\ J_{21}(P_{DF}) & -(\gamma+\eta+\mu) & J_{23}(P_{DF}) \\ 0 & \gamma & -(\alpha+\delta+\mu) \end{bmatrix}$$

where

$$J_{21}(P_{DF}) = \beta_1 I_0 + q\beta_2 I_0 \int_0^h e^{-\mu\tau} d\tau + (1-\nu) q\beta_3 I_0 \int_h^\infty e^{-\mu\tau} d\tau$$

and

$$J_{23}(P_{DF}) = \beta_1 S_0 + q\beta_2 S_0 \int_0^h e^{-\mu\tau} d\tau + (1-\nu) q\beta_3 S_0 \int_h^\infty e^{-\mu\tau} d\tau.$$

For the point $(S_0, E_0, I_0) = \left(\frac{b}{q+\mu}, 0, 0\right)$, $J(P_{DF})$ corresponds to the following form

$$J(P_{DF}) = \begin{bmatrix} -(q+\mu) & 0 & -\frac{b\beta_1}{q+\mu} \\ 0 & -(\gamma+\eta+\mu) & L \\ 0 & \gamma & -(\alpha+\delta+\mu) \end{bmatrix},$$

where

$$L = \frac{b\left(\mu\beta_1 + q\beta_2\left(1-e^{-\mu h}\right) + (1-\nu)q\beta_3 e^{-\mu h}\right)}{\mu(q+\mu)}.$$

The characteristic equation for this Jacobian matrix is

$$\begin{aligned} \det(J(P_{DF}) - \lambda I_3) &= (-(q+\mu)-\lambda) \begin{vmatrix} -(\gamma+\eta+\mu)-\lambda & L \\ \gamma & -(\alpha+\delta+\mu)-\lambda \end{vmatrix} \\ &= 0. \end{aligned}$$

Hence

$$(-(q+\mu)-\lambda)\left(\lambda^2+[(\gamma+\eta+\mu)+(\alpha+\delta+\mu)]\lambda+(\gamma+\eta+\mu)+(\alpha+\delta+\mu)-\gamma L\right)=0, \tag{2.10}$$

and so the characteristic equation (2.10) always have the negative root $-(q+\mu)$. The other eigenvalues of this equation come from the equation

$$\lambda^2+[(\gamma+\eta+\mu)+(\alpha+\delta+\mu)]\lambda+[(\gamma+\eta+\mu)+(\alpha+\delta+\mu)](1-\mathscr{R}_0)=0. \tag{2.11}$$

For this quadratic equation,

$$\lambda_1\lambda_2=[(\gamma+\eta+\mu)+(\alpha+\delta+\mu)](1-\mathscr{R}_0)$$

and

$$\lambda_1+\lambda_2=-(\gamma+\eta+\mu)-(\alpha+\delta+\mu)<0.$$

If $\mathscr{R}_0<1$ then $\lambda_1\lambda_2>0$ and so two roots of Eq. (2.11) are also negative. If $\mathscr{R}_0=1$ we say that one of roots of Eq.(2.11) is zero. On the other hand, when $\mathscr{R}_0>1$ one of roots of Eq. (2.11) has positive real parts. So the disease-free equilibrium P_{DF} is locally asymptotically stable for $\mathscr{R}_0<1$; is stable for $\mathscr{R}_0=1$, and is unstable for $\mathscr{R}_0>1$. □

To prove that the disease-free equilibrium point P_{DF} is globally asymptotically stable for $\mathscr{R}_0<1$, we use to the global attractivity of it. To see this property of the P_{DF}, we need the following lemma.

Lemma 2.3. *[18] Assume that f be a bounded, real-valued function defined on $[0,\infty)$ and be twice differentiable with bounded second derivative. Also, let us define notations f_∞ and f^∞ as*

$$f_\infty=\liminf_{t\to\infty}f(t),\qquad f^\infty=\limsup_{t\to\infty}f(t),$$

where

$$\begin{aligned}\inf f(t) &= \inf\{f(u):u\in[t,\infty),\ t>0\},\\ \sup f(t) &= \sup\{f(u):u\in[t,\infty),\ t>0\}.\end{aligned}$$

Letting $k\to\infty$, $t_k\to\infty$ and $f(t_k)$ converges to f_∞ and f^∞, then $\lim_{k\to+\infty}f'(t_k)=0$.

Theorem 2.4. *The disease-free equilibrium point P_{DF} is globally asymptotically stable in Υ for $\mathscr{R}_0<1$.*

Proof. We investigate that P_{DF} is globally attractive. From the first equation of the system (2.5) we write

$$\frac{dS}{dt}\le b-(q+\mu)S(t). \tag{2.12}$$

Let us say $\frac{dX}{dt}=b-(q+\mu)X(t)$. Clearly, a solution of the equation $\frac{dX}{dt}=b-(q+\mu)X(t)$ is a supper solution of $S(t)$. Therefore $X(t)\ge S(t)$ for every $t\ge0$. We immediately note that $X(t)\to\frac{b}{(q+\mu)}$ while $t\to\infty$. Then for a given $\varepsilon_S>0$, there exists a t_0 such that

$$S(t)\le X(t)\le\frac{b}{q+\mu}+\varepsilon_S$$

for $t\ge t_0$. So we say $S^\infty\le\frac{b}{q+\mu}+\varepsilon_S$. In the event of $\varepsilon_S\to0$, we write

$$S^\infty\le\frac{b}{q+\mu}. \tag{2.13}$$

From the third equation of the system (2.5) we obtain

$$I(t)\to\frac{\gamma\lim_{t\to\infty}E(t)}{\alpha+\delta+\mu}\le\frac{\gamma E^\infty}{\alpha+\delta+\mu},$$

as $t\to\infty$. So we can say

$$I^\infty\le\frac{\gamma E^\infty}{\alpha+\delta+\mu}. \tag{2.14}$$

Taking into account the inequation (2.13), from the second equation of the system (2.5) we write

$$\begin{aligned}E(t) &\to \frac{\beta_1S(t)I(t)}{\gamma+\eta+\mu}+\frac{q\beta_2I(t)\int_0^h e^{-\mu\tau}S(t-\tau)\,d\tau}{\gamma+\eta+\mu}+\frac{(1-\nu)q\beta_3I(t)\int_h^\infty e^{-\mu\tau}S(t-\tau)\,d\tau}{\gamma+\eta+\mu}\\ &\le \left(\frac{b\beta_1}{(q+\mu)(\gamma+\eta+\mu)}+\frac{bq\beta_2\left(1-e^{-\mu h}\right)}{\mu(q+\mu)(\gamma+\eta+\mu)}+\frac{b(1-\nu)q\beta_3e^{-\mu h}}{\mu(q+\mu)(\gamma+\eta+\mu)}\right)I^\infty\end{aligned}$$

as $t \to \infty$. So we get

$$E^{\infty} \leq \left(\frac{b\left(\mu\beta_1 + q\beta_2\left(1-e^{-\mu h}\right) + (1-\nu)\, q\beta_3 e^{-\mu h}\right)}{\mu\,(q+\mu)\,(\gamma+\eta+\mu)} \right) I^{\infty}. \tag{2.15}$$

Substituting the inequality (2.15) into (2.14), we obtain

$$\begin{aligned} I^{\infty} &\leq \left(\frac{b\gamma\left(\mu\beta_1 + q\beta_2\left(1-e^{-\mu h}\right) + (1-\nu)\, q\beta_3 e^{-\mu h}\right)}{\mu\,(q+\mu)\,(\gamma+\eta+\mu)\,(\alpha+\delta+\mu)} \right) I^{\infty} \\ &= \mathscr{R}_0 I^{\infty}. \end{aligned}$$

Also, substituting (2.14) into the inequality (2.15), we attain

$$\begin{aligned} E^{\infty} &\leq \left(\frac{b\gamma\left(\mu\beta_1 + q\beta_2\left(1-e^{-\mu h}\right) + (1-\nu)\, q\beta_3 e^{-\mu h}\right)}{\mu\,(q+\mu)\,(\gamma+\eta+\mu)\,(\alpha+\delta+\mu)} \right) E^{\infty} \\ &= \mathscr{R}_0 E^{\infty}. \end{aligned}$$

Hence if $\mathscr{R}_0 < 1$, we say that $I^{\infty} \leq 0$ and $E^{\infty} \leq 0$.
On the other hand, clearly $I_{\infty} \geq 0$ and $E_{\infty} \geq 0$. So $I_{\infty} = I^{\infty} = 0$ and $E_{\infty} = E^{\infty} = 0$. Therefore $(E(t), I(t)) \to (0,0)$ while $t \to \infty$.
Now we show that

$$\lim_{t\to\infty} S(t) = \frac{b}{q+\mu}.$$

According to Lemma 2.3, we can determine the sequences (r_n) and (s_n) such that

$$S(r_n) \to S_{\infty} \text{ and } S(s_n) \to S^{\infty}$$

while $(r_n) \to \infty$ and $(s_n) \to \infty$.
Again, in accordance with the same Lemma, we have

$$S'(r_n) \to 0 \text{ and } S'(s_n) \to 0. \tag{2.16}$$

From (2.12) and (2.16), we obtain

$$b - (q+\mu) \liminf_{t\to\infty} S(t) = 0$$

and

$$b - (q+\mu) \limsup_{t\to\infty} S(t) = 0.$$

Therefore $\lim_{t\to\infty} S(t) = \frac{b}{q+\mu}$. Consequently disease-free equilibrium point P_{DF} is global attractive for $\mathscr{R}_0 < 1$.
We have concluded that disease-free equilibrium point is locally asymptotically stable with previous theorem. Since P_{DF} is both locally asymptotically stable and globally attractive, it is globally asymptotically stable in Υ for $\mathscr{R}_0 < 1$. □

3. Conclusion

When it is carried out immunization with the vaccine, not only the vaccinated individual is protected against infectious disease, but also indirectly, it is prevented from infecting other individuals. Therefore, if the number of vaccinated individuals against the disease in the society is how much higher, the probability of the occurrence of that disease is lower at that rate. It is even possible to eliminate some diseases completely. For example, through successful vaccination programs, diseases such as smallpox, measles, polio have been completely eradicated or have been reduced to almost non-existent levels. This situation has increased the interest to models with vaccine in dynamical systems.
In this paper, it has been investigated the disease-free dynamics of a time delayed *SVEIR* epidemic model with a different perspective from the models in the literature. For the model it has been obtained the threshold quantity $\mathscr{R}_0$, called as the basic reproduction number. Next, as $\mathscr{R}_0 < 1$, it has been shown that disease-free equilibrium is locally asymptotically stable and is globally attractive, and as a result of this, is globally asymptotically stable. Vaccination always has a strong effect for disease control by decreasing the basic reproduction number. So, when $\mathscr{R}_0 < 1$,with effective, preventative and sustained vaccinations the disease can disappear ultimately.

References

[1] S. Çakan, *Dynamic analysis of a mathematical model with health care capacity for COVID-19 pandemic*, Chaos, Solitons and Fractals, **139**, 110033 (2020), 1-8.
[2] P. Kumar, V.S. Ertürk, *The analysis of a time delay fractional COVID-19 model via Caputo type fractional derivative*, Math Meth Appl Sci., (2020), 1-14. https://doi.org/10.1002/mma.6935
[3] Z. Zhang, A. Zeb, S. Hussain, E. Alzahrani, *Dynamics of COVID-19 mathematical model with stochastic perturbation*, Adv. Differ. Equ., **451** (2020), 1-12. https://doi.org/10.1186/s13662-020-02909-1
[4] W.O. Kermack, A.G. McKendrick, *A contributions to the mathematical theory of epidemics*, Proc. Roy. Soc. A, **115** (1927), 700-721.
[5] N.F. Tehrani, M.R. Razvan, S. Yasaman, *Global Analysis of a delay SVEIR epidemiological model*, Iran. J. Sci. Technol. Trans. A Sci., **37A4** (2013), 483-489.
[6] R. Xu, *Global stability of a delayed epidemic model with latent period and vaccination strategy*, Appl. Math. Model., **36** (2012), 5293–5300.

[7] A.A. Momoh, M.O. Ibrahim, B.A. Madu, *Stability Analysis of an infectious disease free equilibrium of Hepatitis B model*, Research Journal of Applied Sciences, Engineering and Technology, **3(9)** (2011), 905-909.
[8] C. Wu, Z. Jiang, *Global stability for the disease free equilibrium of a delayed model for malaria transmission*, Int. Journal of Math. Analysis, **6(38)** (2012), 1877-1881.
[9] C. Wu and Y. Zhang, *Stability Analysis for the disease free equilibrium of a discrete malaria model with two delays*, Intelligent Computing Theories and Applications. ICIC 2012, LNAI **7390** (2012), 341-349.
[10] R. Akbari, A.V. Kamyad, A.A. Heydari, A. Heydari, *The analysis of a disease-free equilibrium of hepatitis B model*, Sahand Communications in Mathematical Analysis (SCMA), **3(2)** (2016), 1-11.
[11] J.C. Kamgang, G. Sallet, *Computation of threshold conditions for epidemiological models and global stability of the disease-free equilibrium (DFE)*, Math. Biosci., **213** (2008), 1-12.
[12] J.C. Kamgang, G. Sallet, *Global asymptotic stability for the disease free equilibrium for epidemiological models*, Comptes Rendus Mathematique, **341(7)** (2005), 433-438.
[13] K.M. Bello, I.N. Akinwande, S. Abdurhman, A.F. Kuta, Y. Bello, *Global stability analysis of the disease-free equilibrium state of a mathematical model of trypanosomiasis*, J. Appl. Sci. Environ. Manage., **23(2)** (2019), 201-208.
[14] K.B. Blyuss, Y.N. Kyrychko. *Instability of disease-free equilibrium in a model of malaria with immune delay. Math Biosci.* **248** (2014), 54-6. doi: 10.1016/j.mbs.2013.12.005. Epub 2013 Dec 25. PMID: 24373861.
[15] S. Lakshmikantham, S. Leela, A.A. Martynyuk, *Stability Analysis of Nonlinear Systems*, Marcel Dekker, Inc., New York, 1989.
[16] P.V.D. Driessche, J. Watmough, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Math.Biosci., **180** (2002), 29-48.
[17] O. Diekmann, J.A.P. Heesterbeek, J.A.J Metz, *On the definition and the computation of the basic reproduction ratio $\mathscr{R}_0$ in models for infectious diseases in heterogeneous populations*, Journal of Mathematical Biology, **28(4)** (1990), 365-382.
[18] R.H. Thieme, Persitence under relaxed point dissipativity (with application to an endemic model), SIAM J. Math. Anal., **24** (1993), 407-435.

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On the Asymptotic Stability of the Nonlinear Difference Equation System

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Abstract

In this paper, we obtain some new results on the equi-boundedness of solutions and asymptotic stability for a class of nonlinear difference systems with variable delay of the form

$$x(n+1) = ax(n) + B(n)F(x(n-m(n))), \qquad n = 0,1,2,...$$

where F is the real valued vector function, $m : \mathbb{Z} \to \mathbb{Z}^+$, which is bounded function and maximum value of m is k and $B(n)$ is a $k \times k$ variable coefficient matrix. We carry out the proof of our results by using the Banach fixed point theorem and we use these results to determine the asymptotic stability conditions of an example.

1. Introduction

Nonlinear difference equations are the proper mathematical representation for discrete processes, which have remarkable importance in areas such as Nicholson's blowflies model, bobwhite quail population model and predator-prey models. Recently, many researchers have investigated asymptotic behaviour of the solutions of the nonlinear difference equations. Especially, Huong et al. [5,6,9,10,11,12,13,14] have done important studies in this area in the last 20 years. For instance Giang-Huong [5,6] obtained some new results for the asymptotic behaviour of solutions of nonlinear difference equations with time-invariant delay of the form

$$x(n+1) = \lambda x(n) + F(x(n-m)) \quad n = 0,1,...,$$

where $F : [0,\infty) \to [0,\infty)$ is a continuous function, $m \geq 0$ is a fixed integer and $\lambda \in (0,1)$. In addition, Huong [10] studied nonlinear difference equation with bounded multiple delay of the form

$$x(n+1) = \lambda(n)x(n) + \sum_{i=1}^{r} \alpha_i(n)F(x(n-m(i))), \tag{1.1}$$

where $n \in \mathbb{N}$ with $n \geq a$ for some $a \in \mathbb{N}$, where $r, m(i) \geq 1$, $1 \leq i \leq r$ are fixed positive integers, the functions α_i are defined on $\mathbb{N}$ and the functions F are defined on $\mathbb{R}$. Also, Huong [11] obtained some oscillatory results for equation (1.1). Huong and Nam [12] investigated the oscillation, convergence and boundedness of solution of some nonlinear difference equations with multiple delay of the form

$$x(n+1) = G(x(n), x(n-m_1), ..., x(n-m_r)) \quad n = 0,1,...,$$

where $m_i \in \mathbb{N}_0$, $\forall i = 1,...,r$ and the function $G : \mathbb{R}^{m+1} \to \mathbb{R}$. After, Huong and Mau [9] studied on the stability of the zero of autonomous nonlinear difference equation with variable delay of the form

$$x(n+1) = \lambda(n)x(n) + \alpha(n)F(x(n-m(n))) \quad n = 0,1,...$$

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where the functions λ, α are defined on the set of integers, the function m maps the set of integers to the set of positive integers, the function F is defined on the set of real numbers. Moreover, Huong [14] gave the some new results stability and strict boundedness conditions of non-autonomous nonlinear difference equation with time-varying delay of the form

$$x(n+1) = \lambda(n)x(n) + \alpha(n)F(n, x(n-m(n))) \quad n = 0,1,...$$

In addition to Huong's investigations, in the last century, there has been many literature on stability of delay difference equations. In particular, as examples of studies, Győri, Ladas and Vlahos [8], Chen and Liu [2], Graef and Qian [7], Edwards and Neville [4], Rao and Sudha [18], Asiri and Elsayed [19], Değer and Bolat [20], Muroya, Ishiwata and Guglielmi [16], Akgul, Inc and Karatas [22], Akgul [23], Zhang [21], Liao [15] can be given. In this study, we obtain some new results on the equi-boundedness of solutions and asymptotic stability for a class of nonlinear difference systems with variable delay of the form

$$x(n+1) = ax(n) + B(n)F(x(n-m(n))), \quad n = 0,1,2,... \tag{1.2}$$

where F is the real valued vector function, $m : \mathbb{Z} \to \mathbb{Z}^+$, which is bounded function and maximum value of m is k and $B(n)$ is a $k \times k$ variable coefficient matrix. Let $\mathbb{Z}_0$ be as a set of integers belonging to the interval $[n_0 - k, n_0]$ for each integer $n_0 \geq 0$ (if m is unbounded, then $\mathbb{Z}_0$ as a set of integers belonging to the interval $(-\infty, n_0]$). Also, let $\phi : \mathbb{Z}_0 \to \mathbb{R}^k$ be an initial discrete bounded vector function. Now, we can give some definitions and proposition for the proof of the Lemma and Theorems.

Definition 1.1. *If $x(n) = \phi(n)$ on $\mathbb{Z}_0$ and satisfies (1.2) for $n \geq n_0$, then $x(n) = x(n, n_0, \phi)$ is a solution of (1.2)* [9].

Definition 1.2. *If for any $\varepsilon > 0$ and any integer $n_0 \geq 0$ there exists a $\delta(n_0, \varepsilon) > 0$ such that $\|\phi(n)\| \leq \delta$ on $\mathbb{Z}_0$ implies*

$$\|x(n, n_0, \phi)\| \leq \varepsilon \text{ for } n \geq n_0,$$

then the zero solution of (1.2) is Liapunov stable [9].

Definition 1.3. *If the zero solution of (1.2) is Liapunov stable and if for any integer $n_0 \geq 0$ there exists $\mu(n_0) > 0$ such that $\|\phi(n)\| \leq \mu(n_0)$ on $\mathbb{Z}_0$ implies*

$$\|x(n, n_0, \phi)\| \to 0 \text{ as } n \to \infty,$$

then the zero solution of (1.2) is asymptotically stable [9].

Definition 1.4. *If there exists a $B(n_0, \phi) > 0$ such that $\|x(n, n_0, \phi)\| \leq B(n_0, \phi)$ for $n \geq n_0$, then a solution $x(n) = x(n, n_0, \phi)$ of (1.2) is said to be bounded* [9].

Definition 1.5. *If for any n_0 and any $B_1 > 0$ there exists $B_2 = B_2(n_0, B_1) > 0$ such that $\phi(n) \leq B_1$ on $\mathbb{Z}_0$ implies*

$$\|x(n, n_0, \phi)\| \leq B_2 \text{ for } n \geq n_0,$$

then solutions of (1.2) is equi-bounded [9].

Definition 1.6. *A Banach space is a complete, normed, vector space.*

Definition 1.7. *Let $P : X \to X$ be a mapping from a set X to itself. We call a point $x \in X$ a fixed point of P if $P(x) = x$* [3].

Proposition 1.8. *Let X be a Banach space and $P : X \to X$ be a map such that*

$$\|Px - Py\| \leq \alpha \|x - y\| \tag{1.3}$$

for some $0 \leq \alpha < 1$ and all $x, y \in X$. Then P has a unique fixed point in X. Moreover for any $x_0 \in X$ the sequence of iterates $x_0, Px_0, PPx_0, ...$ converges to the fixed point of P [3].

When $\|Px - Py\| \leq \alpha \|x - y\|$ for some $0 \leq \alpha < 1$ and all $x, y \in X$, P is called contraction. Also, the fact that P is contraction implies the existence of a solution of equation (1.3). A contraction shrinks distances by a uniform factor α less than 1 for all pairs of points. Proposition 1.8 is called the contraction mapping theorem or Banach's fixed-point theorem. Also, for any sequence (x_k), we denote $\sum_{k=a}^{b} x_k = 0$, $\prod_{k=a}^{b} x_k = 1$ for any $a > b$.

2. Main Results

Proposition 2.1. *Suppose that $a \in (-1,1) - \{0\}$ and $B(n)$ is a nonsingular matrix function for all $n \in \mathbb{Z}$. Then $x(n)$ is a solution of system (1.2) if and only if*

$$x(n) = x(n_0)a^{n-n_0} + \sum_{t=n_0}^{n-1} B(t)F(x(t-m(t)))a^{n-t-1}. \tag{2.1}$$

Proof. Consider the system (1.2). We multiply both sides of equation (1.2) by $\prod_{s=n_0}^{n} a^{-1}$

$$\Delta\left(x(n)\prod_{s=n_0}^{n-1}a^{-1}\right)=B(n)F\left(x(n-m(n))\right)\prod_{s=n_0}^{n}a^{-1} \tag{2.2}$$

is obtained. If equation (2.2) is summed from n_0 to $n-1$,

$$\begin{aligned}\sum_{t=n_0}^{n-1}\Delta\left(x(n)\prod_{s=n_0}^{n-1}a^{-1}\right) &= \sum_{t=n_0}^{n-1}B(n)F\left(x(n-m(n))\right)\prod_{s=n_0}^{t}a^{-1},\\ x(n)\prod_{s=n_0}^{n-1}a^{-1}-x(n_0) &= \sum_{t=n_0}^{n-1}B(n)F\left(x(n-m(n))\right)\prod_{s=n_0}^{t}a^{-1},\\ x(n)\prod_{s=n_0}^{n-1}a^{-1} &= x(n_0)+\sum_{t=n_0}^{n-1}B(n)F\left(x(n-m(n))\right)\prod_{s=n_0}^{n-1}a^{-1}.\end{aligned}$$

At last we have

$$x(n)=x(n_0)\prod_{s=n_0}^{n-1}a+\sum_{t=n_0}^{n-1}B(n)F\left(x(n-m(n))\right)\prod_{s=t+1}^{n-1}a. \tag{2.3}$$

The proof is completed. □

Theorem 2.2. *Suppose that the following conditions are satisfied:*
(i) $F(0)=0$ and F is locally Lipschitz in x. That is, there is a $M>0$ such that if $\|x\|,\|y\|\le M$, then

$$\|F(x)-F(y)\|\le L\|x-y\| \tag{2.4}$$

for positive constant L.
(ii) There exists $a\in(-1,1)-\{0\}$, $b\in(0,1)$ and $\|B(t)\|=\|B(t)\|_1$ such that

$$L\sum_{t=n_0}^{n-1}\|B(t)\|\le b, \tag{2.5}$$

for $n\ge n_0$. Then the solutions of (1.2) are equi-bounded.

Proof. Let B_1 be a positive constant. We choose $B_2>0$ such that $B_1\le(1-b)B_2$. Also, let $\phi(n)$ be an initial discrete bounded function which satisfies $\|\phi(n)\|\le B_1$ on $\mathbb{Z}_0$ of (1.2) for $n\ge n_0$. We define

$$H=\left\{\mu:\mathbb{Z}\to\mathbb{R}^k \mid \mu(n)=\phi(n) \text{ on } \mathbb{Z}_0 \text{ and } \|\mu\|\le B_2\right\}, \tag{2.6}$$

where $\|\mu\|=\max_{n\in\mathbb{Z}}\|\mu(n)\|$. Firstly, we must show that $(H,\|.\|)$ is a complete metric space. $(H,\|.\|)$ is a metric space. It is clear that the metric space conditions are provided. Now we shall prove that every Cauchy sequence of points in H has a limit that is also in H. Assume that $\left(\mu^{\beta}\right)$ is a Cauchy sequence in H. Then, we have

$$\forall\varepsilon>0,\ \exists\beta_0:\ \forall\alpha,\beta\ge\beta_0:\left\|\mu^{\beta}-\mu^{\alpha}\right\|<\varepsilon,$$

or

$$\forall\varepsilon>0,\ \exists\beta_0:\ \forall\alpha,\beta\ge\beta_0:\max_{n\in\mathbb{Z}}\left\|\left(\mu^{\beta}-\mu^{\alpha}\right)(n)\right\|<\varepsilon,$$

or

$$\forall\varepsilon>0,\ \exists\beta_0:\ \forall\alpha,\beta\ge\beta_0:\left\|\left(\mu^{\beta}-\mu^{\alpha}\right)(n)\right\|<\varepsilon,\quad \forall n\in\mathbb{Z}.$$

$\left(\mu^{\beta}(n)\right)$ is a Cauchy sequence in $\mathbb{R}^k$ for a fixed $n\in\mathbb{Z}$. we know that $\mathbb{R}^k$ is a complete metric space and thus, we can write

$$\mu(n)=\lim_{l\to\infty}\mu^{\beta}(n),\quad \exists\mu(n)\in\mathbb{R}^k. \tag{2.7}$$

Since $\mu^{\beta}\in H$, we get $\mu^{\beta}(n)=\phi(n)$ on $\mathbb{Z}_0$. By (2.6), we know that

$$\mu(n)=\phi(n).$$

Furthermore, we have $\|\mu\|\le B_2$ with $\left\|\mu^{\beta}\right\|\le B_2$. That is $\mu\in H$. We define a mapping $P:H\to H$ such that

$$(P\mu)(n)=\phi(n) \text{ on } \mathbb{Z}_0,$$

and

$$(P\mu)(n) = \phi(n_0)a^{n-n_0} + \sum_{t=n_0}^{n-1} B(t)F(\mu(t-m(t)))a^{n-t-1}. \tag{2.8}$$

Notice that P maps from H to itself. Indeed, the properties of norm

$$\|(P\mu)(n)\| = \left\| \phi(n_0)a^{n-n_0} + \sum_{t=n_0}^{n-1} B(t)F(\mu(t-m(t)))a^{n-t-1} \right\|,$$

we have

$$\|(P\mu)(n)\| \leq \|\phi(n_0)\| \left|a^{n-n_0}\right| + \sum_{t=n_0}^{n-1} \|B(t)\| \left\| F(\mu(t-m(t)))a^{n-t-1} \right\|. \tag{2.9}$$

By $\|\mu\| \leq B_2$, $\|\mu(t-m(t))\| \leq B_2$. Hence, we can write

$$\|F(\mu(t-m(t)))\| \leq L\|\mu(t-m(t))\| \leq LB_2. \tag{2.10}$$

Then, from (2.9) and (2.10), we obtain

$$\begin{aligned} \|(P\mu)(n)\| &\leq B_1 + B_2L \left\| \sum_{t=n_0}^{n-1} B(t)a^{n-t-1} \right\| \\ &\leq B_1 + B_2L \left\| \sum_{t=n_0}^{n-1} B(t) \right\| \\ &\leq B_1 + B_2b \leq B_2. \end{aligned}$$

So, we can say that P maps from H to H. Now, let $\mu, \upsilon \in H$, then we get

$$\begin{aligned} \|(P\mu)(n) - (P\upsilon)(n)\| &= \left\| \sum_{t=n_0}^{n-1} B(t)F(\mu)a^{n-t-1} - \sum_{t=n_0}^{n-1} B(t)F(\upsilon)a^{n-t-1} \right\| \\ &\leq L\sum_{t=n_0}^{n-1} \|B(t)\| \|\mu-\upsilon\| \left|a^{n-t-1}\right| \\ &\leq b\|\mu-\upsilon\|. \end{aligned}$$

Thus, we have shown that the mapping P is a contraction under the supremum norm. According to the contraction mapping principle, P has a unique fixed point $\mu^* \in H$. So, we have $(P\mu^*)(n) = \mu^*(n)$. By $n_0 \in \mathbb{Z}_0$ and $\mu^* \in H$, we can write $\phi(n_0) = \mu^*(n_0)$. Hence

$$\mu^*(n) = \phi(n_0)a^{n-n_0} + \sum_{t=n_0}^{n-1} B(t)F(\mu^*(t-m(t)))a^{n-t-1},$$

that is, $\mu^* \in H$ is a solution of (1.2) and solutions of (1.2) is equi-bounded. The proof is completed. □

Corollary 2.3. *Suppose that the conditions of Theorem 2.1 are satisfied. Then the zero solution of* (1.2) *is Liapunov stable.*

Proof. Let $\varepsilon > 0$ be such that $b\varepsilon \in (0,1)$. If we choose $0 < \delta \leq \varepsilon(1-b)$, then $\delta + b\varepsilon \leq \varepsilon$. Also, let $\phi(n)$ be an initial discrete bounded function which satisfies $\|\phi(n)\| \leq \delta$ on $\mathbb{Z}_0$ of (1.2) for $n \geq n_0$. We define

$$H = \left\{ \mu : \mathbb{Z} \to \mathbb{R}^k \mid \mu(n) = \phi(n) \text{ on } \mathbb{Z}_0 \text{ and } \|\mu\| \leq \varepsilon \right\}, \tag{2.11}$$

where $\|\mu\| = \max_{n\in\mathbb{Z}} \|\mu(n)\|$. It can be shown easily that $(H, \|.\|)$ is a complete metric space similar to proof of Theorem 2.1. Let us define the mapping $P : H \to H$ as follows

$$(P\mu)(n) = \phi(n_0)a^{n-n_0} + \sum_{t=n_0}^{n-1} B(t)F(\mu(t-m(t)))a^{n-t-1}.$$

Also, by proof of Theorem 2.1, we know that P is a contraction and $\|P\mu\| \leq \varepsilon$ for any $\mu \in H$. Hence, the zero solution of (1.2) is Liapunov stable. Therefore, the proof is completed. □

Theorem 2.4. *Suppose that the following conditions are satisfied:*
(i) $F(0) = 0$ and F is locally Lipschitz in x. That is, there is a $M > 0$ such that if $\|x\|, \|y\| \leq M$, then

$$\|F(x) - F(y)\| \leq L\|x-y\| \tag{2.12}$$

for positive constant L.
(ii) There exist $a \in (-1,1) - \{0\}$, $b \in (0,1)$ and $\|B(t)\| = \|B(t)\|_1$ such that

$$L\sum_{t=n_0}^{n-1} \|B(t)\| \leq b, \tag{2.13}$$

for $n \geq n_0$.
(iii) $|n - m(n)| \to \infty$ as $n \to \infty$.
Then the zero solution of (1.2) *is asymptotically stable.*

Proof. Let $\phi(n)$ be an initial discrete bounded function which satisfies $\|\phi(n)\| \leq \eta(n_0)$ on $\mathbb{Z}_0$ of (1.2) for $n \geq n_0$. We define

$$H^\star = \left\{ \mu : \mathbb{Z} \to \mathbb{R}^k \mid \mu(n) = \phi(n) \text{ on } \mathbb{Z}_0,\ \|\mu\| \leq \varepsilon \text{ and } \|\mu(n)\| \to 0 \text{ as } n \to \infty \right\},$$

where $\|\mu\| = \max_{n\in\mathbb{Z}} \|\mu(n)\|$. It can be easily shown that $(H^\star, \|.\|)$ is a complete metric space similar to proof of Theorem 2.1. Let us define the mapping $P : H^\star \to H^\star$ as follows

$$(P\mu)(n) = \phi(n_0)\, a^{n-n_0} + \sum_{t=n_0}^{n-1} B(t) F(\mu(t-m(t)))\, a^{n-t-1}. \tag{2.14}$$

From the proof of Theorem 2.1, P is a contraction and it maps from H to itself. For the asymptotic stability, we shall show that

$$(P\mu)(n) \to 0 \text{ as } n \to \infty. \tag{2.15}$$

Now, since $|a| < 1$,

$$\prod_{s=n_0}^{n-1} a = a^{n-n_0} \to 0 \text{ as } n \to \infty. \tag{2.16}$$

Thus, $\phi(n_0)\, a^{n-n_0} \to 0$. In that case, it will be enough to show that

$$\sum_{t=n_0}^{n-1} B(t) F(\mu(t-m(t))) \to 0 \text{ as } n \to \infty$$

Let $\mu \in H^\star$, then $\|\mu(t-m(t))\| \leq \varepsilon$. Since $\mu(t-m(t)) \to 0$ as $n-m(n) \to \infty$, there exists an $\varepsilon_1 > 0$ and $n_1 > 0$ such that $\|\mu(t-m(t))\| < \varepsilon_1$ for $n > n_1$. Also, from (2.16), there exist an $n_2 > n_1$ such that $\left|a^{n-n_0}\right| \leq \dfrac{\varepsilon_1}{b\varepsilon}$ for $n > n_2$. Hence, for all $n > n_2$

$$\begin{aligned}
\left\| \sum_{t=n_0}^{n-1} B(t) F(\mu(t-m(t)))\, a^{n-t-1} \right\| &\leq \sum_{t=n_0}^{n-1} \left\| B(t) F(\mu(t-m(t)))\, a^{n-t-1} \right\|, \\
&\leq \sum_{t=n_0}^{n_1-1} \left\| B(t) F(\mu(t-m(t)))\, a^{n-t-1} \right\| + \sum_{t=n_1}^{n-1} \left\| B(t) F(\mu(t-m(t)))\, a^{n-t-1} \right\| \\
&\leq L \sum_{t=n_0}^{n_1-1} \left\| B(t)\mu(t-m(t))\, a^{n-t-1} \right\| + L \sum_{t=n_1}^{n-1} \left\| B(t)\mu(t-m(t))\, a^{n-t-1} \right\| \\
&\leq \varepsilon L \sum_{t=n_0}^{n_1-1} \left\| B(t)\, a^{n-t-1} \right\| + \varepsilon_1 L \sum_{t=n_1}^{n-1} \left\| B(t)\, a^{n-t-1} \right\| \\
&\leq \varepsilon L \sum_{t=n_0}^{n_1-1} \left\| B(t)\, a^{n-t-1} \right\| + \varepsilon_1 L \sum_{t=n_1}^{n-1} \left\| B(t)\, a^{n-t-1} \right\| \\
&\leq \varepsilon L \sum_{t=n_0}^{n_1-1} \left\| B(t) \prod_{s=t+1}^{n-1} a \prod_{s=n_1}^{n-1} a \right\| + \varepsilon_1 b \\
&\leq \varepsilon L \left| \prod_{s=n_1}^{n-1} a \right| \sum_{t=n_0}^{n_1-1} \|B(t)\| \left| \prod_{s=t+1}^{n_1-1} a \right| + \varepsilon_1 b \\
&\leq \varepsilon_1 + \varepsilon_1 b.
\end{aligned}$$

This implies that (2.15) is occurs. According to the contraction mapping principle, P has a unique fixed point which solves (1.2). The proof is completed. □

Example 2.5. *Consider difference equation system*

$$x(n+1) = \frac{1}{2} x(n) + B(n) F\left(x\left(n - \left[\left| \frac{n}{2} \right| \right] \right) \right), \qquad n \geq 0, \tag{2.17}$$

where $[|.|]$ *denotes greater integer function,* $a = \frac{1}{2}$, $F(x) = \begin{pmatrix} x_1^2 \\ x_2^2 \end{pmatrix}$ *for* $x = \begin{pmatrix} x_1 \\ x_2 \end{pmatrix}$, $2^{-2n+4m(n)} > 1$ *for* $m(n) = \left[\left| \frac{n}{2} \right| \right]$ *and*

$$B(n) = \begin{pmatrix} \frac{1+2^{-2n+2m(n)}-2^{-2m(n)}}{2^{-m(n)}\left(1+2^{-2n+2m(n)}\right)} & \frac{2^{-m(n)}}{1+2^{-2n+2m(n)}} \\ \frac{3+4.2^{-2n+2m(n)}}{2^{2m(n)+2}\left(1+2^{-2n+2m(n)}\right)} & \frac{1}{2^{2m(n)+2}\left(1+2^{-2n+2m(n)}\right)} \end{pmatrix}, \text{ for } \forall n \in \mathbb{N}.$$

then the zero solution of (2.17) *is asymptotically stable. Indeed,*
(i) there is a $L = 1$ *such that* $\|x\|_1 = |x_1| + |x_2| \leq 1$, *then*

$$\begin{aligned}
\|F(x)\|_1 &= \left| x_1^2 \right| + \left| x_2^2 \right| = |x_1|^2 + |x_2|^2 \\
&\leq (|x_1| + |x_2|)^2 \\
&\leq |x_1| + |x_2| = \|x\|_1
\end{aligned}$$

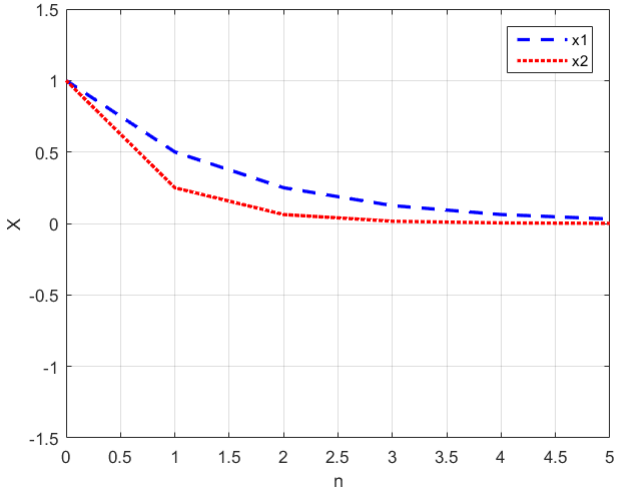

Figure 2.1: The behavior of the solution $x(n) = \begin{pmatrix} 2^{-n} \\ 4^{-n} \end{pmatrix}$ of system (2.17)

for $M = 1$.

(ii) $\|B(n)\|_1 = \max\limits_{1\le i\le n}\left(\sum\limits_{j=1}^{n}|a_{ij}|\right) = \dfrac{1}{2^{m(n)}} \le 1$

and $\lim\limits_{n\to\infty}\dfrac{1}{2^{m(n)}} = 0$. *Furthermore, there exist* $b \in (0,1)$ *such that*

$$\sum_{t=n_0}^{n-1}\|B(t)\| \le b, \tag{2.18}$$

for $n \ge n_0$.

(iii) $\left|n - \left[\left|\frac{n}{2}\right|\right]\right| \to \infty$ *as* $n \to \infty$.

Since the conditions of the theorem are provided, the system (2.17) is asymptotically stable. The behavior of the solutions of the system (2.17) is as Figure 1.

3. Conclusion

In this paper, we investigate the asymptotic stability of zero solution of system (1.2) which as an generalization of [9]. We used the Banach's fixed point theorem for the proof of the results. In this study, by using the definition of equi-bounded, initially, we showed that equation (1.2) is Liapunov stable after that we proved that this equation is asymptotically stable. Finally, we applied the these results to an example. As a result of this application we verified that equation (2.17) is suitable to the example

$$x(n+1) = \frac{1}{2}x(n) + B(n)F\left(x\left(n - \left[\frac{n}{2}\right]\right)\right), \qquad n \ge 0,$$

where F is the real valued vector function, $m : \mathbb{Z} \to \mathbb{Z}^+$, which is bounded function and maximum value of m is k and $B(n)$ is a $k \times k$ variable coefficient matrix.

References

[1] RP. Agarwal, *Difference equations and inequalities: theory, methods, and applications* CRC Press, (2000).
[2] MP. Chen, B. Liu, *Asymptotic behavior of solutions of first order nonlinear delay difference equations* Computers & Mathematics with Applications, (1996), 32.4: 9-13.
[3] K. Conrad, *The Contraction Mapping Theorem* II, (2014).
[4] J.T. Edwards, J.F. Neville, *Boundedness and stability of solutions to difference equations* Journal of Computational and Applied Mathematics, (2002), 140.1-2: 275-289.
[5] D.V. Giang, D.C. Huong, *Extinction, Persistence and global stability in models of population growth* Journal of mathematical analysis and applications, (2005), 308.1: 195-207.
[6] D.V. Giang, D.C. Huong, *Nontrivial periodicity in discrete delay models of population growth* Journal of mathematical analysis and applications, (2005), 305.1: 291-295.
[7] J.R. Graef, C. Qian, *Global stability in a nonlinear difference equation* Journal of Difference Equations and Applications, (1999), 5.3: 251-270.
[8] I. Győri, G. Ladas, P.N. Vlahos, *Global attractivity in a delay difference equation* Nonlinear Analysis: Theory, Methods & Applications, (1991), 17.5: 473-479.
[9] D.C. Huong, N.V. Mau, *On a nonlinear difference equation with variable delay* Demonstratio Mathematica, (2013), 46.1: 123-135.
[10] D.C. Huong, *On the asymptotic behaviour of solutions of a nonlinear difference equation with bounded multiple delay* Vietnam Journal of Mathematics, (2006), 34.2: 163-170.
[11] D.C. Huong, *Oscillation for a Nonlinear Difference Equation* Vietnam Journal of Mathematics, (2009), 37.4: 537-549.
[12] D.C. Huong, P.T. Nam, *On Oscillation, Convergence and Boundedness of Solutions of Some Nonlinear Difference Equations with Multiple Delay*Vietnam Journal of Mathematics, (2008), 36.2: 151-160.

[13] D.C. Huong, *Persistence and global attractivity for a discretized version of a general model of glucose-insulin interaction* Demonstratio Mathematica, (2016), 49.3: 302-318.
[14] D.C. Huong, *Asymptotic Stability and Strict Boundedness for Non-autonomous Nonlinear Difference Equations with Time-varying Delay* Vietnam Journal of Mathematics, (2016), 44.4: 789-800.
[15] M. Liao, *Global asymptotic stability of a family of nonlinear difference equations.* Discrete Dynamics in Nature and Society, (2013).
[16] Y. Muroya, E. Ishiwata, N. Guglielmi, *Global stability for nonlinear difference equations with variable coefficients* Journal of mathematical analysis and applications 334.1 (2007): 232-247.
[17] S.U. Deger, Y.Bolat, *Stability conditions a class of linear delay difference systems* Cogent Mathematics, (2017), 4.1:129445.
[18] V. Sree Hari Rao, G.Jaya Sudha, *Global Behaviour of Solutions of Nonlinear Delay Difference Equations.* The Journal of Difference Equations and Applications, (2002), 8.2: 101-124.
[19] A. Asiri, E.M. Elsayed, *Dynamics and Solutions of Some Recursive Sequences of Higher Order* Journal of Computational Analysis& Applications, (2019), 27.1.
[20] X. Zhang, J. Yan, *Global asymptotic behavior of nonlinear difference equations* Computers & Mathematics with Applications, (2005), 49.9-10: 1335-1345.
[21] X. Zhang, *Global attractivity for nonlinear difference equations with delay.* Journal of Mathematical Analysis and Applications, (2007), 336 : 975-986.
[22] A. Akgul, M.Inc, E. Karatas *Reproducing kernel functions for difference equations.* Discrete and Continuous Dynamical Systems, (2015), 8(6).
[23] A. Akgul, *On the solution of higher-order difference equationsy.* Mathematical Methods in the Applied Sciences, (2015).

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Simulation on The Mathematical Model for the Control Of Hepatitis B Virus-Hepatitis D Virus (HBV-HDV) Co-infection Transmission Dynamics in a Given Population

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Abstract

This paper investigates the impact of the various parameters of the mathematical model for Hepatitis B virus-Hepatitis D virus (HBV-HDV) co-infection with controls (awareness, vaccine and therapy). It establishes that the model is biologically meaningful and epidemiologically well posed. Furthermore, simulations are carried out on the equations of the model using MATLAB and the results indicate that; when c_1(awareness) increase from 0.08 to 0.70, then the number of exposed HB individuals in the population will also increase. Conversely, we notice a drastic decrease in the number of exposed HBD individuals in the population when c_1(awareness) increase from 0.08 to 0.70. Again, we observe a decrease in the number of exposed treated individuals in the population when c(therapy) increase from 0.08 to 0.50. Similarly, we notice an increase in the number of recovered HBD individuals in the population upon the increase of c(therapy) from 0.08 to 0.50. We therefore conclude that awareness, vaccine and therapy are good measure which can be used to effectively control HBV-HDV co-infection in a population. However, awareness and vaccine are better control strategies than therapy. Hence, these simulation results provide the best framework for the control of the disease; Hepatitis B virus-Hepatitis D virus (HBV-HDV) co-infection in a population.

1. Introduction

Hepatitis B virus (HBV) and Hepatitis D virus (HDV) infections are major health problem. HBV virus is a DNA virus from hepadnaviridae family. Infected person or asymptomatic carriers with viral HBV are only reservoir of infection [1]. Researches show us that world prevalence of Hepatitis B surface antigen (HBsAg) carriers is from $0.1-20\%$ with high percentage in tropical countries [1]. The risk of transmission of HBV via blood and blood product is much higher than Hepatitis C (HCV) and HIV. HBV may induce chronic hepatitis that could progress to cirrhosis and hepatocellurar carcinoma [2].
Hepatitis D virus (HDV) sometimes called Hepatitis Delta Virus, was detected by Rizzetto among patients with a severe form of Hepatitis B virus (HBV) infection in the year of 1977 [3]. Chronic Hepatitis D still remains a major cause of liver transplantation and death [4]. Cirrhosis the final stage of chronic Hepatitis, has been considered to be irreversible [5]. HDV induces a broad range of clinical manifestations in humans, ranging from asymptomatic cases to patients with fulminant hepatitis and hepatocellular carcinoma ([6], [7]). HDV or Delta virus is an incomplete defective RNA virus requiring concomitant presence of HBV for its survival and replication ([8]; [9]; [10]; [11]). Thus, HDV can replicate only in people who are also infected with HBV.
The epidemiology of HDV infection is similar to HBV with some exception ([10]; [12]). It is estimated that approximately 5% of the HBV carrier are co-infected with HDV infection worldwide [6]. However, the prevalence of HDV in HBV carriers varies around the world [13].

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As mentioned above, HDV is a defective virus since it cannot produce infectious virions without the help of a co-infecting helper virus. The helper virus is Hepatitis B that supplies the HBsAg surface protein. In budding out of the cell, HDV acquires a membrane containing HBsAg. It uses HBsAg as its envelope protein; thus, HBV co-infection is necessary for the packaging and release of HDV virions from infected hepatocytes. The smallest HBsAg is even sufficient to package the HDV genome.
Due to the complexity of the health problems caused by HBV infection and HDV infection as mentioned above, it becomes imperative to study the co-infection of the two deadly diseases. Thus, [14] developed a mathematical model for Hepatitis B virus - Hepatitis D virus (HBV-HDV) co-infection with controls. They obtained the basic reproduction number R_0 for the model and consequently carried out sensitivity analysis on the model using MATHCAD. [15], investigated stability analysis of Hepatitis B virus-Hepatitis D virus co-infection using Jacobian method. Valid results were obtained by evaluating the Jacobian matrix formed numerically.
This paper investigates the impact of the various parameters of the mathematical model developed by [14]. It establishes that the model is biologically meaningful and epidemiologically well posed. Furthermore, simulations are carried out on the equations of the model using MATLAB and the results are vividly discussed. From the results, awareness and vaccine are better control strategies than therapy. Thus, the results of the simulation provide the best framework for the control of HBV-HDV co-infection in a population.

2. Model formulation

Section 2 and its subheadings are taken from references [14] and [15]

2.1. Assumptions of the Model

The model is based on the following assumptions:

1. The individuals that make up the population are grouped into different compartments or groups according to their epidemiological state.
2. The population size in a compartment varies with respect to time.
3. The population mixes homogeneously. That is, all susceptible individuals are equally likely to be infected by infectious individuals if they come in contact with one another.
4. The infection does not confer immunity to the recovered individuals and so they can go back to the susceptible class at any given time.
5. The individuals in each compartment have equal natural death rate given as μ.
6. The gain in the infectious class is at a rate proportional to the number of infectious and susceptible individuals. That is, βSI, where $\beta > 0$ is a contact parameter (effective contact rate). The susceptible are lost at the same rate.
7. The rate of removal of infectious to the recovered or removed class is proportional to the number of infectious individuals.

2.2. Model Variables

The following variables are used in this study, thus:
S: The number of susceptible individuals
$\mathbf{E_{H}}_{B}$: The number of individuals who are exposed to HBV
$\mathbf{E_{T}}_{HB}$: The number of individuals who are exposed to HBV and are being treated
$\mathbf{I_{H}}_{B}$: The number of individuals who are infectious of HBV
$\mathbf{I_{N}}_{HB}$: The number of individuals who are infectious of HBV and not being treated
$\mathbf{I_{T}}_{HB}$: The number of individuals who are infected with HBV and are being treated
$\mathbf{R_{H}}_{B}$: The number of individuals who have been treated of HBV and have recovered
$\mathbf{E_{H}}_{BD}$: The number of individuals who are infectious of HBV and latently infected with HDV (exposed of HDV)
$\mathbf{E_{T}}_{HBD}$: The number of individuals who are infectious of HBV and now exposed to HDV and being treated
$\mathbf{I_{H}}_{BD}$: The number of individuals who are infected with HBV and HDV at the same time
$\mathbf{I_{N}}_{HBD}$: The number of individuals who are infected with HBV and HDV and not being treated of any
$\mathbf{I_{T}}_{HBD}$: The number of individuals who are infected with HBV and HDV and are being treated of both
$\mathbf{R_{H}}_{BD}$: The number of individuals who have recovered from HBV-HDV co-infection after they have been treated

2.3. Parameters of the Model

We shall also use the following parameters in this model, thus:
π: The number of people that enter the population (the number of individuals that enter into the susceptible class)
β_1 : Contact rate for I_{HB} with susceptible individuals (S). i.e., the rate at which individuals who had contact with HBV infectious person become exposed to HBV
β_2 : Contact rate for I_{HBD} with susceptible classes $(E_{HB}, I_{NHB} and I_{THB})$
τ: The rate at which individuals who are exposed to HBV become infectious of HBV
ω: The rate at which individuals who are exposed to HBV enter into exposed and being treated compartment or class (E_{THB})
ρ_2: The rate at which individuals who are infectious of HBV enter into infectious and being treated HBV class (I_{THB})
ρ_1: The rate at which individuals who are infectious of HBV enter into infectious and not being treated of HBV (I_{NHB})
λ_1: The rate at which individuals that are infectious and being treated of HBV goes back to exposed HBV class
λ_2: The rate at which individuals who are infectious and being treated of HBV recover from HBV
α: The rate at which individuals who are exposed and being treated of HBV recover
ϕ_1: The rate at which individuals who recovered from HBV goes back to susceptible class
φ: The rate at which individuals that are infectious and being treated of HBV become exposed to HDV, that is E_{HBD}
ψ: The rate at which individuals who are infectious of HBV and not being treated become exposed to HDV, that is E_{HBD}

$\beta_2[1-(c_1+c_2)]$: The rate at which individuals who are exposed of HBV become exposed to HDV (E_{HBD})
ζ: The rate at which HBV infectious individuals who are exposed to HDV enter into exposed HDV being treated class (E_{THBD}). that is the rate at which individuals who are exposed of HBV-HDV co-infection enter into exposed HBV-HDV being treated class (E_{THBD})
κ: The rate at which HBV infectious individuals who are exposed to HDV become infectious of HBV-HDV
ε_1: The rate at which individuals who are infectious of HBV-HDV enter into infectious HBV-HDV not being treated class (I_{NHBD})
ε_2: The rate at which individuals who are infectious of HBV-HDV enter into infectious HBV-HDV being treated class (I_{THBD})
η: The rate at which individuals that are infectious of HBV-HDV and are being treated recover
$\in$: The rate at which HBV infectious individuals who are exposed to HDV and then being treated become infectious of HBV-HDV
$\theta(1+c)$: The rate at which HBV infectious individuals that are exposed to HDV and are being treated recover
ϕ_2: The rate at which individuals who recovered from HBV-HDV goes back to susceptible class again
μ: The natural mortality/death rate
δ: HBV-induced mortality/death rate for people infectious of HBV treated class
δ_1: HBV-induced mortality/death rate for people infectious of HBV but not being treated class
δ_2: HDV-induced mortality/death rate for HBV infectious individuals who are exposed and being treated of HDV
δ_3: HBV-HDV-induced mortality/death rate for HBV infectious individuals who are infectious of HBV-HDV and are being treated
δ_4: HBV-HDV-induced mortality/death rate for individuals who are infectious of HBV-HDV and are not being treated
θ: Cure rate
$\left.\begin{matrix} c \\ c_1 \\ c_2 \end{matrix}\right\}$: Infectivity controls; where c_1 is awareness, c_2 is vaccine and c is therapy

2.4. Model Description

Based on the standard SEIR model, the population is partitioned into thirteen compartments or classes namely: Susceptible (S), Exposed to HBV (E_{HB}), Exposed to HBV and Treated (E_{THB}), Infectious of HBV (I_{HB}), Infectious of HBV and Treated (I_{THB}), Infectious of HBV and not Treated (I_{NHB}), Recovered of HBV (R_{HB}), HBV infectious now Exposed to HDV (E_{HBD}), HBV infectious now Exposed to HDV and treated (E_{THBD}), Infectious of HBV-HDV co-infection (I_{HBD}), Infectious of HBV-HDV co-infection and treated (I_{THBD}), Infectious of HBV-HDV co-infection and not treated (I_{NHBD}), Recovered of HBV-HDV co-infection (R_{HBD}) Compartments.

2.5. Model Equations

$$\frac{dS}{dt} = \pi + \phi_1 R_{HB} + \phi_2 R_{HBD} - \beta_1 S I_{HB} - \mu S \tag{2.1}$$

$$\frac{dE_{HB}}{dt} = \beta_1 S I_{HB} - \beta_2[1-(c_1+c_2)]E_{HB}I_{HBD} + \lambda_1 I_{THB} - \omega E_{HB} - \tau E_{HB} - \mu E_{HB} \tag{2.2}$$

$$\frac{dE_{THB}}{dt} = \omega E_{HB} - \mu E_{THB} - \alpha E_{THB} \tag{2.3}$$

$$\frac{dI_{HB}}{dt} = \tau E_{HB} - \rho_1 I_{HB} - \rho_2 I_{HB} \tag{2.4}$$

$$\frac{dI_{NHB}}{dt} = \rho_1 I_{HB} - \psi I_{NHB} I_{HBD} - \mu I_{NHB} - \delta_1 I_{NHB} \tag{2.5}$$

$$\frac{dI_{THB}}{dt} = \rho_2 I_{HB} - \varphi I_{THB} - \lambda_1 I_{THB} - \lambda_2 I_{THB} - \mu I_{THB} - \delta I_{THB} \tag{2.6}$$

$$\frac{dR_{HB}}{dt} = \lambda_2 I_{THB} + \alpha E_{THB} - \phi_1 R_{HB} - \mu R_{HB} \tag{2.7}$$

$$\frac{dE_{HBD}}{dt} = \beta_2 E_{HB} I_{HBD} + \psi I_{NHB} I_{HBD} + \varphi I_{THB} E_{HBD} - \kappa E_{HBD} - \zeta E_{HBD} - \mu E_{HBD} \tag{2.8}$$

$$\frac{dE_{THBD}}{dt} = \zeta E_{HBD} - \theta(1+c)E_{THBD} - \in E_{THBD} - \mu E_{THBD} - \delta_2 E_{THBD} \tag{2.9}$$

$$\frac{dI_{HBD}}{dt} = \kappa E_{HBD} + \in E_{THBD} - \varepsilon_1 I_{HBD} - \varepsilon_2 I_{HBD} \tag{2.10}$$

$$\frac{dI_{NHBD}}{dt} = \varepsilon_1 I_{HBD} - \mu I_{NHBD} - \delta_4 I_{NHBD} \tag{2.11}$$

$$\frac{dI_{THBD}}{dt} = \varepsilon_2 I_{HBD} - \eta I_{THBD} - \mu I_{THBD} - \delta_3 I_{THBD} \tag{2.12}$$

$$\frac{dR_{HBD}}{dt} = \theta(1+c) E_{THBD} + \eta I_{THBD} - \mu R_{HBD} - \phi_2 R_{HBD} \tag{2.13}$$

$$N = S + E_{HB} + E_{THB} + I_{NHB} + I_{THB} + R_{HB} + E_{HBD} + E_{THBD} + I_{NHBD} + I_{THBD} + R_{HBD} \tag{2.14}$$

Susceptible individuals acquire HBV infection following effective contact with individuals infected with HBV (i.e. those in the E_{HB}, I_{NHB} and I_{THB} classes) at a rate β_1, given by

$$\beta_1 = \frac{\theta_B (E_{HB} + \mu_3 I_{NHB} + \mu_4 I_{THB})}{N};\ N = S + E_{HB} + I_{NHB} + I_{THB}$$

where θ_B is the effective contact rate for HBV transmission. Further, the modification parameters $\mu_3 = 1$ and $\mu_4 < 1$ account for the relative infectiousness of individuals in the I_{NHB} and I_{THB} classes in comparison to those in the E_{HB} class. That is individuals in I_{NHB} are more infectious than those individuals in the E_{HB} class, likewise, I_{THB} are less infectious than those in the I_{NHB} class (because the use of treatment significantly reduces the viral load in those being treated).
Similarly, individuals in the susceptible classes $(E_{HB}, I_{NHB}$ and $I_{THB})$ acquire HBD, following effective contact with individuals infected with HBD
(i.e., those in the E_{HBD}, I_{NHBD} and I_{THBD}) at a rate β_2, given by

$$\beta_2 = \frac{\theta_{BD} (E_{HBD} + \mu_5 I_{NHBD} + \mu_6 I_{THBD})}{N};\ N = S + E_{HBD} + I_{NHBD} + I_{THBD}$$

where θ_{BD} is the effective contact rate for HBD transmission. Further, the modification parameters $\mu_5 = 1$ and $\mu_6 < 1$ account for the relative infectiousness of individuals in the I_{NHBD} and I_{THBD} classes in comparison to those in the E_{HBD} class. That is, individuals in the I_{NHBD} class are more infectious than those in the E_{HBD} class, and likewise, I_{THBD} are less infectious than those in I_{NHBD} class (because the use of treatment significantly reduces the viral load in those being treated)

3. Invariant Region

The HBV-HDV co-infection model will be analyzed in a validity region in order to show that it is biologically meaningful and the region is feasible for human population. We assume that all the state variables and parameters are positive all the time, $t \geq 0$. We shall prove that the HBV-HDV co-infection model is well-posed by proving the **boundedness** and **positivity** of the solutions of the model with the non-negative initial solution for all time. We will obtain the region by considering the following theorems;

Theorem 3.1 (Boundedness)**.** *The solution set*

$$\{S(t), E_{HB}(t), E_{THB}(t), I_{HB}(t), I_{NHB}(t), I_{THB}(t), R_{HB}(t), E_{HBD}(t), E_{THBD}(t), I_{HBD}(t), I_{NHBD}(t), I_{THBD}(t), R_{HBD}(t)\}$$

is contained and bounded in the feasible region D.

Proof. The total human population can be determined by

$$N(t) = S(t) + E_{HB}(t) + E_{THB}(t) + I_{NHB}(t) + I_{THB}(t) + R_{HB}(t) + E_{HBD}(t) + E_{THBD}(t) + I_{NHBD}(t) + I_{THBD}(t) + R_{HBD}(t)$$

Now adding the right hand side of equations (2.1) - (2.13)
So, the time derivatives, $\frac{dN}{dt}$, along solutions of system is obtained as

$$\begin{aligned}\frac{dN}{dt} = \pi &- \mu S - \mu E_{HB} - \mu E_{THB} - \mu I_{NHB} - \mu I_{THB} - \mu R_{HB} - \mu E_{HBD} - \mu E_{THBD} - \mu I_{NHBD} - \mu I_{THBD} - \mu R_{HBD} - \delta I_{THB} \\ &- \delta_1 I_{NHB} - \delta_2 E_{THBD} - \delta_3 I_{THBD} - \delta_4 I_{NHBD} \leq \pi - \mu N.\end{aligned}$$

That is, $\frac{dN}{dt} = \pi - \mu N - \delta I_{THB} - \delta_1 I_{NHB} - \delta_2 E_{THBD} - \delta_3 I_{THBD} - \delta_4 I_{NHBD} \leq \pi - \mu N$
Assume that the initial condition for model satisfies $N(0) \leq \frac{\pi}{\mu}$, where

$$N(0) = S(0) + E_{HB}(0) + E_{THB}(0) + I_{NHB}(0) + I_{THB}(0) + R_{HB}(0) + E_{HBD}(0) + E_{THBD}(0) + I_{NHBD}(0) + I_{THBD}(0) + R_{HBD}(0)$$

Then, applying the Gronwall's inequality gives
$N(t) \leq \frac{\pi}{\mu} + \left(N(0) - \frac{\pi}{\mu}\right) e^{-\mu t}$ whenever $(0) \leq \frac{\pi}{\mu}$.
So, taking the limit as $t \to \infty$ yields $(t) \leq \frac{\pi}{\mu}$. This shows that the feasible region for the model exists and is bounded by $N(t) \leq \frac{\pi}{\mu}$. It means that all the solutions of system are nonnegative in D for any time $t > 0$ and this represents human population. □

3.1. Positivity of solutions

Lemma1: Let the set of initial solution be

$$\{(S(0),E_{HB}(0),E_{THB}(0),I_{HB}(0),I_{NHB}(0),I_{THB}(0),R_{HB}(0),E_{HBD}(0),E_{THBD}(0),I_{HBD}(0),I_{NHBD}(0),I_{THBD}(0),R_{HBD}(0))\in\Phi\}.$$

Then, the solution set

$$\{S(t),E_{HB}(t),E_{THB}(t),I_{HB}(t),I_{NHB}(t),I_{THB}(t),R_{HB}(t),E_{HBD}(t),E_{THBD}(t),I_{HBD}(t),I_{NHBD}(t),I_{THBD}(t),R_{HBD}(t)\}$$

of the HBV-HDV co-infection transmission model is non-negative for $t>0$.

Proof. Assume that the set of initial solutions,

$$\{(S(0),E_{HB}(0),E_{THB}(0),I_{HB}(0),I_{NHB}(0),I_{THB}(0),R_{HB}(0),E_{HBD}(0),E_{THBD}(0),I_{HBD}(0),I_{NHBD}(0),I_{THBD}(0),R_{HBD}(0))\ \geq\ 0\},$$

then the first equation, that is equation (2.1) can be written as

$$\frac{dS}{dt}\geq\pi-(\beta_1 I_{HB}-\mu)S=\pi-\beta(t)S \tag{3.1}$$

where $\beta(t)=\ \beta_1 I_{HB}+\mu$.
Equation (3.1) is a linear first order ordinary differential equation in S with the solution

$$S(t)=S(0)\,exp\left(\int_0^t-\beta(S)\,ds\right)\times\int_0^t\pi exp\left(\int_0^u\beta(\omega)\,d\omega\right)\geq\ 0.$$

Hence, $S(t)\geq\ 0\quad\forall\quad t\geq 0$.
In a similar manner, the remaining state variables are obtained such that

$$E_{HB}(t)\ \geq\ E_{HB}(0)\,Exp(\ -(\mu\ +\tau+\omega))\ \geq\ 0,$$

$$E_{THB}(t)\geq\ E_{THB}(0)\,Exp(\ -(\alpha+\mu))\ \geq\ 0,$$

$$I_{HB}(t)\geq\ I_{HB}(0)\,Exp\,(-(\rho_2+\rho_1))\geq 0,$$

$$I_{NHB}(t)\ \geq\ I_{NHB}(0)\,Exp(\ -(\mu+\delta_1))\ \geq\ 0,$$

$$I_{THB}(t)\ \geq\ I_{THB}(0)\,Exp(\ -(\mu+\lambda_1+\lambda+\delta))\ \geq\ 0,$$

$$R_{HB}(t)\ \geq\ R_{HB}(0)\,Exp(\ -(\mu\ +\phi_1))\ \geq\ 0.$$

$$E_{HBD}(t)\ \geq\ E_{HBD}(0)\,Exp(\ -(\mu\ +\kappa+\zeta))\ \geq\ 0,$$

$$E_{THBD}(t)\geq\ E_{THBD}(0)\,Exp(\ -(\theta(1+c)+\mu+\varepsilon+\delta_2))\ \geq\ 0,$$

$$I_{HBD}(t)\geq\ I_{HBD}(0)\,Exp\,(-(\varepsilon_2+\varepsilon_1))\geq 0,$$

$$I_{NHBD}(t)\ \geq\ I_{NHBD}(0)\,Exp(\ -(\mu+\delta_4))\ \geq\ 0,$$

$$I_{THBD}(t)\ \geq\ I_{THBD}(0)\,Exp(\ -(\mu+\eta+\delta_3))\ \geq\ 0,$$

$$R_{HBD}(t)\ \geq\ R_{HBD}(0)\,Exp(\ -(\mu\ +\phi_2))\ \geq\ 0.$$

This completes the proof of the lemma 1. □

Therefore, the biological validity of the HBV-HDV co-infection model is stated in lemma 2.
Lemma 2: The HBV-HDV co-infection model is well posed and valid in the set

$$D\ =\ \left\{S(t),E_{HB}(t),E_{THB}(t),I_{HB}(t),I_{NHB}(t),I_{THB}(t),R_{HB}(t),E_{HBD}(t),E_{THBD}(t),I_{HBD}(t),I_{NHBD}(t),I_{THBD}(t),R_{HBD}(t)\in\mathbb{R}_+^{13}\right.$$
$$\left.:\ N(t)\leq\ \frac{\pi}{\mu}\right\}$$

According to [16], the HBV-HDV co-infection model is biologically meaningful and epidemiologically well posed in the region D.

S/N	Parameter	Value	Reference
1	μ	0.021	[17] and [18]
2	π	1000	Assumed
3	β_1	0.37	Assumed
4	τ	0.50	[19] and [20]
5	β_2	0.37	Assumed
6	c_1	0.08	[17]
7	c_2	0.08	[17]
8	ψ	0.047	Assumed
9	$\varphi \Rightarrow \Phi$	0.043	Assumed
10	λ_1	0.01	Assumed
11	λ_2	0.015	[17]
12	ω	0.02	Assumed
13	α	0.015	[17]
14	ρ_1	0.33	[19] and [20]
15	ρ_2	0.33	[19] and [20]
16	θ	0.08	[17]
17	c	0.08	[17]
18	ε_1	0.038	Assumed
19	ε_2	0.038	Assumed
20	η	0.028	Assumed
21	K	0.50	[19] and [20]
22	ζ	0.032	Assumed
23	$\in$	0.035	Assumed
24	ϕ_1	0.92	Assumed
25	ϕ_2	0.92	Assumed
26	δ	0.068	[17]
27	δ_1	0.068	[17]
28	δ_2	0.068	[17]
29	δ_3	0.068	[17]
30	δ_4	0.068	[17]

Table 1: Parameters value for HBV-HDV co-infection

4. Simulation Results and Discussion

Here we investigates the impact of the various parameters of the model developed by [14] through simulation using MATLAB. This will help provide the best framework for control strategies of the HBV-HDV co-infection in a population. Table 1 shows the set of parameter values used in the simulation for HBV-HDV co-infection.

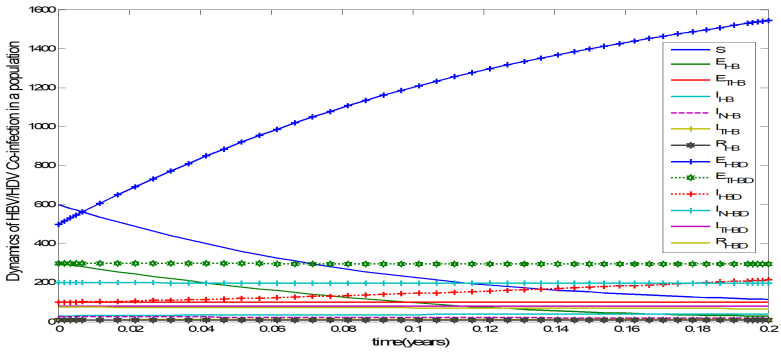

Figure 4.1: A graph showing the dynamics of HBV-HDV co-infection in a population.

Figure 4.1, shows all the equations describing the dynamics of HBV-HDV co-infection in a population.

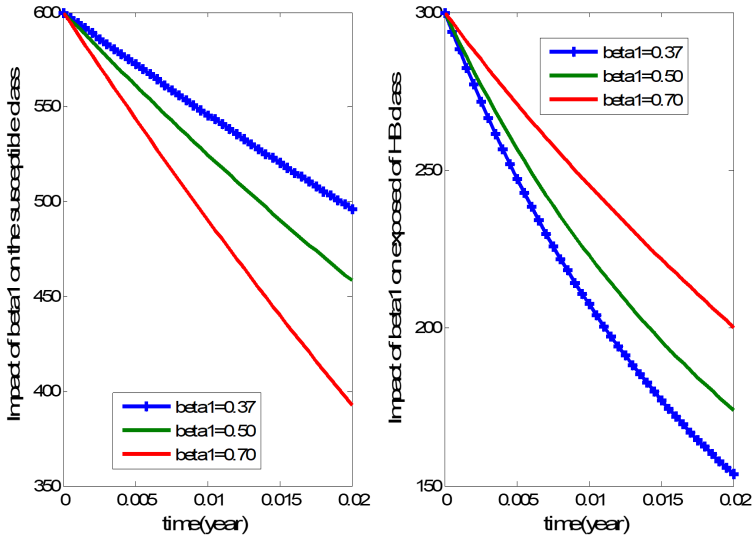

Figure 4.2: Graphs showing the impact of β_1 (contact rate for I_{HB} with susceptible individuals) in the population.

Figure 4.2(a): The graph on the left, showing the impact of β_1 on the susceptible class.
Figure 4.2(b): The graph on the right, showing the impact of β_1 on exposed of HB class.
From figure 4.2(a), increasing β_1 decreases the number of susceptible people in the population. On the contrary, increasing β_1 increases the number of individuals in the population who are exposed to HB (see figure 4.2b). Therefore, as a control strategy, efforts should be geared towards ensuring that the contact rate is drastically reduced. This is achievable if more susceptible individuals are immunized against HBV infection. This is so because upon immunization the individual will lose his/her potency of contacting the disease for a considerable period of time. Active vaccination (Energix-B and Recombivax-HB) is recommended as this will offer long lasting active immunity against HBV and invariably will help control the spread of HBV in the population.

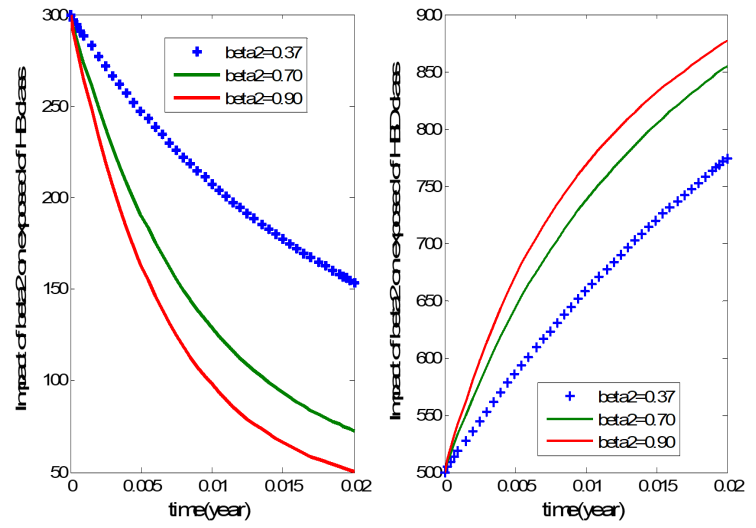

Figure 4.3: Graphs showing the impact of β_2 (contact rate for I_{HBD} with susceptible classes) in the population.

Figure 4.3(a): The graph on the left, showing the impact of β_2 on exposed of HB class.
Figure 4.3(b): The graph on the right, showing the impact of β_2 on exposed of HBD class.
In figure 4.3(a), we observe that increasing β_2 brings about a decrease in the number of exposed of HB people in the population. On the contrary, increasing β_2 increases the number of individuals in the population who are exposed of HBD as can be clearly seen in figure 4.3(b) above. Therefore, as a control strategy, we must increase awareness of the disease amongst the people. Once people are aware of the disease, then they can indulge on other diligent preventive measures: vaccine, not sharing needles or other drug paraphernalia and not sharing items such as tooth brushes and razors. Individuals can also get vaccinated against HBV once they become aware. Therefore, efforts should be targeted at increasing the above mentioned strategies for effective control of HBV in the population. Once we are able to effectively reduce the number of HBV individuals in the population through the above mentioned control strategies, then we would have succeeded in reducing the contact rate of HB and exposed of HBD in the population.

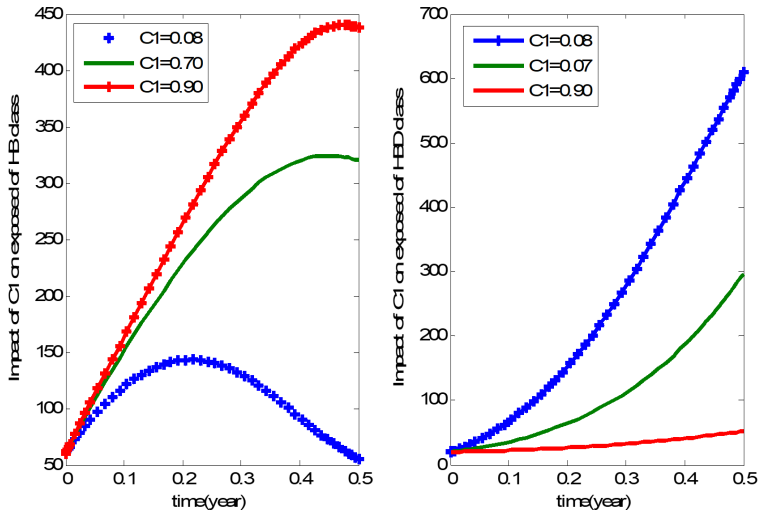

Figure 4.4: Graphs showing the impact of c_1(awareness) in the population.

Figure 4.4(a): The graph on the left, showing the impact of c_1 on exposed of HB class.
Figure 4.4(b): The graph on the right, showing the impact of c_1 on exposed of HBD class.
In figure 4.4(a), increasing c_1 (awareness), shows an increase on the number of exposed of HB individuals in the population as compare to the number of exposed of HBD individuals in the population. Conversely, in figure 4.4(b), increasing c_1 shows a drastic decrease on the number of exposed HBD people in the population in contrast to HBV individuals in the population. From this we can see that awareness is a very good control measure, this is so because when awareness increased more people will remain in exposed of HB class which is better and much easier to treat than exposed of HBD patients. Therefore, to effectively control HBV-HDV co-infection in any given population, we must increase awareness of the HBV disease amongst the people. Once people are aware of the disease, then they can indulge on other diligent preventive measures: condom use, not sharing needles or other drug paraphernalia, not sharing items such as tooth brushes and razors. Individuals can also get vaccinated against HBV once they become aware. Therefore, efforts should be targeted at increasing the above mentioned strategies for effective control of HBV - HDV co-infection in the population.

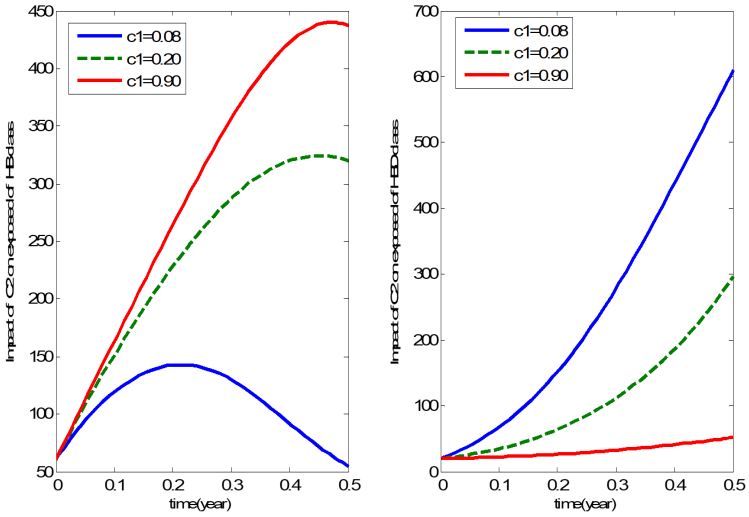

Figure 4.5: Graphs showing the impact of c_2(vaccine) in the population.

Figure 4.5(a): The graph on the left, showing the impact of c_2 on exposed of HB class.
Figure 4.5(b): The graph on the right, showing the impact of c_2 on exposed of HBD class.
Note: Vaccine is to prevent people from contracting the infection while therapy is used to cure those infected with the disease.
Again we see in figure 4.5(a), increasing c_2(vaccine), shows a great increase on the number of exposed of HB individuals in the population compare to the number of HBD individuals in the population. Subsequently, in figure 4.5(b), increasing c_2 shows a drastic decrease on the number of exposed HBD people in the population compare to the number of HBV individuals in the population. From this we can see that vaccine is a very good control measure, this is so because when more people are vaccinated even if they are already exposed to HB, their condition will never deteriorate to exposed of HBD (that is HBV-HDV co-infection) which always has a worst outcome than exposed of HBV or even infectious of HBV. Therefore, to effectively control HBV-HDV co-infection in any given population, the need for people to be

vaccinated against HBV is highly recommended as this will go a long way in helping to reduce HBV-HDV co-infection. Active vaccination (Energix-B and Recombivax-HB) is recommended; this will offer long lasting active immunity against HBV and invariably will help control HBV-HDV co-infection in the population.

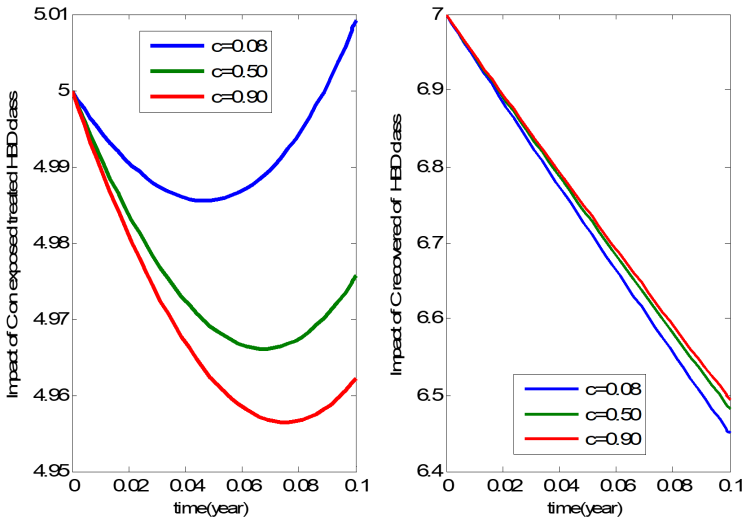

Figure 4.6: Graphs showing the impact of c (therapy) in the population.

Figure 4.6(a): The graph on the left, showing the impact of c on exposed treated HBD class.
Figure 4.6(b): The graph on the right, showing the impact of c on recovered of HBD class.
Note: Vaccine is used as a preventive measure while therapy is used to cure those infected with the disease already.
From figure 4.6(a), the impact of c(therapy) as a control measure is noticeable. If c(therapy) increases, then there is a noticeable decrease on the number of exposed and treated individuals in the population. Similarly, in figure 4.6(b), we can see an increase on the number of recovered HBD individual in the population upon the increase of c(therapy). This implies that therapy has a positive impact as control measure in the population. More exposed treated HBD individuals will recover after using the drug. The available drugs for the treatment of HBV which also should be used for patients exposed of HBD are: (1) Lamivudine (LMV) or Ribavirine (2) Eggylated Alpa-interferon (IFN). However, it is worthy to note; (i) a large doses of Alpha-interferon may be given for up to 12 months (ii) Alpha-interferon can cause the disease to go into remission (according to World Health Organisation).

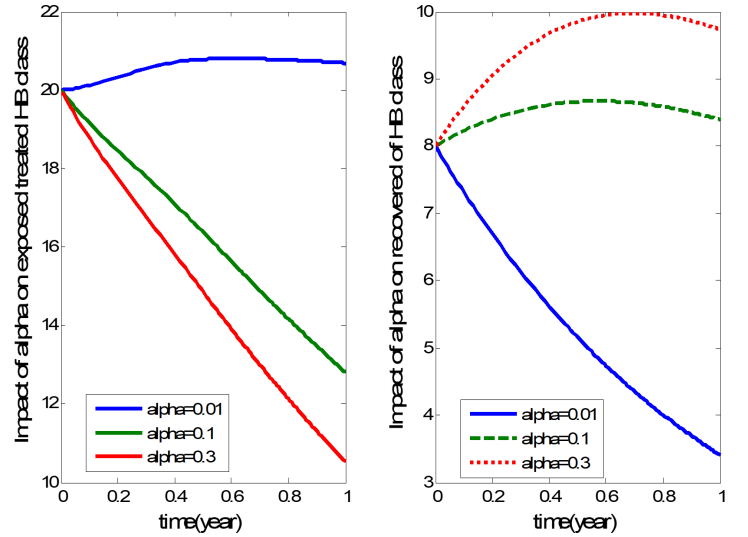

Figure 4.7: Graphs showing the impact of α (the rate at which individuals who are exposed and being treated of HBV recover) in the population.

Figure 4.7(a): The graph on the left, showing the impact of α on exposed treated HB class.
Figure 4.7(b): The graph on the right, showing the impact of α on recovered of HB class.
In figure 4.7(a), we can clearly see that increasing alpha shows a reduction on the number of exposed treated individuals in the population. In figure 4.7(b), increasing alpha shows an increase on the number of people who recovered from HB in the population. As a control measure, people who are diagnosed of being exposed to HBV should be encouraged to go for treatment. From the graphs above, more than 80% of exposed of HBV individuals who are treated of HBV recovered.

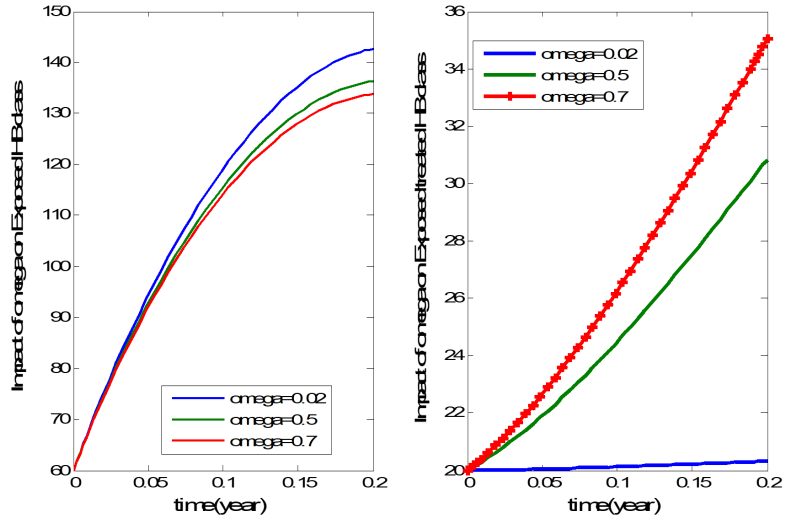

Figure 4.8: Graphs showing the impact of ω (the rate at which individuals who are exposed to HBV enter into exposed and treated compartment) in the population.

Figure 4.8(a): The graph on the left, showing the impact of ω on exposed HB class.
Figure 4.8(b): The graph on the right, showing the impact of ω on exposed treated HBD class. Graph in figure 4.8(a) shows a gradual decrease on the number of exposed HB individuals in the population as the value of omega was increasing. In figure 4.8(b), an increase in omega shows a great increase on the number of exposed treated of HB individuals in the population. The reason for the increase on the number of treated people in the population could be as a result of awareness. When more people are aware of the disease and go for screening, the infected ones will start treatment; this will eventually lead to recovery. Often time, people may not know that they are exposed of HBV until irreversible damage is done to the liver before they will become aware of the infection so as a control strategy, awareness is strongly recommended.

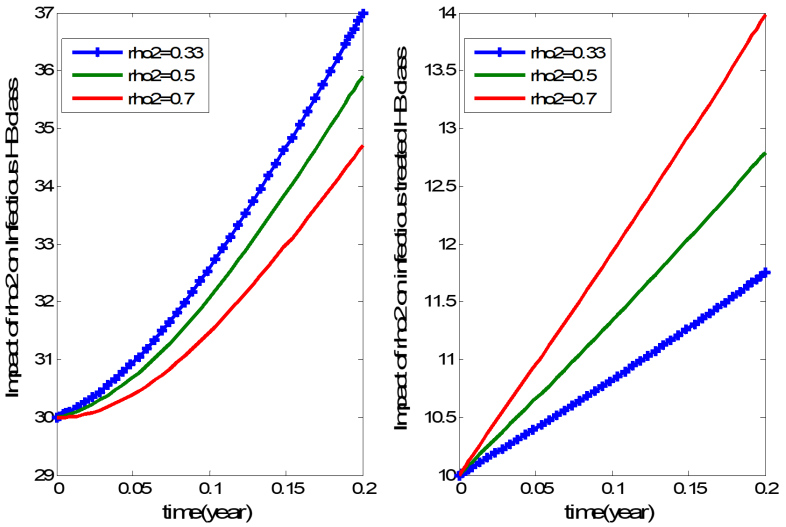

Figure 4.9: Graphs showing the impact of ρ_2 in the population.

Figure 4.9(a): The graph on the left, showing the impact of ρ_2 on infectious HB class.
Figure 4.9(b): The graph on the right, showing the impact of ρ_2 on infectious treated HB class.
From figure 4.9(a), increasing ρ_2 shows a decrease in the number of infectious HB individuals in the population. Thus, an increase in ρ_2 shows a very high increase on the number of infectious treated HB people in the population (see figure 4.9b). From the above graphs, it is obvious that the more people who are infected with HBV go for treatment the less the number of HBV infected individuals in the population. Therefore, as a control strategy, people who are diagnosed of HBV should be encouraged to go for treatment. Government should also help by providing free medical care for HBV infected persons. Also non-governmental organizations should even help in sponsoring medical treatment for HBV patients. Therefore, as a control measure, HBV infected individuals should be made to go for treatment this will help reduce HBV in a population and invariably reduce HBV-HDV co-infection in the population.

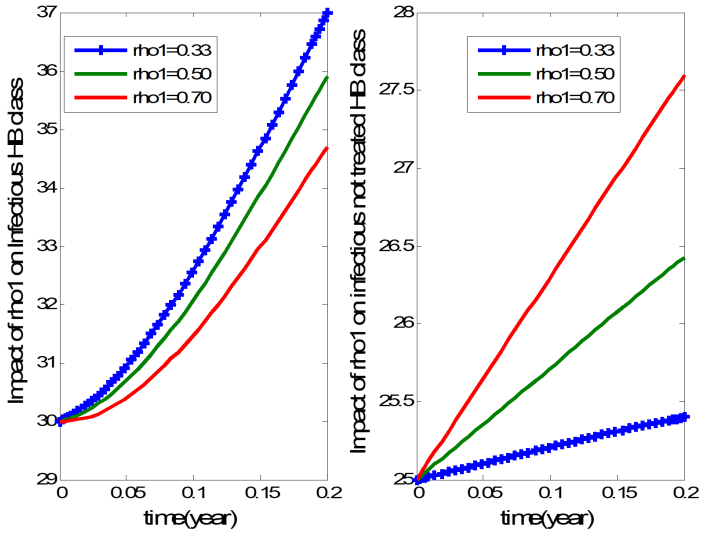

Figure 4.10: Graphs showing the impact of ρ_1 (the rate at which individuals who are infectious of HBV enter into infectious and not being treated of HBV class) in the population.

Figure 4.10(a): The graph on the left, showing the impact of ρ_1 on infectious HB class.
Figure 4.10(b): The graph on the right, showing the impact of ρ_1 on infectious not treated HB class.
From figure 4.10(a), increasing ρ_1 reduces the number of infectious of HB individuals in the population. Also in figure 4.10(b), increasing ρ_1 increases the number of infectious not treated HB individuals in the population. Actually, ρ_1 has a very negative impact or effect in the population. People who are infectious of HBV should be encouraged to go for treatment as recommended earlier. For no reason should people who are diagnosed of HBV stay without going for treatment. Therefore, ρ_1 is not contributing in any way to the control of HBV and HBV/HDV co-infection in the population, concerted effort should be made to avoided this ugly scenario.

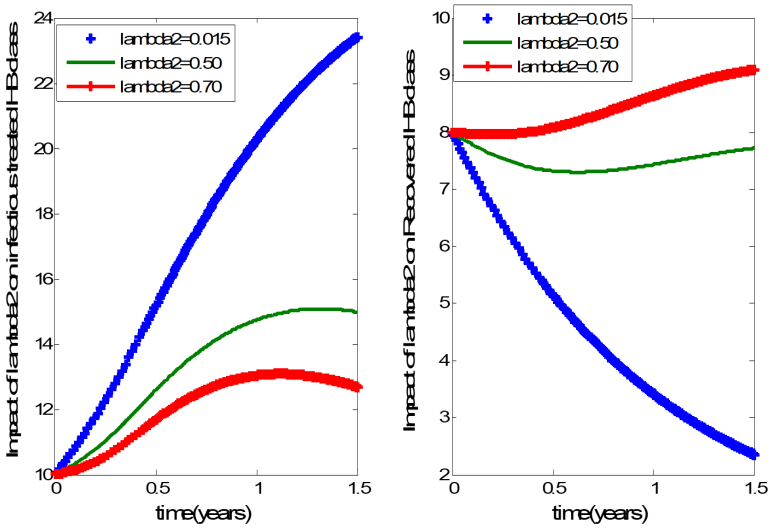

Figure 4.11: Graphs showing the impact of λ_2 (the rate at which individuals who are infectious and being treated of HBV recover from HBV) in the population.

Figure 4.11(a): The graph on the left, showing the impact of λ_2 on infectious treated HB class.
Figure 4.11(b): The graph on the right, showing the impact of λ_2 on recovered HB class.
From figure 4.11(a) above, if λ_2 increases then the number of infectious treated HB people decreases in the population. On the other hand, in figure 4.11(b), increasing λ_2 also increases the number of recovered of HB individuals in the population. This implies that treatment of HBV infected people has a positive impact in the population as more infected individuals will recover after treatment as shown in the graph (figure 4.11b) above. If the infection of HBV is acute, then enough rest is highly recommended and also care should be taken to treat the exhibited symptoms. But on the other hand, if the infection of HBV is chronic, then the use of HBV drugs is recommended. The available drugs for the treatment of HBV are: (1) Lamivudine (LMV) or Ribavirine (2) Eggylated Alpa-interferon (IFN). If more infected HBV people are treated, it will help control HBV infection in the population and in turn control HBV-HDV co-infection . The recovered individuals can even get immunized to avoid contracting it again.
Figure 4.12(a): The graph on the left, showing the impact of λ_1 on exposed HB class.
Figure 4.12(b): The graph on the right, showing the impact of λ_1 on infectious treated HB class.

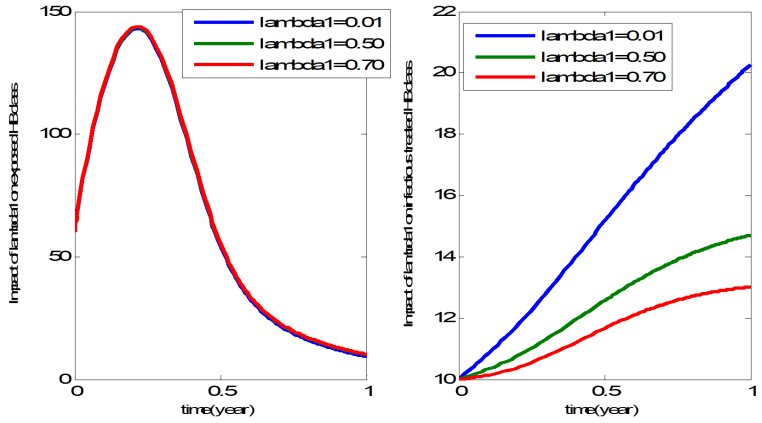

Figure 4.12: Graphs showing the impact of λ_1 (the rate at which individuals that are infectious and being treated of HBV goes back to exposed HBV class) in the population.

In figure 4.12(a) above, we will observe that increasing the value of λ_1 does not show any noticeable increase or decrease on the exposed HB class. On the contrary, in figure 4.12(b), it can be clearly seen that increasing λ_1 decreases the number of infectious treated HB individuals in the population. The condition such as shown here occurs when the disease (HBV) goes into remission in the body of the HBV infected person who is receiving treatment for HBV. When this happens, the patient will not exhibit any symptom(s) of HBV again, and will conclude that he or she has recovered from the infection. From the graphs above, we can see that this situation will arise when more people are infected of HBV and are being treated, some will recover fully and move to recovered class while very few(unnoticeable number) will go back again to exposed class (that is, the disease goes into remission in their body without their knowledge). This has a negative impact in the population and should be avoided. Therefore, as a control strategy, we recommend that the blood of HBV patients who is receiving treatment be screened thoroughly before they are certified recovered from HBV infection. This will help control HBV-HDV co-infection and or HBV-HDV super-infection in the population.

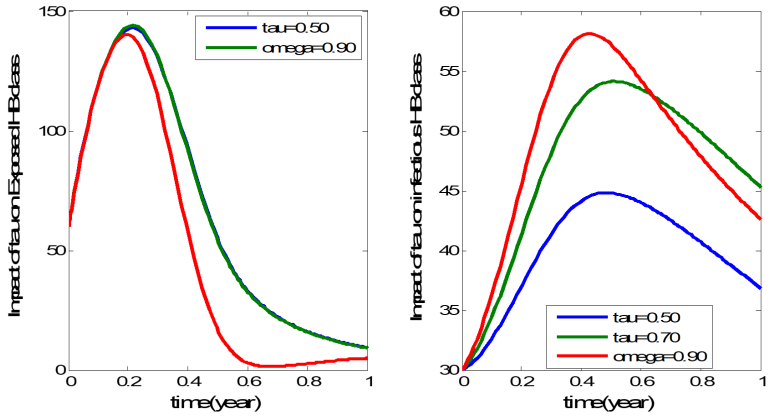

Figure 4.13: Graphs showing the impact of τ (the rate at which individuals who are exposed to HBV become infectious of HBV) in the population.

Figure 4.13(a): The graph on the left, showing the impact of τ on exposed HB class.
Figure 4.13(b): The graph on the right, showing the impact of τ on infectious HB class.
From figure 4.13(a), increasing τ decreases the number of individuals who are exposed of HBV in the population. On the contrary, increasing τ increases the number of infectious individual in the population (see figure 4.13 b). Therefore, efforts should be made through awareness for people to know their HBV status. This if done will help individuals who are exposed of HBV to discover on time and go for treatment so that the person will not migrate to infectious stage. Therefore, as a control strategy, we recommend awareness and that people should have a blood test for the antibody to hepatitis B surface antigen. Early dictation of HBV is an advantage in curing the disease than when it has reached an advanced stage, often time the advance stage would have caused Fibrosis (scarring) and Cirrhosis (hardening of the liver). These conditions are too hard to reverse. This explains why persons diagnosed of HBV that has reached advanced stage often do not survive because of the irreversible damage already done to the liver as a result of the unawareness of the disease.

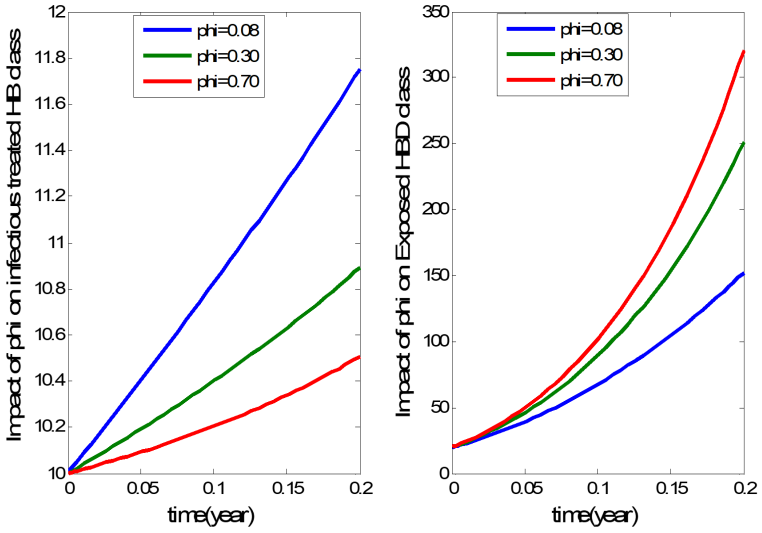

Figure 4.14: Graphs showing the impact of φ (the rate at which individuals that are infectious and being treated of HBV become exposed to HDV) in the population.

Figure 4.14(a): The graph on the left, showing the impact of φ (phi) on infectious treated HB class.
Figure 4.14(b): The graph on the right, showing the impact of φ (phi) on exposed HBD class.
From figure 4.14(a), increasing phi decreases the numbers of infectious treated HB individuals in the population compare to the number of exposed HBD in the population. Also we notice an increase in the number of exposed of HBD people in the population by increasing phi compare to the number of infectious treated HB individuals in the population (see figure 4.14b). This condition has a negative impact on the control of HBV and the resulting consequence is that more people in the population will become exposed to HBD. As a control measure, adequate care should be taken to ensure that HB infectious people who are being treated do not contact HDV, for this will worsen their condition.

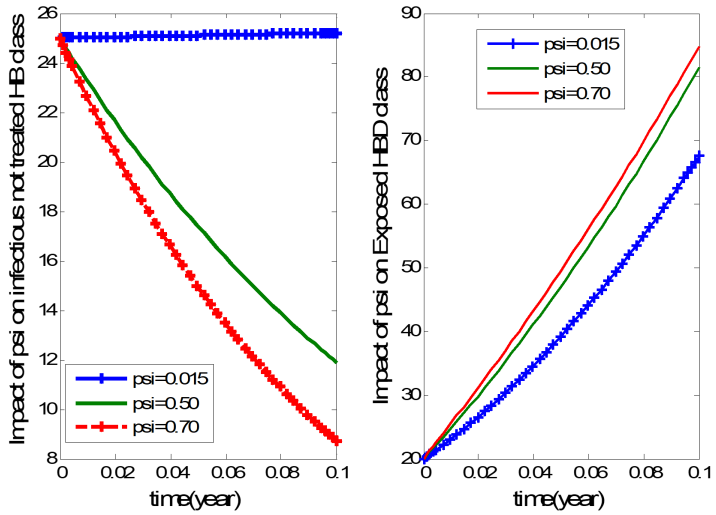

Figure 4.15: Graphs showing the impact of ψ (the rate at which individuals who are infectious of HBV and not being treated become exposed to HDV) in the population.

Figure 4.15(a): The graph on the left, showing the impact of ψ (psi) on infectious not treated HB class.
Figure 4.15(b): The graph on the right, showing the impact of ψ (psi) on exposed HBD class.
In figure 4.15(a), increasing ψ shows a very high decrease on the number of infectious not treated HB individuals in the population. Also in figure 4.15(b), we notice a small increase in the number of exposed of HBD people in the population. This condition again has a negative impact on the control of HBV; hence the resulting consequence is that more people in the population will become exposed to HBD. As a control measure, HB infectious people should be given treatment.

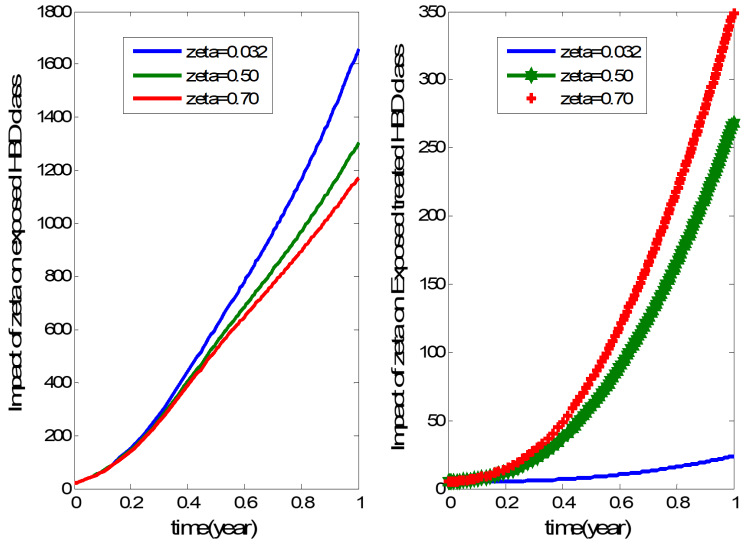

Figure 4.16: Graphs showing the impact of ζ (the rate at which HBV infectious individuals who are exposed to HDV enter into exposed HDV being treated class) in the population.

Figure 4.16(a): The graph on the left, showing the impact of ζ (zeta) on exposed HBD class.
Figure 4.16(b): The graph on the right, showing the impact of ζ (zeta) on exposed treated HBD class.
Graph in figure 4.16(a) shows a gradual decrease on the number of exposed of HBD individuals in the population as omega values increases. In figure 4.16(b), an increase in omega shows a great increase on the number of exposed treated of HBD individuals in the population. The reason for the increase on the number of treated people in the population could be as a result of awareness. When more people are aware of the disease and go for screening, the infected ones will start treatment; this will eventually lead to recovery. Often time, people may not know that they are exposed of HBD until irreversible damage is done to the liver before they will become aware of the infection so as a control strategy, awareness is strongly recommended.

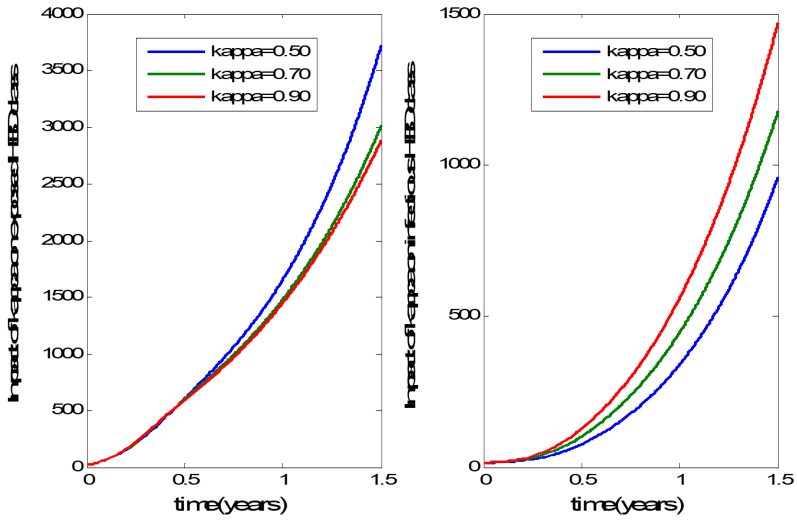

Figure 4.17: Graphs showing the impact of κ (the rate at which HBV infectious individuals who are exposed to HDV become infectious of HBV-HDV) in the population.

Figure 4.17(a): The graph on the left, showing the impact of κ (kappa) on exposed HBD class.
Figure 4.17(b): The graph on the right, showing the impact of κ (kappa) on infectious HBD class.
From figure 4.17(a), increasing κ decreases the number of individuals who are exposed of HBD in the population. Conversely, increasing κ increases the number of infectious of HBD individual in the population (see figure 4.17b). Therefore, intensive effort should be made by the government through awareness so that people will become aware and have their blood tested for the antibody to hepatitis B surface antigen. Hepatitis B surface antigen (HBsAg) is a platform for HDV to replicate and cause infection in the body, therefore early dictation of HBV and or HDV is an advantage in curing the disease than when it has reached an advanced stage, often time the advance stage would have caused Fibrosis (scarring) and Cirrhosis (hardening of the liver). These conditions are too hard to reverse. This explains why persons diagnosed of HBV that has reached advanced stage often do not survive because of the irreversible damage already done to the liver as a result of unawareness of the disease(s). The above measure if followed will help control HBV-HDV co-infection in a population.

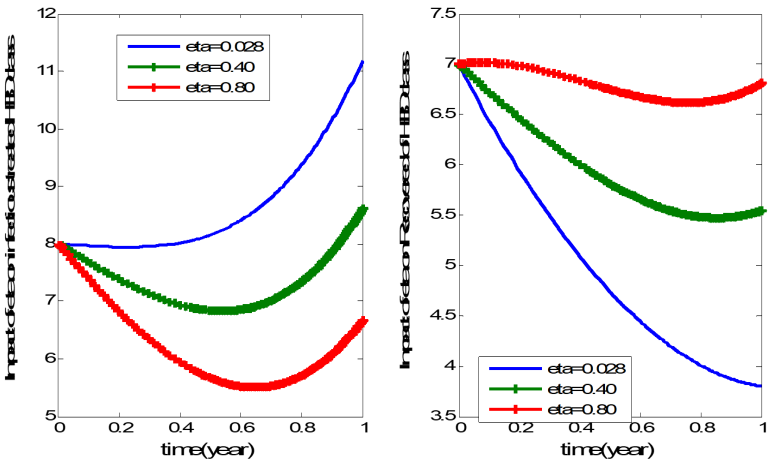

Figure 4.18: Graphs showing the impact of η (the rate at which individuals that are infectious of HBV-HDV and are being treated recover in the population.

Figure 4.18(a): The graph on the left, showing the impact of η (eta) on infectious treated HBD class.
Figure 4.18(b): The graph on the right, showing the impact of η (eta) on recovered of HBD class.
From figure 4.18(a) above, if η increases then the number of infectious treated HBD people decreases in the population. On the other hand, in figure 4.18(b), increasing η also increases the number of recovered of HBD individuals in the population. This implies that treatment of HBD infected people has a positive impact in the population as more infected individuals will recover after treatment as shown in the graph (figure 4.18b) above. Worthy to note are: (i) large doses of Alpha-interferon may be given for up to 12 months for HDV patients (ii) Alpha-interferon can cause the disease (HDV) to go into remission (according to World Health Organisation) (iii) after treatment , people with HDV can still test positive for the condition. Therefore, the best control strategy for HDV and HBV-HDV co-infection in a population is to avoid exposure to HBV (that is, to effectively control HBV in the population).

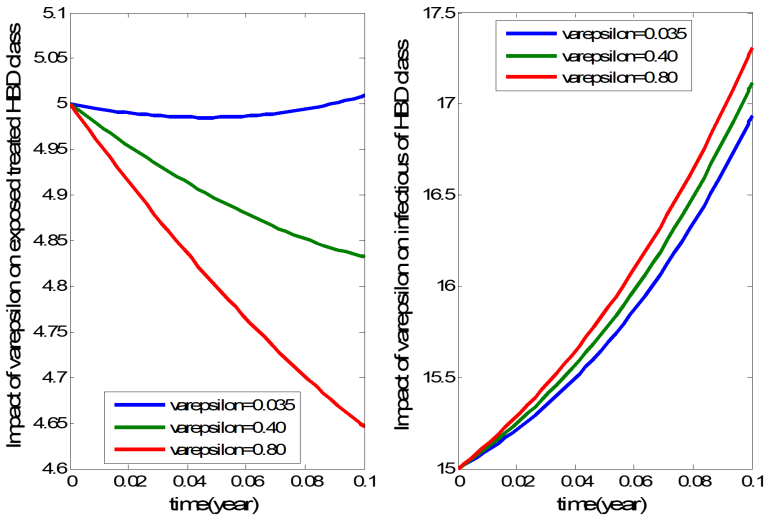

Figure 4.19: Graphs showing the impact of ε (the rate at which HBV infectious individuals who are exposed to HDV and then being treated become infectious of HBV-HDV) in the population.

Figure 4.19(a): The graph on the left shows the impact of ε (epsilon variant) on exposed treated HBD class.
Figure 4.19(b): The graph on the right shows the impact of ε (epsilon variant) on infectious of HBD class.
From figure 4.19(a), increasing epsilon variant decreases the number of exposed treated HBD individuals in the population. Also we notice an increase in the number of infectious of HBD people in the population by increasing epsilon variant (see figure 4.19b). This condition has a negative impact on the control of HBD in the population. As a control measure, adequate care should be taken to ensure that HBD exposed treated people are faithful to their drugs, this will go a long way in helping them recover completely instead of deteriorating to infectious of HBD class .

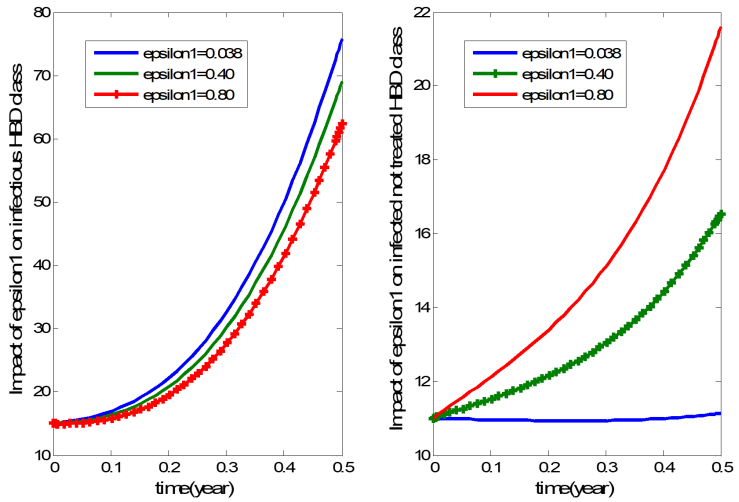

Figure 4.20: Graphs showing the impact of ε_1 (the rate at which individuals who are infectious of HBV-HDV enter into infectious HBV-HDV not being treated class) in the population.

Figure 4.20(a): Graph on the left, showing the impact of ε_1 (epsilon 1) on infectious of HBD class.

Figure 4.20(b): Graph on the right, shows the impact of ε_1 (epsilon 1) on infectious not treated HBD class.

From figure 4.20(a), increasing ε_1 reduces the number of infectious HBD individuals in the population. Also in figure 4.20(b), increasing ε_1 increases the number of infectious not treated HBD individuals in the population. Actually, ε_1 has a very negative impact or effect in the population. People who are infectious of HBD should be encouraged to go for treatment as recommended earlier. For no reason should people who are diagnosed of HBD be allowed to go away without receiving treatment. Therefore, ε_1 is not contributing in any way to the control of HBV in the population, this should be avoided.

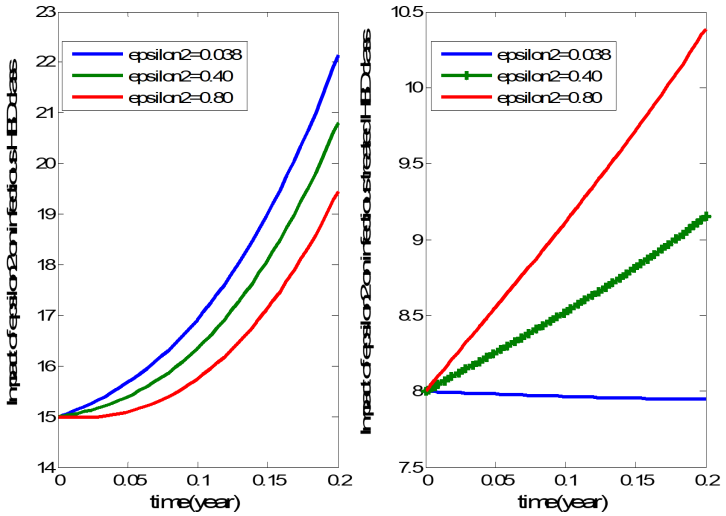

Figure 4.21: Graphs showing the impact of ε_2 (the rate at which individuals who are infectious of HBV-HDV enter into infectious HBV-HDV being treated class) in the population.

Figure 4.21(a): Graph on the left, showing the impact of ε_2 (epsilon 2) on infectious of HBD class.

Figure 4.21(b): Graph on the right, shows the impact of ε_2 (epsilon 2) on infectious treated HBD class.

From figure 4.21(a), increasing ε_2 shows a decrease in the number of infectious of HBD individuals in the population. Conversely, an increase in ε_2 shows a very high increase on the number of infectious treated HBD people in the population (see figure 4.21b). From the above graphs, it is obvious that the more people who are infected with HBD go for treatment the less the number of HBD infected individuals in the population. Therefore, as a control strategy, people who are diagnosed of HBD should be encouraged to go for treatment. Government should also help by providing free medical care for HBD infected persons. Also non-governmental organizations should even help in sponsoring medical treatment for HBD patients. Therefore, as a control measure, HBD infected individuals should be made to go for treatment this will help reduce HBV-HDV co-infection in the population.

5. Conclusion

We investigates the impact of the various parameters of the mathematical model for Hepatitis B virus - Hepatitis D virus (HBV - HDV) co-infection with controls (awareness, vaccine and therapy). Furthermore, simulations are carried out on the equations of the model using MATHLAB and the results are discussed. From the results of the simulation, we observe that awareness, vaccine and therapy are good measure which can be used to effectively control HBV-HDV co-infection in a population. However, awareness and vaccine are better control strategies than therapy. Hence, these simulation results provide the best framework for the control of Hepatitis B virus-Hepatitis D virus (HBV-HDV) co-infection in a population, and effective control of HBV implies effective control of HBV-HDV co-infection in a population.

References

[1] H. Fejza, S. Telaku, *Prevalence of HBV and HCV among blood donors in Kosovo*, Virol. J., 13, (2009), 6-21.
[2] S. A. Kafi-abad, H. Rezvan, H. Abolghasemi, *Trends in prevalence of hepatitis B virus infection among Iranian blood donors*, 1998-2007. Transfus Med., **19**(4), (2009), 189-94
[3] M. Rizzetto, G. Verme, S. Recchia, *Immunflorescence detection of a new antigen-antibody system (delta-antidelta) associated to hepatitis virus in the liver and serum of HBsAg carriers*, Gut., 18, (1977), 996.
[4] A. Smedile, A. Ciancio, M. Rizzetto, *Hepatitis D virus. In: Richman*, D.D, Whitley, R.J, Hayden, F.G, eds. Clinical virology. Washington, DC: ASM Press, (2002), 1227–1240.
[5] S. I. Friedman, *Seminars in medicine of the Beth Israel Hospital, Boston. The cellular basis of hepatic fibrosis. Mechanisms and treatment strategies*, England Journal of Medicine; 328, (1993), 1828–1835.
[6] P. Farci, *Delta hepatitis: an update*, J. Hepatol., 39, Suppl., 1, (2003), 212-9.
[7] S. A. Hughes, H. Wedemeyer, P. M. Harrison, *Hepatitis delta virus*, Lancet, 378, (2011), 73–85.
[8] M. Rizzetto, G. Verme, *Delta hepatitis-present status*, J. Hepatol., 1, (1985), 187-93.
[9] J. M. Taylor, *Hepatitis delta virus*, Virology, 5; **344**(1), (2006), 71-6.
[10] S. M. Alavian, S. H. Alavian, *Hepatitis D virus infection; Iran, Middle East and Central Asia*, Hepatitis Monthly, 5, (2005), 137-143.
[11] R. Esmaeili, S. M. Alavian, B. Hajibeigi, E. Sabouri, R. Edalat, A. Adeli, *Phylogenetic analysis of twenty-six cases of hepatitis delta virus isolates in Tehran, Iran*, Hepat Mon, **9**(3), (2009), 196-200.
[12] Z. Abbas, W. Jafri, S. Raza, *Hepatitis D: Scenario in the Asia-Pacific region*, World J. Gastroenterol, 7, **16**(5), (2010), 554-62.
[13] S. M. Alavian, *Unthought of Problems Regarding Hepatitis D Virus Infection*, Hepat Mon., **10**(2), (2010), 77- 79.
[14] R. O. Aja, D. Omale, G. C. E. Mbah, *Sensitivity Analysis of the Mathematical Model on the Control of HBV-HDV co-infection Transmission Dynamics in a Given Population*, Journal of the Nigerian Association of Mathematical Physics, **39**, (2017), 457 - 470.
[15] R. O. Aja, T. I. Chinebu, E. O. Eze, *On the Stability of Hepatitis B Virus-Hepatitis D Virus (HBV-HDV) co-infection with Controls in a Dynamic Population*, International Journal of Advances in Mathematics, **2019**(2), (2019), 17-30.
[16] H. W. Hethcote, *The Mathematics of Infectious Diseases*, SIAM REVIEW, **42**(4), (2000), 599-653.
[17] S. Abdulrahman, N. I. Akinwande, O. B. Awojoyogbe, U. Y. Abubakar, *Sensitivity Analysis of the parameters of a Mathematical Model of Hepatitis B virus transmission*, Universal Journal of Applied Mathematics, **1**(4), (2013), 230-241.
[18] I. K. Adu, A. Y. Aidoo, I. O. Darko, E. O. Frimpong, *Mathematical Model of Hepatitis B in the Bosomtwe District of Ashanti Region*, Ghana Applied Mathematical Sciences, **8**(64), (2014), 3343 - 3358.
[19] L. Zou, W. Zhang, Modelling the transmission dynamics and control of hepatitis B virus in China, Journal of Theoretical Biology, **10**, (2009), 1-9.
[20] A. R. Kimbir, T. Aboiyar, O. Abu, E. S. Onah, *Simulation of a Mathematical Model of Hepatitis B virus Transmission Dynamics in the presence of vaccination and treatment*, Mathematical Theory and Modelling, **4**(12), (2014).

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Dynamics and Expression of Solution of a Sixth Order Difference Equation

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Abstract

This paper deals with the solution behavior and periodic nature of the solutions of the difference equation

$$s_{n+1} = \alpha s_n + \frac{\beta s_n s_{n-4}}{\gamma s_{n-4} + \delta s_{n-5}}, \quad n = 0, 1, \ldots \tag{0.1}$$

where the initial conditions s_{-5}, s_{-4}, s_{-3}, s_{-2}, s_{-1}, s_0 are arbitrary positive real numbers and α, β, γ, δ are positive constants. Also we obtain the closed form of the solutions of some special cases of this equation.

1. Introduction

This paper deals with the solution behaviour of the difference equation

$$s_{n+1} = \alpha s_n + \frac{\beta s_n s_{n-4}}{\gamma s_{n-4} + \delta s_{n-5}}, \quad n = 0, 1, .. \tag{1.1}$$

where the initial conditions s_{-5}, s_{-4}, s_{-3}, s_{-2}, s_{-1}, s_0, are arbitrary positive real numbers and α, β, γ, δ are positive constants. Also we obtain the form of solution of some special cases of this equation.
Various biological systems naturally leads to their study by means of a discrete variable. Appropriate examples include population dynamics and medicine. Some fundamental models of biological phenomena, including harvesting of fish, a single species population model, ventilation volume and blood CO2 levels, the production of red blood cells, a simple epidemics model, and a model of waves of disease that can be analyzed by difference equations are shown in [1]. Newly, there has been interest in so-called dynamical diseases, which correspond to physiological disorders for which a generally stable control system becomes unstable. One of the first papers on this subject was that of Mackey and Glass [2]. In which they investigated a first-order difference-delay equation that models the concentration of blood-level CO2. They also discussed models of a second class of diseases associated with the production of red cells, white cells, and platelets in the bone marrow. The dynamical characteristics of population system have been modeled, among others by differential equations in the case of species with overlapping generations and by difference equations in the case of species with non-overlapping generations. In process, one can developed a discrete model directly from observations and experiments. Periodically, for numerical purposes, one wants to propose a finite-difference scheme to numerically solved a given differential equation model, especially when the differential equation cannot be solved explicitly. For a given differential equation, a difference equation approximation would be most acceptable if the solution of the difference equation is the same as the differential equation at the discrete points [3]. But unless we can explicitly solve both equations, it is impossible to satisfy this requirements. Most of the time, it is fascinating that a differential equation, when extracted from a difference equation, marmalade the dynamical features of the corresponding continuous-time model such as equilibria, their local and global stability characteristics, and bifurcation behaviors. If alike discrete models can be derived from continuous time models, and it will preserve the considered realities, such discrete-time models can be called ‘dynamically consistent’ with the continuous-time models.

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The study of oscillatory and asymptotic stability properties of solution behavior of difference equations is extremely advantageous in the behavior of various biological system and other applications. This is because difference equations are relevant models for expressing situations where the variable is assumed to take only a discrete set of values and they appear frequently in the formulation and analysis of discrete time systems, in the study of biological systems, the study of deterministic chaos, the numerical integration of differential equations by finite difference schemes and so on. Difference equations are good models for describing situations where population growth is not continuous but seasonal with overlapping generations. "For example, the difference equation

$$\omega_{n+1} = \omega_n e^{\left[r(1-\frac{\phi_n}{k})\right]}$$

has been expressed to model different animal populations.
The generalized Beverton-Holt stock recruitment model has been investigated in [4, 5].

$$z_{n+1} = Az_n + \frac{Bz_{n-1}}{1+Cz_{n-1}+Dz_n}.$$

Several other researchers have studied the behavior of the solution of difference equations for example in [6] E. M. Elsayed investigated the solution of the following non-linear difference equation.

$$w_{n+1} = aw_n + \frac{bw_n^2}{cw_n + dw_{n-1}}.$$

Elabbasy et al. [7] studied the boundedness, global stability, periodicity character and gave the solution of some special cases of the difference equation.

$$y_{n+1} = \frac{Ay_{n-l} + By_{n-k}}{\alpha y_{n-l} + \beta y_{n-k}}.$$

Keratas et al. [8] gave the solution of the following difference equation

$$\ell_{n+1} = \frac{\ell_{n-5}}{1+\ell_{n-2}\ell_{n-5}}.$$

Elabbasy et al. [9] investigated the global stability, periodicity character and gave the solution of some special cases of the difference equation

$$x_{n+1} = \frac{ax_{n-l}x_{n-k}}{bx_{n-p} + cx_{n-q}}.$$

Yalçınkaya et al. [10] has studied the following difference equation

$$x_{n+1} = \alpha + \frac{x_{n-m}}{x_n^k}.$$

Saleh et. al. [11] study the solution of difference

$$y_{n+1} = A + \frac{y_n}{y_{n-k}}.$$

Elsayed et al. [12] studied the global behavior of rational recursive sequence

$$x_{n+1} = ax_{n-l} + \frac{bx_{n-k} + cx_{n-s}}{d + ex_{n-t}}$$

As a matter of fact, numerous papers negotiate with the problem of solving nonlinear difference equations in any way possible, see, for instance [4]-[6], [13]-[18]. The long-term behavior and solutions of rational difference equations of order greater than one has been extensively studied during the last decade. For example, various results about periodicity, boundedness, stability, and closed form solution of the second-order rational difference equations have been investigated see [12]-[15], [19]-[25]. Other related work on rational difference equations see in refs. [26]-[28].
Here, we recall some basic definitions and some theorems that we need in the sequel.

2. Basic definitions

Let I be some interval of real numbers and let

$$F : I^{k+1} \to I,$$

be a continuously differentiable function. Then for every set of initial conditions $s_{-k}, s_{-k+1}, ..., s_0 \in I$, the difference equation

$$s_{n+1} = F(s_n, s_{n-1}, ..., s_{n-k}), \quad n = 0, 1, ..., \tag{2.1}$$

has a unique solution $\{s_n\}_{n=-k}^{\infty}$.
A point $\bar{s} \in I$ is called an equilibrium point of 2.1 if

$$\bar{s} = F(\bar{s}, \bar{s}, ..., \bar{s}).$$

That is, $s_n = \bar{s}$ for $n \geq 0$, is a solution of 2.1 or equivalently $\bar{s}$ is a fixed point of F.

Definition 2.1. *(Periodicity) A Sequence* $\{s_n\}_{n=-k}^{\infty}$ *is said to be periodic with period p if* $s_{n+p} = s_n$ *for all* $n \geq -k$.

Definition 2.2. *(Fibonacci Sequence). The sequence* $\{F_m\}_{m=1}^{\infty} = \{1,2,3,5,8,13,....\}$ *i.e.* $F_m = F_{m-1} + F_{m-2} \geq 0$, $F_{-2} = 0$, $F_{-1} = 1$ *is called Fibonacci Sequence.*

Definition 2.3. *(Stability) (i) The equilibrium point* $\bar{s}$ *of Eq.(1.2) is locally stable if for every* $\varepsilon > 0$, *there exists* $\delta > 0$ *such that for all* $s_{-k}, s_{-k+1}, ..., s_{-1}, s_0 \in I$ *with*

$$|s_{-k} - \bar{s}| + |s_{-k+1} - \bar{s}| + ... + |s_0 - \bar{s}| < \delta,$$

we have

$$|s_n - \bar{s}| < \varepsilon \quad \text{for all} \quad n \geq -k.$$

(ii) The equilibrium point $\bar{s}$ *of 2.1 is locally asymptotically stable if* $\bar{s}$ *is locally stable solution of Eq.(1.2) and there exists* $\gamma > 0$, *such that for all* $s_{-k}, s_{-k+1}, ..., s_{-1}$, $s_0 \in I$ *with*

$$|s_{-k} - \bar{s}| + |s_{-k+1} - \bar{s}| + ... + |s_0 - \bar{s}| < \gamma,$$

we have

$$\lim_{n\to\infty} s_n = \bar{s}.$$

(iii) The equilibrium point $\bar{s}$ *of 2.1 is global attractor if for all* $s_{-k}, s_{-k+1}, ..., s_{-1}$, $s_0 \in I$, *we have*

$$\lim_{n\to\infty} s_n = \bar{s}.$$

(iv) The equilibrium point $\bar{s}$ *of 2.1 is globally asymptotically stable if* $\bar{s}$ *is locally stable, and* $\bar{s}$ *is also a global attractor of 2.1.*
(v) The equilibrium point $\bar{s}$ *of 2.1 is unstable if* $\bar{s}$ *is not locally stable.*
(vi) The linearized equation of 2.1 about the equilibrium $\bar{s}$ *is the linear difference equation*

$$y_{n+1} = \sum_{i=0}^{k} \frac{\partial F(\bar{s}, \bar{s}, ..., \bar{s})}{\partial s_{n-i}} y_{n-i}.$$

Theorem 2.4. *[2] Assume that* $p, q \in R$ *and* $k \in \{0,1,2,...\}$. *Then*

$$|p| + |q| < 1,$$

is a sufficient condition for the asymptotic stability of the difference equation

$$s_{n+1} + p s_n + q s_{n-k} = 0, \quad n = 0, 1, ...$$

Remark 2.5. *Theorem 2.4 can be easily extended to a general linear equation of the form*

$$s_{n+k} + p_1 s_{n+k-1} + ... + p_k s_n = 0, \qquad n = 0, 1, ... \tag{2.2}$$

where $p_1,\ p_2, ..., p_k \in R$ and $k \in \{1,2,...\}$. Then 2.2 is asymptotically stable provided that

$$\sum_{i=1}^{k} |p_i| < 1.$$

Consider the following equation

$$s_{n+1} = g(s_n,\ s_{n-1}, s_{n-2}). \tag{2.3}$$

The following theorem will be useful for the proof of our results in this paper.

Theorem 2.6. *[1] Let* $[\alpha, \beta]$ *be an interval of real numbers and assume that*

$$g : [\alpha, \beta]^3 \to [\alpha, \beta],$$

is a continuous function satisfying the following properties:
(a) $g(x,y,z)$ *is non-decreasing in* x *and* $y \in [\alpha, \beta]$ *for each fixed* $z \in [\alpha, \beta]$.*and* $g(x,y,z)$ *is non-increasing in* $z \in [\alpha, \beta]$ *for each fixed* x *and* $y \in [\alpha, \beta]$
(b) If $(\lambda, \mu) \in [\alpha, \beta] \times [\alpha, \beta]$ *is a solution of the system*

$$\mu = g(\mu,\ \mu,\ \lambda) \qquad \text{and} \qquad \lambda = g(\lambda,\ \lambda,\ \mu),$$

then $\mu = \lambda$,
and 2.3 has a unique equilibrium $\bar{s} \in [\alpha, \beta]$ *and every solution of 2.3 converges to* $\bar{s}$."

3. Main results

3.1. Local stability of the equilibrium point of equation 1.1

This section elaborates the equilibrium point is local stable. Following relation shows the equilibrium points of 1.1

$$\bar{s} = \alpha\bar{s} + \frac{\beta\bar{s}^2}{\gamma\bar{s} + \delta\bar{s}}.$$

or

$$\bar{s}^2(1-\alpha)(\gamma+\delta) = \beta\bar{s}^2$$

If $(1-\alpha)(\gamma+\delta) \neq \beta$, then the unique equilibrium point is $\bar{s} = 0$
Let $f : (0,\infty)^3 \longrightarrow (0,\infty)$ be a continuously differentiable function defined by

$$f(\xi,\eta,\omega) = \alpha\xi + \frac{\beta\xi\eta}{\gamma\eta + \delta\omega}.$$

Therefore at $\bar{s} = 0$

$$\left(\frac{\partial f}{\partial \xi}\right)_{\bar{s}} = \alpha + \frac{\beta v}{(\gamma\eta + \delta\omega)}, \quad \left(\frac{\partial f}{\partial \eta}\right)_{\bar{s}} = \frac{\beta\delta\xi w}{(\gamma\eta + \delta\omega)^2}, \quad \left(\frac{\partial f}{\partial \omega}\right)_{\bar{s}} = \frac{-\beta\delta\xi\eta}{(\gamma\eta + \delta\omega)^2}$$

Then the linearized equation of 1.1 about $\bar{s}$ is

$$y_{n+1} - \left(\alpha + \frac{\beta v}{(\gamma\eta + \delta\omega)}\right) y_n + \left(\frac{\beta\delta u\omega}{(\gamma\eta + \delta\omega)^2}\right) y_{n-1} + \left(\frac{-\beta\delta\xi\eta}{(\gamma\eta + \delta\omega)^2}\right) y_{n-2} = 0. \tag{3.2}$$

Theorem 3.1. *The equilibrium point $\bar{s} = 0$ of (1) is locally asymptotically stable if* $\beta(\gamma+3\delta) < (\gamma+\delta)^2(1-\alpha), \qquad \alpha < 1.$

Proof. It is follows by Theorem A that 3.2 is asymptotically stable if

$$\left|\alpha + \frac{\beta}{(\gamma+\delta)}\right| + \left|\frac{\beta\delta}{(\gamma+\delta)^2}\right| + \left|\frac{-\beta\delta}{(\gamma+\delta)^2}\right| < 1,$$

or

$$\alpha + \frac{\beta\gamma + 3\beta\delta}{(\gamma+\delta)^2} < 1$$

and so

$$\beta(\gamma+3\delta) < (\gamma+\delta)^2(1-\alpha).$$

Which completes the proof. □

3.2. Global attractivity of the equilibrium point of equation 1.1

This section investigate the global attractivity character of solutions of 1.1.

Theorem 3.2. *The equilibrium point $\bar{s}$ of 1.1 is global attractor. if*

$$\gamma(1-\alpha) \neq \beta$$

Proof. Let α, β are real numbers and assume that $g : [\alpha,\beta]^3 \to [\alpha,\beta]$, be a function defined by $g(u,v,w) = \alpha u + \frac{\beta uv}{\gamma v + \delta w}$ then we can easily see that the function $g(u,v,w)$ is increasing in u,v and decreasing in w.
Suppose that $(\lambda,\ \mu)$ is a solution of the system

$$\mu = g(\mu,\ \mu,\ \lambda) \quad \textit{and} \quad \lambda = g(\lambda,\lambda,\ \mu).$$

Then from 1.1 we see that

$$\mu = \alpha\mu + \frac{\beta\mu^2}{\gamma\mu + \delta\lambda}, \quad \lambda = \alpha\lambda + \frac{\beta\lambda^2}{\gamma\lambda + \delta\mu},$$

Therefore,

$$\mu(1-\alpha) = \frac{\beta\mu^2}{\gamma\mu + \delta\lambda}, \quad \lambda(1-\alpha) = \frac{\beta\lambda^2}{\gamma\lambda + \delta\mu},$$

or

$$\beta\mu^2 = \gamma(1-\alpha)\mu^2 + \delta(1-\alpha)\mu\lambda \qquad \textit{and} \beta\lambda^2 = \gamma(1-\alpha)\lambda^2 + \delta(1-\alpha)\mu\lambda,$$

subtracting

$$\gamma(1-\alpha)(\mu^2 - \lambda^2) = \beta(\mu^2 - \lambda^2), \qquad \gamma(1-\alpha) \neq \beta.$$

Thus

$$\mu = \lambda.$$

It follows by the Theorem B that $\bar{x}$ is a global attractor of 1.1 and then the proof is complete. □

4. Boundedness of solutions of 1.1

In this section we study the boundedness of solution of 1.1.

Theorem 4.1. *Every solution of 1.1 is bounded if*

$$(\alpha+\frac{\beta}{\gamma})<1.$$

Proof. Let $\{s_n\}_{n=-5}^{\infty}$ be a solution of 1.1. It follows from 1.1 that □

$$s_{n+1}=\alpha s_n+\frac{\beta s_n s_{n-4}}{\gamma s_{n-4}+\delta s_{n-5}}\leq \alpha s_n+\frac{\beta s_n s_{n-4}}{\gamma s_{n-4}}=(\alpha+\frac{\beta}{\gamma})s_n$$

Then

$$s_{n+1}\leq s_n,\ \text{ for all } n\geq 0$$

Then the sub-sequence $\{s_{5n-1}\}_{n=-5}^{\infty}$, $\{s_{5n-2}\}_{n=-5}^{\infty}$, $\{s_{5n-3}\}_{n=-5}^{\infty}$, $\{s_{5n-4}\}_{n=-5}^{\infty}$, and $\{s_{5n-4}\}_{n=-5}^{\infty}$ are decreasing and so are bounded from above by $M=\max\{s_{-5},\ s_{-4},\ s_{-3},\ s_{-2},\ s_{-1},\ s_0\}$

Example 4.2. *Let* $\alpha=0.03,\beta=0.6,\ \gamma=0.8,\ \delta=0.01$*and* $\alpha=0.3,\ \beta=0.06,\ \gamma=0.7\ \delta=0.01$ *Then 1.1 in this case will be*

$$s_{n+1}=0.03s_n+\frac{0.6s_n s_{n-4}}{0.8s_{n-4}+0.01s_{n-5}} \tag{4.1}$$

$$s_{n+1}=0.3s_n+\frac{0.06s_n s_{n-4}}{0.7s_{n-4}+0.1s_{n-5}} \tag{4.2}$$

with initial condition for 4.1 $s_{-5}=9,\ s_{-4}=4,\ s_{-3}=7,\ s_{-2}=1,\ s_{-1}=10,\ s_0=8$. *The plot for solution of* s_n *is shown in (Figure 4.1) and for 4.2* $s_{-5}=5.2,\ s_{-4}=2.3,\ s_{-3}=1.2,\ s_{-2}=0.07,\ s_{-1}=0.2,\ s_0=0.01$. *The plot for solution of* s_n *is shown in (Figure 4.2.)*

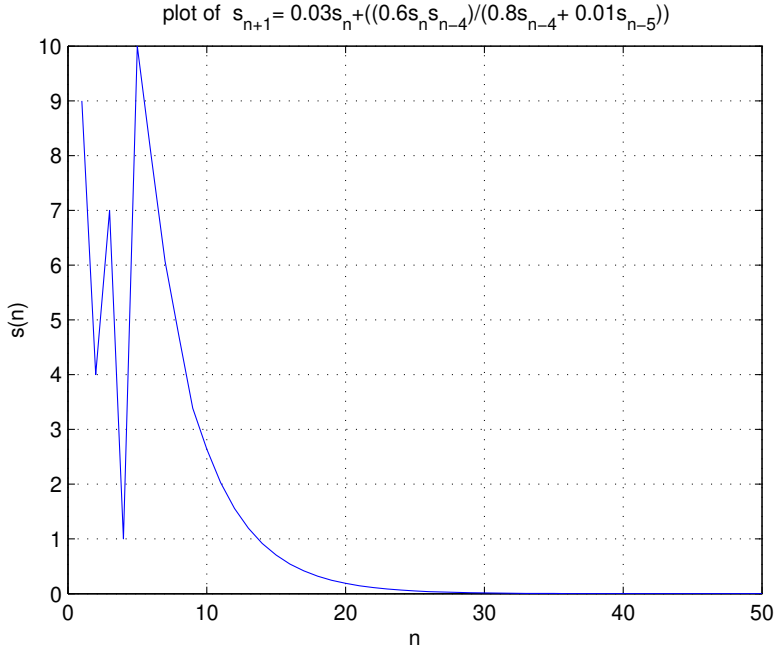

Figure 4.1: Shows Bounded Solution of 4.1

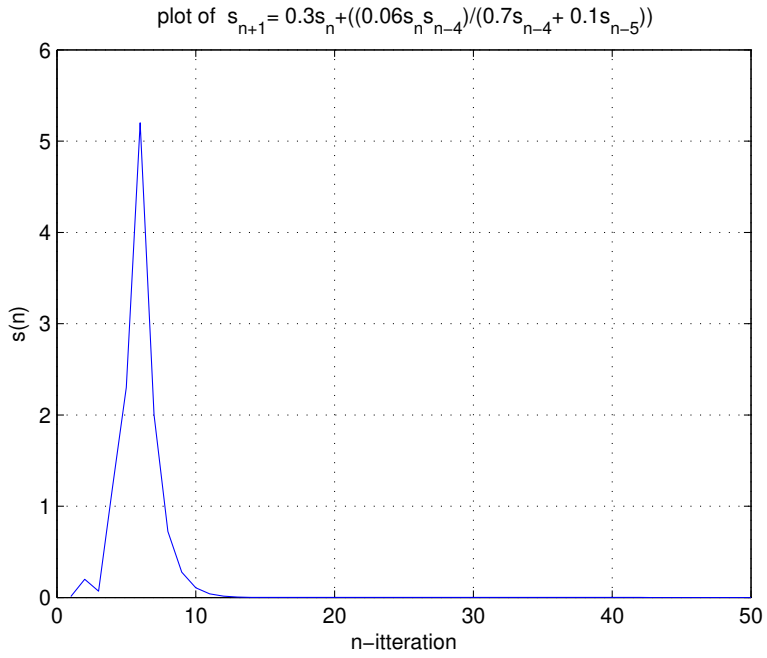

Figure 4.2: Shows Bounded Solution of 4.2

5. Some special cases of 1.1

5.1. First equation

Here we will find the closed form expression of solution of special case of 1.1

$$s_{n+1} = s_n + \frac{s_n s_{n-4}}{s_{n-4} + s_{n-5}}, \qquad n = 0, 1, ... \tag{5.1}$$

where the initial conditions $s_{-5},\ s_{-4},\ s_{-3},\ s_{-2},\ s_{-1},\ s_0$ are arbitrary positive real numbers.

Theorem 5.1. *Let* $\{s_n\}_{n=-5}^{\infty}$ *be a solution of 5.1. Then for* $n = 0, 1, 2, ...$

$$s_{5n} = \varkappa \prod_{i=0}^{n-1} \left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota + f_{2i+2}\rho}{f_{2i+2}\iota + f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma + f_{2i+2}\iota}{f_{2i+2}\sigma + f_{2i+1}\iota}\right)\left(\frac{f_{2i+3}\varkappa + f_{2i+2}\sigma}{f_{2i+2}\varkappa + f_{2i+1}\sigma}\right),$$

$$s_{5n+1} = \varkappa \prod_{i=0}^{n} \left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\rho + f_{2i}\varepsilon}{f_{2i}\rho + f_{2i-1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota + f_{2i}\rho}{f_{2i}\iota + f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}l + f_{2i-1}\sigma}\right),$$

$$s_{5n+2} = \varkappa \prod_{i=0}^{n} \left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota + f_{2i}\rho}{f_{2i}\iota + f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}l + f_{2i-1}k}\right),$$

$$s_{5n+3} = \varkappa \prod_{i=0}^{n} \left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota + f_{2i+2}\rho}{f_{2i+2}\iota + f_{2i+1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}\varkappa + f_{2i-1}\sigma}\right),$$

$$s_{5n+4} = \varkappa \prod_{i=0}^{n} \left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota + f_{2i+2}\rho}{f_{2i+2}\iota + f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma + f_{2i+2}\iota}{f_{2i+2}\sigma + f_{2i+1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}\varkappa + f_{2i-1}\sigma}\right),$$

where $s_{-5} = \varepsilon,\ s_{-4} = \varepsilon,\ s_{-3} = \rho,\ s_{-2} = \iota,\ s_{-1} = \sigma,\ s_0 = \varkappa,\ \ \{f_m\}_{m=1}^{\infty} = \{1, 1, 2, 3, 5, 8, 13, ...\},\ \ f_{-1} = f_0 = 1$

Proof. For $n=0$ result holds. Now suppose that $n>0$ and that our assumption holds for $n-1,\ n-2.$ That is,

$$s_{5n-6}=\varkappa\prod_{i=0}^{n-2}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma+f_{2i+2}\iota}{f_{2i+2}\sigma+f_{2i+1}\iota}\right)\left(\frac{f_{2i+1}\varkappa+f_{2i}\sigma}{f_{2i}\varkappa+f_{2i-1}\sigma}\right),$$

$$s_{5n-5}=\varkappa\prod_{i=0}^{n-2}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma+f_{2i+2}\iota}{f_{2i+2}\sigma+f_{2i+1}\iota}\right)\left(\frac{f_{2i+3}\varkappa+f_{2i+2}\sigma}{f_{2i+2}\varkappa+f_{2i+1}\sigma}\right),$$

$$s_{5n-4}=\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\rho+f_{2i}\varepsilon}{f_{2i}\rho+f_{2i-1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota+f_{2i}\rho}{f_{2i}\iota+f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma+f_{2i}\iota}{f_{2i}\sigma+f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa+f_{2i}\sigma}{f_{2i}\varkappa+f_{2i-1}\sigma}\right),$$

$$s_{5n-3}=\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota+f_{2i}\rho}{f_{2i}\iota+f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma+f_{2i}\iota}{f_{2i}\sigma+f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa+f_{2i}\sigma}{f_{2i}\varkappa+f_{2i-1}\sigma}\right),$$

$$s_{5n-2}=\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+1}\sigma+f_{2i}\iota}{f_{2i}\sigma+f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa+f_{2i}\sigma}{f_{2i}\varkappa+f_{2i-1}\sigma}\right),$$

$$s_{5n-1}=\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma+f_{2i+2}\iota}{f_{2i+2}\sigma+f_{2i+1}\iota}\right)\left(\frac{f_{2i+1}\varkappa+f_{2i}\sigma}{f_{2i}\varkappa+f_{2i-1}\sigma}\right),$$

Now, we see from 5.1 that

$$s_{5n+1}=s_{5n}+\frac{s_{5n}s_{5n-4}}{s_{5n-4}+s_{5n-5}}$$

$$=\left(\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma+f_{2i+2}\iota}{f_{2i+2}\sigma+f_{2i+1}\iota}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\sigma}{f_{2i+2}\varkappa+f_{2i+1}\sigma}\right)\right)$$

$$+\frac{\left(\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1q}}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma+f_{2i+2}\iota}{f_{2i+2}\sigma+f_{2i+1}\iota}\right)\left(\frac{f_{2i+3}\varkappa+f_{2i+2}\sigma}{f_{2i+2}\varkappa+f_{2i+1}\sigma}\right)\right)\times\left(\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\rho+f_{2i}\varepsilon}{f_{2i}\rho+f_{2i-1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota+f_{2ip}}{f_{2i}\iota+f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma+f_{2i}\iota}{f_{2i}\sigma+f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa+f_{2i}\sigma}{f_{2i}\varkappa+f_{2i-1}\sigma}\right)\right)}{\left[\begin{array}{l}\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma+f_{2i+2}\iota}{f_{2i+2}\sigma+f_{2i+1}\iota}\right)\left(\frac{f_{2i+3}\varkappa+f_{2i+2}\sigma}{f_{2i+2}\varkappa+f_{2i+1}k}\right)\\ +\varkappa\prod_{i=0}^{n-2}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma+f_{2i+2}\iota}{f_{2i+2}\sigma+f_{2i+1}\iota}\right)\left(\frac{f_{2i+3}\varkappa+f_{2i+2}\sigma}{f_{2i+2}\varkappa+f_{2i+1}\sigma}\right)\end{array}\right]}$$

$$=\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma+f_{2i+2}\iota}{f_{2i+2}\sigma+f_{2i+1}\iota}\right)\left(\frac{f_{2i+3}\varkappa+f_{2i+2}\sigma}{f_{2i+2}\varkappa+f_{2i+1}\sigma}\right)$$

$$+\frac{\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma+f_{2i+2}\iota}{f_{2i+2}\sigma+f_{2i+1}\iota}\right)\left(\frac{f_{2i+3}\varkappa+f_{2i+2}\sigma}{f_{2i+2}\varkappa+f_{2i+1}\sigma}\right)\left(\frac{f_{2n+1}\varepsilon+f_{2n}\varepsilon}{f_{2n}\varepsilon+f_{2n-1}\varepsilon}\right)}{\left(\frac{f_{2n+1}\varepsilon+f_{2n}\varepsilon}{f_{2n}\varepsilon+f_{2n-1}\varepsilon}\right)+1}$$

$$=\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma+f_{2i+2}\rho}{f_{2i+2}\sigma+f_{2i+1}\iota}\right)\left(\frac{f_{2i+3}\varkappa+f_{2i+2}\sigma}{f_{2i+2}\varkappa+f_{2i+1}\sigma}\right)$$

$$+\frac{\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma+f_{2i+2}\iota}{f_{2i+2}\sigma+f_{2i+1}\iota}\right)\left(\frac{f_{2i+3}\varkappa+f_{2i+2}\sigma}{f_{2i+2}\varkappa+f_{2i+1}\sigma}\right)(f_{2n}\varepsilon+f_{2n-1}\varepsilon)}{f_{2n+1}\varepsilon+f_{2n}\varepsilon+f_{2n}\varepsilon+f_{2n-1}\varepsilon}$$

$$=\varkappa\prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho+f_{2i+2}\varepsilon}{f_{2i+2}\rho+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota+f_{2i+2}\rho}{f_{2i+2}\iota+f_{2i+1}\rho}\right)\left(\frac{f_{2i+3}\sigma+f_{2i+2}\iota}{f_{2i+2}\sigma+f_{2i+1}\iota}\right)\left(\frac{f_{2i+3}\varkappa+f_{2i+2}\sigma}{f_{2i+2}\varkappa+f_{2i+1}\sigma}\right)\left(1+\frac{f_{2n+1}\varepsilon+f_{2n}\varepsilon}{f_{2n+2}\varepsilon+f_{2n+1}\varepsilon}\right)$$

Thus,

$$s_{5n+1}=\varkappa\prod_{i=0}^{n}\left(\frac{f_{2i+3}\varepsilon+f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon+f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\rho+f_{2i}\varepsilon}{f_{2i}\rho+f_{2i-1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota+f_{2i}\rho}{f_{2i}\iota+f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma+f_{2i}\iota}{f_{2i}\sigma+f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa+f_{2i}\sigma}{f_{2i}\varkappa+f_{2i-1}\sigma}\right)$$

Again, it follows from equation 5.1 that,

$$s_{5n+3}=s_{5n+2}+\frac{s_{5n+2}s_{5n-2}}{s_{5n-2}+s_{5n-3}}$$

$$= \varkappa \prod_{i=0}^{n} \left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota + f_{2i}\rho}{f_{2i}\iota + f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}\varkappa + f_{2i-1}\sigma}\right)$$

$$+ \frac{\left(\varkappa \prod_{i=0}^{n}\left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota + f_{2i}\rho}{f_{2i}\iota + f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}\varkappa + f_{2i-1}\sigma}\right)\right)\times \left(\varkappa \prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota + f_{2i+2}\rho}{f_{2i+2}\iota + f_{2i+1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}\varkappa + f_{2i-1}\sigma}\right)\right)}{\left[\begin{array}{l}\left(\varkappa \prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota + f_{2i+2}\rho}{f_{2i+2}\iota + f_{2i+1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}\varkappa + f_{2i-1}\sigma}\right)\right) \\ + \left(\varkappa \prod_{i=0}^{n-1}\left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota + f_{2i}\rho}{f_{2i}\iota + f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}\varkappa + f_{2i-1}\sigma}\right)\right)\end{array}\right]}$$

$$= \prod_{i=0}^{n} \left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota + f_{2i}\rho}{f_{2i}\iota + f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}\varkappa + f_{2i-1}\sigma}\right)$$

$$+ \left[\frac{\varkappa \prod_{i=0}^{n}\left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota + f_{2i}\rho}{f_{2i}\iota + f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}\varkappa + f_{2i-1}\sigma}\right)\left(\frac{f_{2n+1}\iota + f_{2n}\rho}{f_{2n}\iota + f_{2n-1}\rho}\right)}{\left(\frac{f_{2n+1}\iota + f_{2n}\rho}{f_{2n}\iota + f_{2n-1}\rho}\right) + 1}\right]$$

$$= \varkappa \prod_{i=0}^{n} \left[\left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota + f_{2i}\rho}{f_{2i}\iota + f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}\varkappa + f_{2i-1}\sigma}\right)\left(1 + \frac{f_{2n+1}\iota + f_{2n}\rho}{f_{2n+1}\iota + f_{2n}\rho + f_{2n}\iota + f_{2n-1}\rho}\right)\right]$$

$$= \varkappa \prod_{i=0}^{n} \left[\left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+1}\iota + f_{2i}\rho}{f_{2i}\iota + f_{2i-1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}\varkappa + f_{2i-1}\sigma}\right)\left(1 + \frac{f_{2n+1}\iota + f_{2n}\rho}{f_{2n+2}\iota + f_{2n+1}\rho}\right)\right]$$

Therefore

$$s_{5n+3} = \varkappa \prod_{i=0}^{n} \left(\frac{f_{2i+3}\varepsilon + f_{2i+2}\varepsilon}{f_{2i+2}\varepsilon + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\rho + f_{2i+2}\varepsilon}{f_{2i+2}\rho + f_{2i+1}\varepsilon}\right)\left(\frac{f_{2i+3}\iota + f_{2i+2}\rho}{f_{2i+2}\iota + f_{2i+1}\rho}\right)\left(\frac{f_{2i+1}\sigma + f_{2i}\iota}{f_{2i}\sigma + f_{2i-1}\iota}\right)\left(\frac{f_{2i+1}\varkappa + f_{2i}\sigma}{f_{2i}\varkappa + f_{2i-1}\sigma}\right).$$

Other relations can be done similarly. So, the proof is completed.

□

Example 5.2. *To confirm the result in this case we consider numerical example. Let* $\alpha = 1$, $\beta = 1$, $\gamma = 1$, $\delta = 1$. *Then 1.1 in this case will be*

$$s_{n+1} = s_n + \frac{s_n s_{n-4}}{s_{n-4} + s_{n-5}} \tag{5.2}$$

with initial condition $s_{-5} = 2$, $s_{-4} = 8$, $s_{-3} = 5$, $s_{-2} = 3$, $s_{-1} = 1$, $s_0 = 6$. *The plot for solution of* s_n *is shown in Figure 5.1.)*

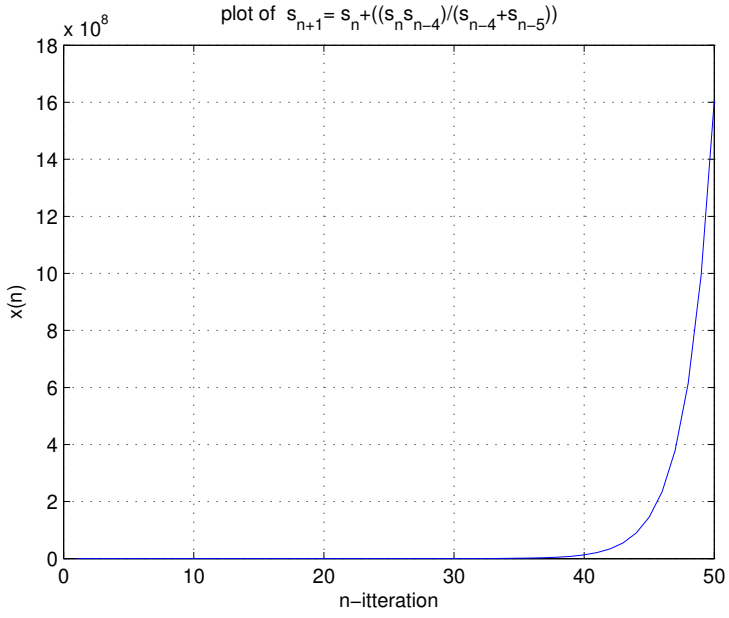

Figure 5.1: Shows Unbounded Solution of 5.2

5.2. Second equation

In this section we solve the specific form of the 1.1

$$s_{n+1} = s_n + \frac{s_n s_{n-4}}{s_{n-4} - s_{n-5}}, \qquad n = 0, 1, ... \tag{5.3}$$

where the initial conditions s_{-5}, s_{-4}, s_{-3}, s_{-2}, s_{-1}, s_0 are arbitrary positive real numbers.

Theorem 5.3. *Let $\{s_n\}_{n=-5}^{\infty}$ be a solution of 5.3. Then for $n = 0, 1, 2, ...$*

$$s_{5n} = \vartheta \prod_{i=0}^{n-1} \left(\frac{f_{i+3}\sigma - f_{i+1}\omega}{f_{i+1}\sigma - f_{i-1}\omega}\right)\left(\frac{f_{i+3}\rho - f_{i+1}\sigma}{f_{i+1}\rho - f_{i-1}\sigma}\right)\left(\frac{f_{i+3}r - f_{i+1}\rho}{f_{i+1}r - f_{i-1}\rho}\right)\left(\frac{f_{i+3}k - f_{i+1}r}{f_{i+1}k - f_{i-1}r}\right)\left(\frac{f_{i+3}\vartheta - f_{i+1}k}{f_{i+1}\vartheta - f_{i-1}k}\right),$$

$$s_{5n+1} = \vartheta\left(\tfrac{2\sigma-\omega}{\sigma-\omega}\right) \prod_{i=0}^{n-1} \left(\frac{f_{i+4}\sigma - f_{i+2}\omega}{f_{i+2}\sigma - f_i\omega}\right)\left(\frac{f_{i+3}\rho - f_{i+1}\sigma}{f_{i+1}\rho - f_{i-1}\sigma}\right)\left(\frac{f_{i+3}r - f_{i+1}\rho}{f_{i+1}r - f_{i-1}\rho}\right)\left(\frac{f_{i+3}k - f_{i+1}r}{f_{i+1}k - f_{i-1}r}\right)\left(\frac{f_{i+3}\vartheta - f_{i+1}k}{f_{i+1}\vartheta - f_{i-1}k}\right),$$

$$s_{5n+2} = \vartheta\left(\tfrac{2\sigma-\omega}{\sigma-\omega}\right)\left(\tfrac{2\rho-\sigma}{\rho-\sigma}\right) \prod_{i=0}^{n-1} \left(\frac{f_{i+4}\sigma - f_{i+2}\omega}{f_{i+2}\sigma - f_i\omega}\right)\left(\frac{f_{i+4}\rho - f_{i+2}\sigma}{f_{i+2}\rho - f_i\sigma}\right)\left(\frac{f_{i+3}r - f_{i+1}\rho}{f_{i+1}r - f_{i-1}\rho}\right)\left(\frac{f_{i+3}k - f_{i+1}r}{f_{i+1}k - f_{i-1}r}\right)\left(\frac{f_{i+3}\vartheta - f_{i+1}k}{f_{i+1}\vartheta - f_{i-1}k}\right),$$

$$s_{5n+3} = \vartheta\left(\tfrac{2\sigma-\omega}{\sigma-\omega}\right)\left(\tfrac{2\rho-\sigma}{\rho-\sigma}\right)\left(\tfrac{2r-\rho}{r-\rho}\right) \prod_{i=0}^{n-1} \left(\frac{f_{i+4}\sigma - f_{i+2}\omega}{f_{i+2}\sigma - f_i\omega}\right)\left(\frac{f_{i+4}\rho - f_{i+2}\sigma}{f_{i+2}\rho - f_i\sigma}\right)\left(\frac{f_{i+4}r - f_{i+2}\rho}{f_{i+2}r - f_i\rho}\right)\left(\frac{f_{i+3}k - f_{i+1}r}{f_{i+1}k - f_{i-1}r}\right)\left(\frac{f_{i+3}\vartheta - f_{i+1}k}{f_{i+1}\vartheta - f_{i-1}k}\right),$$

$$s_{5n+4} = \vartheta\left(\tfrac{2\sigma-\omega}{\sigma-\omega}\right)\left(\tfrac{2\rho-\sigma}{\rho-\sigma}\right)\left(\tfrac{2r-\rho}{r-\rho}\right)\left(\tfrac{2k-r}{k-r}\right) \prod_{i=0}^{n-1} \left(\frac{f_{i+4}\sigma - f_{i+2}\omega}{f_{i+2}\sigma - f_i\omega}\right)\left(\frac{f_{i+4}\rho - f_{i+2}\sigma}{f_{i+2}\rho - f_i\sigma}\right)\left(\frac{f_{i+4}r - f_{i+2}\rho}{f_{i+2}r - f_i\rho}\right)\left(\frac{f_{i+4}k - f_{i+2}r}{f_{i+2}k - f_i r}\right)\left(\frac{f_{i+3}\vartheta - f_{i+1}k}{f_{i+1}\vartheta - f_{i-1}k}\right),$$

where $s_{-5} = \omega$, $s_{-4} = \sigma$, $s_{-3} = \rho$, $s_{-2} = r$, $s_{-1} = k$, $s_0 = \vartheta$, $\{f_m\}_{m=1}^{\infty} = \{1, 1, 2, 3, 5, 8, 13, ...\}$, $f_{-1} = f_0 = 1$

Proof. Same as the Theorem 5.1 and is omitted. □

Example 5.4. *We will confirm our result by considering some numerical examples. Assume $s_{-5} = 1$, $s_{-4} = 3$, $s_{-3} = 2$, $s_{-2} = 9$, $s_{-1} = 6$, $s_0 = 7$ (see Figure 5.2) and $s_{-5} = 13$, $s_{-4} = 12$, $s_{-3} = 18$, $s_{-2} = 16$, $s_{-1} = 15$, $s_0 = 10$ (see behavior of solution of 5.3 Figure 5.3).*

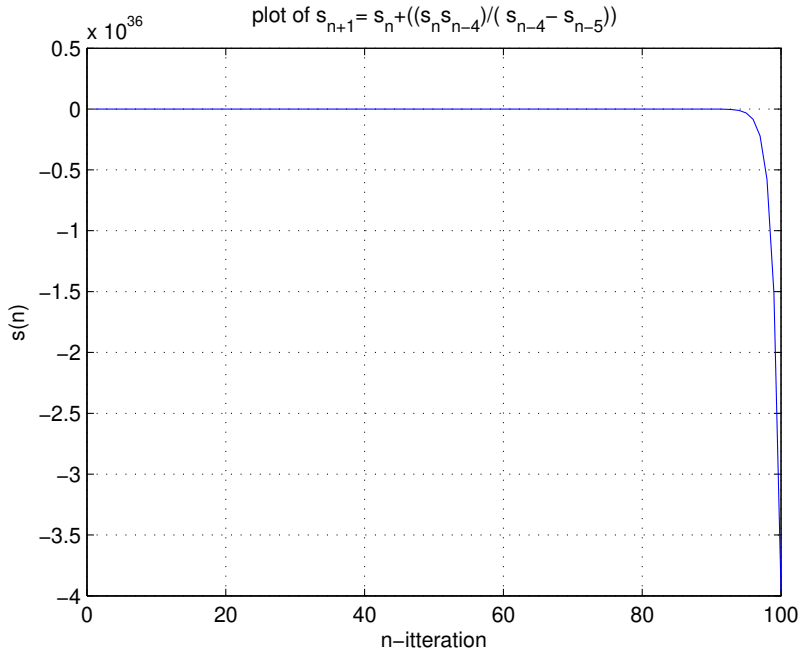

Figure 5.2

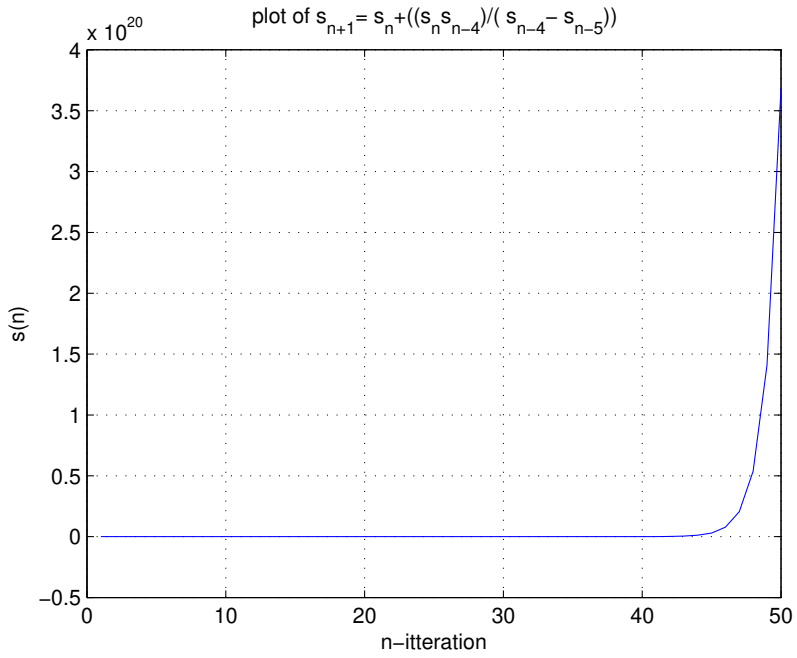

Figure 5.3

5.3. Third equation

In this section we deal with the specific form of the 1.1

$$s_{n+1} = s_n - \frac{s_n s_{n-4}}{s_{n-4} + s_{n-5}}, \qquad n = 0, 1, ... \tag{5.4}$$

where the initial conditions $s_{-5},\ s_{-4},\ s_{-3},\ s_{-2},\ s_{-1},\ s_0$ are arbitrary positive real numbers.

Theorem 5.5. *Let* $\{s_n\}_{n=-5}^{\infty}$ *be a solution of 5.4. Then for* $n = 0, 1, 2, ...$

$$s_{5n} = \frac{lkrpqt}{(f_n q + f_{n+1} t)(f_n p + f_{n+1} q)(f_n r + f_{n+1} p)(f_n k + f_{n+1} r)(f_n l + f_{n+1} k)},$$

$$s_{5n+1} = \frac{lkrpqt}{(f_{n+1} q + f_{n+2} t)(f_n p + f_{n+1} q)(f_n r + f_{n+1} p)(f_n k + f_{n+1} r)(f_n l + f_{n+1} k)},$$

$$s_{5n+2} = \frac{lkrpqt}{(f_{n+1} q + f_{n+2} t)(f_{n+1} p + f_{n+2} q)(f_n r + f_{n+1} p)(f_n k + f_{n+1} r)(f_n l + f_{n+1} k)},$$

$$s_{5n+3} = \frac{lkrpqt}{(f_{n+1} q + f_{n+2} t)(f_{n+1} p + f_{n+2} q)(f_{n+1} r + f_{n+2} p)(f_n k + f_{n+1} r)(f_n l + f_{n+1} k)},$$

$$s_{5n+4} = \frac{lkrpqt}{(f_{n+1} q + f_{n+2} t)(f_{n+1} p + f_{n+2} q)(f_{n+1} r + f_{n+2} p)(f_{n+1} k + f_{n+2} r)(f_n l + f_{n+1} k)}.$$

Where $s_{-5} = t,\ s_{-4} = q,\ s_{-3} = p,\ s_{-2} = r,\ s_{-1} = k,\ s_0 = l\ ,\quad \{f_m\}_{m=1}^{\infty} = \{1, 1, 2, 3, 5, 8, 13, ...\}\quad f_0 = 1.$

Proof. For $n = 0$, the result holds. Now suppose that $n > 0$ and that our supposition holds for $n-1,\ n-2$. That is

$$s_{5n-6} = \frac{lkrpqt}{(f_{n-1} q + f_n t)(f_{n-1} p + f_n q)(f_{n-1} r + f_n p)(f_{n-1} k + f_n r)(f_{n-2} l + f_{n-1} k)},$$

$$s_{5n-5} = \frac{lkrpqt}{(f_{n-1} q + f_n t)(f_{n-1} p + f_n q)(f_{n-1} r + f_n p)(f_{n-1} k + f_n r)(f_{n-1} l + f_n k)},$$

$$s_{5n-4} = \frac{lkrpqt}{(f_n q + f_{n-1} t)(f_n p + f_{n+1} q)(f_n r + f_{n+1} p)(f_n k + f_{n+1} r)(f_n l + f_{n+1} k)},$$

$$s_{5n-3} = \frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_{n-1}r+f_np)(f_{n-1}k+f_nr)(f_{n-1}l+f_nk)},$$

$$s_{5n-2} = \frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_{n-1}k+f_nr)(f_{n-1}l+f_nk)},$$

$$s_{5n-1} = \frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_nk+f_{n+1}r)(f_{n-1}l+f_nk)},$$

Now, from equation 5.1, we see that,

$$s_{5n} = s_{5n-1} - \frac{s_{5n-1}s_{5n-5}}{s_{5n-5}+s_{5n-6}}$$

$$= \frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_nk+f_{n+1}r)(f_{n-1}l+f_nk)}$$
$$- \frac{\left(\frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_nk+f_{n+1}r)(f_{n-1}l+f_nk)}\right)\left(\frac{lkrpqt}{(f_{n-1}q+f_nt)(f_{n-1}p+f_nq)(f_{n-1}r+f_np)(f_{n-1}k+f_nr)(f_{n-1}l+f_nk)}\right)}{\left[\begin{array}{l}\left(\frac{lkrpqt}{(f_{n-1}q+f_nt)(f_{n-1}p+f_nq)(f_{n-1}r+f_np)(f_{n-1}k+f_nr)(f_{n-1}l+f_nk)}\right)\\ +\left(\frac{lkrpqt}{(f_{n-1}q+f_nt)(f_{n-1}p+f_nq)(f_{n-1}r+f_np)(f_{n-1}k+f_nr)(f_{n-2}l+f_{n-1}k)}\right)\end{array}\right]}$$

$$= \frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_nk+f_{n+1}r)(f_{n-1}l+f_nk)}$$
$$- \frac{\frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_nk+f_{n+1}r)(f_{n-1}l+f_nk)}\left(\frac{1}{f_{n-1}l+f_nk}\right)}{\left[\frac{1}{(f_{n-1}l+f_nk)}+\frac{1}{(f_{n-2}l+f_{n-1}k)}\right]}$$

$$= \frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_nk+f_{n+1}r)(f_{n-1}l+f_nk)}$$
$$- \left[\frac{\left(\frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_nk+f_{n+1}r)(f_{n-1}l+f_nk)}\right)(f_{n-2}l+f_{n-1}k)}{f_{n-1}l+f_nk+f_{n-2}l+f_{n-1}k}\right]$$

$$= \frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_nk+f_{n+1}r)(f_{n-1}l+f_nk)}\left(1-\frac{f_{n-2}l+f_{n-1}k}{f_nl+f_{n+1}k}\right)$$

$$= \frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_nk+f_{n+1}r)(f_{n-1}l+f_nk)}\left(\frac{f_{n-1}l+f_nk}{f_nl+f_{n+1}k}\right)$$

Therefore,

$$s_{5n} = \frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_nk+f_{n+1}r)(f_nl+f_{n+1}k)}$$

Now, from equation 5.4

$$s_{5n+4} = s_{5n+3} - \frac{s_{5n+3}s_{5n-1}}{s_{5n-1}+s_{5n-2}}$$

$$= \frac{lkrpqt}{(f_{n+1}q+f_{n+2}t)(f_{n+1}p+f_{n+2}q)(f_{n+1}r+f_{n+2}p)(f_nk+f_{n+1}r)(f_nl+f_{n+1}k)}$$
$$- \frac{\left[\begin{array}{c}\left(\frac{lkrpqt}{(f_{n+1}q+f_{n+2}t)(f_{n+1}p+f_{n+2}q)(f_{n+1}r+f_{n+2}p)(f_nk+f_{n+1}r)(f_nl+f_{n+1}k)}\right)\times\\ \left(\frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_nk+f_{n+1}r)(f_{n-1}l+f_nk)}\right)\end{array}\right]}{\left[\begin{array}{c}\frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_nk+f_{n+1}r)(f_{n-1}l+f_nk)}\\ +\frac{lkrpqt}{(f_nq+f_{n+1}t)(f_np+f_{n+1}q)(f_nr+f_{n+1}p)(f_{n-1}k+f_nr)(f_{n-1}l+f_nk)}\end{array}\right]}$$

$$= \frac{\left(\frac{lkrpqt}{(f_{n+1}q+f_{n+2}t)(f_{n+1}p+f_{n+2}q)(f_{n+1}r+f_{n+2}p)(f_nk+f_{n+1}r)(f_nl+f_{n+1}k)}\right)\left(\frac{1}{f_nk+f_{n+1}r}\right)}{\left[\frac{1}{f_nk+f_{n+1}r}+\frac{1}{f_{n-1}k+f_nr}\right]}$$

$$= \frac{lkrpqt}{(f_{n+1}q+f_{n+2}t)(f_{n+1}p+f_{n+2}q)(f_{n+1}r+f_{n+2}p)(f_nk+f_{n+1}r)(f_nl+f_{n+1}k)}\left[1-\frac{f_{n-1}k+f_nr}{(f_{n-1}k+f_nr)+(f_nk+f_{n+1}r)}\right]$$

$$= \frac{lkrpqt}{(f_{n+1}q+f_{n+2}t)(f_{n+1}p+f_{n+2}q)(f_{n+1}r+f_{n+2}p)(f_nk+f_{n+1}r)(f_nl+f_{n+1}k)}\left[1-\frac{f_{n-1}k+f_nr}{f_{n+1}k+f_{n+2}r}\right]$$

Therefore,

$$s_{5n+4} = \frac{lkrpqt}{(f_{n+1}q+f_{n+2}t)(f_{n+1}p+f_{n+2}q)(f_{n+1}r+f_{n+2}p)(f_{n+1}k+f_{n+2}r)(f_nl+f_{n+1}k)}.$$

Remaining relations can be found similarly. Hence, the proof is completed. □

Example 5.6. *Assume* $s_{-5}=1,\ s_{-4}=3,\ s_{-3}=6,\ s_{-2}=5,\ s_{-1}=2,\ s_0=7$. *(Figure 5.4, shows behavior of solution of 5.4)*

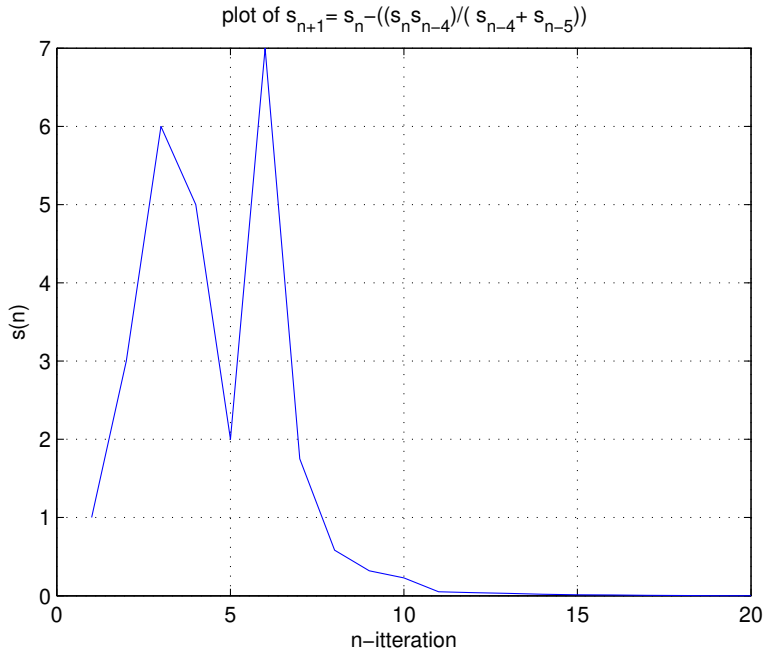

Figure 5.4: Shows behavior of Solution of 5.4

5.4. Fourth equation

In this section we deal with the specific form of the 1.1

$$s_{n+1} = s_n - \frac{s_n s_{n-4}}{s_{n-4}-s_{n-5}}, \quad n=0,1,... \tag{5.5}$$

where the initial conditions $s_{-5},\ s_{-4},\ s_{-3},\ s_{-2},\ s_{-1},\ s_0$ are arbitrary non-zero real numbers with $s_{-5} \neq s_{-4} \neq s_{-3} \neq s_{-2} \neq s_{-1} \neq s_0$.

Theorem 5.7. *Let* $\{s_n\}_{n=-5}^{\infty}$ *be a solution of 5.2. Then every solution of it is periodic with period 24. Moreover,* $\{s_n\}_{n=-5}^{\infty}$ *takes the form*

$$t,\ q,\ p,\ r,\ k,\ l,\ \frac{tl}{l-k},\ \frac{tql}{(t-q)(q-p)},\ \frac{tqpl}{(t-q)(q-p)(p-r)},\ \frac{tqprl}{(t-q)(q-p)(p-r)(r-k)},\ \frac{tqprkl}{(t-q)(q-p)(p-r)(r-k)(k-l)},\ \frac{-tprkl}{(q-p)(p-r)(r-k)(k-l)},$$

or,

$$
\begin{aligned}
s_{24n-5} &= t, & s_{24n-4} &= q,\\
s_{24n-3} &= p, & s_{24n-2} &= r,\\
s_{24n-1} &= k, & s_{24n} &= l,\\
s_{24n+1} &= \frac{tl}{l-k}, & s_{24n+2} &= \frac{tql}{(t-q)(q-p)},\\
s_{24n+3} &= \frac{tqpl}{(t-q)(q-p)(p-r)}, & s_{24n+4} &= \frac{tqprl}{(t-q)(q-p)(p-r)(r-k)},\\
s_{24n+5} &= \frac{tqprkl}{(t-q)(q-p)(p-r)(r-k)(k-l)}, & s_{24n+6} &= \frac{-tprkl}{(q-p)(p-r)(r-k)(k-l)},\\
s_{24n+7} &= \frac{trkl}{(p-r)(r-k)(k-l)}, & s_{24n+8} &= \frac{-tkl}{(r-k)(k-l)},\\
s_{24n+9} &= \frac{tl}{(k-l)}, & s_{24n+10} &= -t,\\
s_{24n+11} &= -q, & s_{24n+12} &= -p,\\
s_{24n+13} &= -r, & s_{24n+14} &= -k,\\
s_{24n+15} &= -l, & s_{24n+16} &= \frac{-tl}{(t-q)},\\
s_{24n+17} &= \frac{-tql}{(t-q)(q-p)}, & s_{24n+18} &= \frac{-tqpl}{(t-q)(q-p)(p-r)},\\
s_{24n+19} &= \frac{-tqprl}{(t-q)(q-p)(p-r)(r-k)}, & s_{24n+20} &= \frac{-tqprkl}{(t-q)(q-p)(p-r)(r-k)(k-l)},\\
s_{24n+21} &= \frac{tprkl}{(q-p)(p-r)(r-k)(k-l)}, & s_{24n+22} &= \frac{-trkl}{(p-r)(r-k)(k-l)},\\
s_{24n+23} &= \frac{tkl}{(r-k)(k-l)}, & s_{24n+24} &= \frac{-tl}{(k-l)}.
\end{aligned}
$$

where $s_{-5}=t,\ s_{-4}=q,\ s_{-3}=p,\ s_{-2}=r,\ s_{-1}=k,\ s_0=l$

Proof. The proof is left to the reader. □

Example 5.8. *Assume* $s_{-5}=2,\ s_{-4}=17,\ s_{-3}=15,\ s_{-2}=14,\ s_{-1}=19,\ s_0=11$. *(See Figure 5.5 for the periodic behavior of 5.5) and* $s_{-5}=-2,\ s_{-4}=17,\ s_{-3}=15,\ s_{-2}=-8,\ s_{-1}=19,\ s_0=1$. *(See Figure 5.6)*

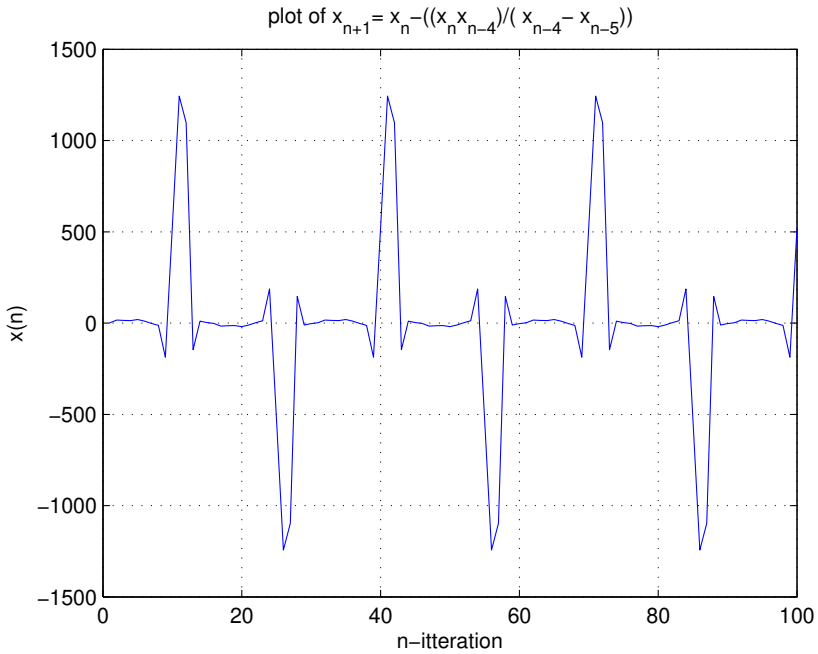

Figure 5.5: Shows periodic solution of 5.5

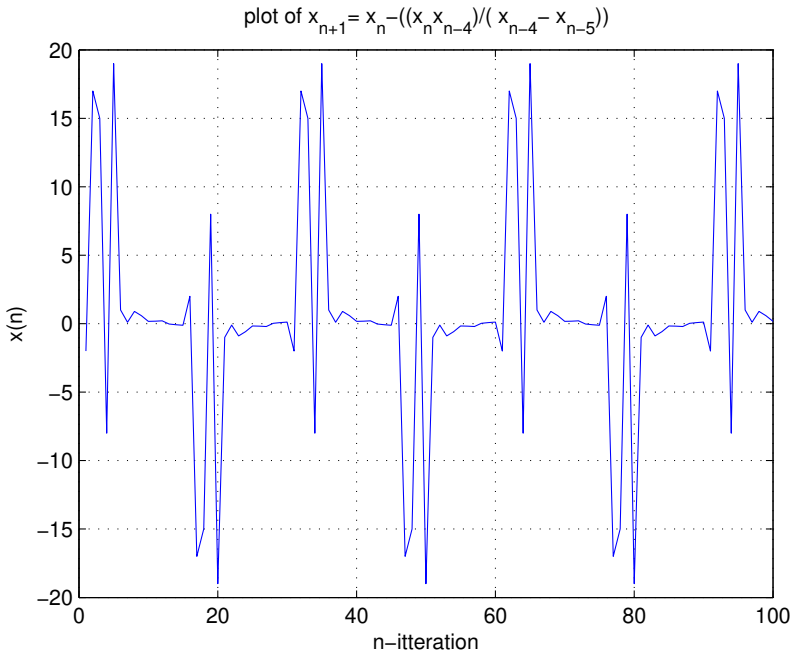

Figure 5.6: Shows periodic solution of 5.5

6. Conclusion

In This paper we studied global stability, boundedness and the solutions of some special cases of equation 1.1. In Section 3 we proved when $\beta(\gamma+3\delta) < (\gamma+\delta)^2(1-\alpha)$, 1.1 has local stability. We proved in the same section that the unique equilibrium of equation 1.1 is globally asymptotically stable if $\gamma(1-\alpha) \neq \beta$. In Section 4 we showed that the solution of equation 1.1 is bounded if $(\alpha + \frac{\beta}{\gamma}) < 1$. In Section 5, we obtained the expression and closed form solution of four special cases of equation 1.1 and gave numerical examples of each of the case, with different initial values.

References

[1] R.E. Mickens, *Difference Equations*, Van Nostrand Reinhold Comp, New York, 1987.
[2] M.C. Mackey, L. Glass, *Oscillation and chaos in physiological control system*, Science, **197**(1977), 287-289.
[3] M.R.S. Kulenovic, G. Ladas, *Dynamics of Second Order Rational Difference Equations with Open Problems and Conjectures*, Chapman & Hall / CRC Press, 2001.
[4] R.J. Beverton, S.J. Holt, *On the Dynamics of Exploited Fish Populations*, Fish Invest., London, 1957.
[5] R. DeVault, G. Dial, V.L., Kocic, G. Ladas, *Global behavior of solutions of* $x_{n+1} = ax_n + f(x_n, x_{n-1})$, Journal of Difference Equations and Applications, **3**(3-4)(1997), 311-330.
[6] E.M. Elsayed, *Qualitative behaviour of difference equation of order two*, Mathematical and Computer Modelling, **50**(2009),1130-1141.
[7] Elabbasy, E. M. Elsayed, *On the difference equation,* $x_{n+1} = \frac{\alpha x_{n-l} + \beta x_{n-k}}{Ax_{n-l} + Bx_{n-k}}$,, Acta Mathematica Vietnamica, **33**(2008), 85-94.
[8] R. Karatas, C. Cinar, D. Simsek, *On positive solutions of the difference equation* $x_{n+1} = \frac{x_{n-5}}{1+x_{n-2}x_{n-5}}$, Int. J. Contemp. Math. Sci., **1**(10)(2006), 495-500.
[9] H. El-Metwally, E. M. Elsayed, *Dynamics of a rational difference equation* $x_{n+1} = \dfrac{ax_{n-l}x_{n-k}}{bx_{n-p} + cx_{n-q}}$, Chinese Annals of Mathematics series B, **15**(5)(2013), 852-857.
[10] E. M. Elsayed, *Behavior and expression of the solutions of some rational difference equations*, Journal of Computational Analysis and Applications, **15**(1)(2013), 73-81.
[11] M. Saleh, M. Aloqeili, *On the difference equation* $y_{n+1} = A + \frac{y_n}{y_{n-k}}$,, Appl. Math. Comput. **176**(1)(2006), 359-363.
[12] E. M. Elsayed, A. Khaliq, *Global attractivity and periodicity behavior of a recursive sequence*, J. Comput. Anal. Appl., **22**(2)(2017), 369-379.
[13] C. Cinar, *On the positive solutions of the difference equation* $x_{n+1} = \dfrac{ax_{n-1}}{1+bx_nx_{n-1}}$, Appl. Math. Comp., **156**(2004) 587-590.
[14] S. E. Das, M.Bayram, *On a System of Rational Difference Equations*, World Applied Sciences Journal, **10**(11)(2010), 1306-1312.
[15] Q. Din, E. M. Elsayed, *Stability analysis of a discrete ecological model*, Computational Ecology and Software, **4**(2)(2014), 89-103.
[16] E. M. Elabbasy, H. El-Metwally, E. M. Elsayed, *On the difference equations* $x_{n+1} = \dfrac{\alpha x_{n-k}}{\beta + \gamma\prod_{i=0}^{k} x_{n-i}}$, J. Conc. Appl. Math., **5**(2) (2007), 101-113.
[17] E. M. Elabbasy, H. El-Metwally, E. M. Elsayed, *Qualitative behavior of higher order difference equation*, Soochow Journal of Mathematics, **33**(4)(2007), 861-873.
[18] E. M. Elabbasy, H. El-Metwally, E. M. Elsayed, *On the Difference Equation* $x_{n+1} = \frac{a_0x_n + a_1x_{n-1} + ... + a_kx_{n-k}}{b_0x_n + b_1x_{n-1} + ... + b_kx_{n-k}}$, Mathematica Bohemica, **133**(2) (2008), 133-147.
[19] R. Agarwal, *Difference equations and inequalities*, Theory, methods and applications, Marcel Dekker Inc., New York, 1992.
[20] H. Chen, H. Wang, *Global attractivity of the difference equation* $x_{n+1} = \dfrac{x_n + \alpha x_{n-1}}{\beta + x_n}$, Appl. Math. Comp., **181**(2006),1431-1438.
[21] E. M. Elsayed, *Dynamics of a Recursive Sequence of Higher Order, Communications on Applied Nonlinear Analysis*, **16**(2)(2009), 37-50.
[22] A. Khaliq, E. M. Elsayed, *The Dynamics and Solution of some Difference Equations*, Journal of Nonlinear Sciences and Applications, **9**(3) (2016), 1052-1063.
[23] A. Khaliq, *On the Solution and Periodic Nature of Higher-order Difference Equation*, Mathematical Sciences Letters, **6**(2)(2017), 177-186.
[24] A. Khaliq, E. M. Elsayed, *Qualitative properties of difference equation of order six*, Mathematics, **4**(2016), 24.

[25] V. L. Kocic, G. Ladas, *Global Behavior of Nonlinear Difference Equations of Higher Order with Applications*, Kluwer Academic Publishers, Dordrecht, 1993.
[26] A. S. Kurbanli, *On the Behavior of Solutions of the System of Rational Difference Equations*, World Applied Sciences Journal, **10**(11) (2010), 1344-1350.
[27] R. Memarbashi, *Sufficient conditions for the exponential stability of nonautonomous difference equations*, Appl. Math. Letter, **21** (2008), 232-235.
[28] A. Neyrameh, H. Neyrameh, M. Ebrahimi, A. Roozi, *Analytic solution diffusivity equation in rational form*, World Applied Sciences Journal, **10**(7)(2010), 764-768.