

Stability Analysis of the Disease Free Equilibrium State of a Mathematical Model of Ebola Fever Disease Epidemic

Abah, R. T

Department of Mathematics, University of Abuja, Abuja, Nigeria. Email: roseabah@yahoo.com

Kuta, F. A. Department of Microbiology, Federal University of Technology, Minna, Nigeria. **Akinwande, N. I** Department of Mathematics & Statistics,

Federal University of Technology, Minna, Nigeria.

Abdulrahaman, S. Department of Mathematics & Statistics, Federal University of Technology, Minna, Nigeria

Enagi, I. A.

Department of Mathematics & Statistics, Federal University of Technology, Minna, Nigeria.

Somma, S. A.

Department of Mathematics & Statistics, Federal University of Technology, Minna, Nigeria. Email: sam.abu@futminna.edu.ng

Abstract – Ebola fever has been a major cause of death in recent times. It has claimed thousands of lives in West Africa since 2014 till date. Very few mathematical models have been developed to study its transmission dynamics. In this paper the stability analysis of the disease free equilibrium state of a mathematical model of Ebola Fever disease epidemic were carried out.

Keywords – Ebola Fever, Quarantine, Equilibrium State, Stability.

I. INTRODUCTION

Ebola fever is an acute viral hemorrhagic fever that is highly contagious, named after a river in the Democratic Republic of the Congo (formerly Zaire) where it was first identified in 1976, (CDC, 2004). It is from a family of RNA (ribonucleic acid) virus called Filoviridae.

Ebola fever is transmitted by physical contact with body fluids, secretions, tissues or semen from infected persons. [1,6]. Nosocomial transmission (transmission from patients within hospital settings) has been typical as patients are often treated by unprepared hospital personnel (barrier nursing techniques must be observed). Individuals exposed to the virus who become infectious do so after a mean incubation period of 1 - 21 days

Ebola fever has been a major cause of death in recent times It has claimed thousands of lives in West Africa since 2014 till date. With reports of outbreaks from nine countries namely, Guinea, Liberia, Sierra Leone, Senegal, Mali, Nigeria,, Spain, United Kingdom and United States as a total of 22,560 cases of infections and 9019 deaths.

Few people have shown considerable interest in the transmission dynamics of Ebola Disease. For example: (Chowell *et al*, 2004), Astacio *et al* (1996), Althaus (2014), Nishiura (2014). Very few mathematical models have been developed to study its transmission dynamics.

II. MODEL FORMULATION

A mathematical model of the dynamics of Ebola Fever incorporating Quarantine and public campaign as controls was formulated. The population is divided into six (6) compartments, namely: Susceptible S(t), Latent L(t), Infectious I(t), Quarantined Q(t) Recovered R(t), and Dead D(t).

The Total population is N(t) = S(t) + L(t) + I(t) + Q(t) + R(t) + D(t) Figure 1.1 is a schematic diagram of Ebola fever transmission and Control model.

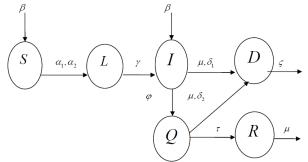


Fig.1.1 Schematic diagram of Ebola Fever transmission and Control model.

Ebola fever models are usually comprised of individuals who have not had effective contact with the virus. These individuals are referred to as Susceptible S(t). When susceptible individuals come into contact with infectious individuals, they get infected but do not become infectious immediately, so they move into a class known as Latent L(t).

After the latency period, these individuals now become infectious, meaning they can now spread Ebola Fever. And so they move into a class known as Infectious I(t).

To prevent the spread of the Ebola Fever, these individuals are isolated into a class known as Quarantine Q(t) for some treatments.

During the treatment period, some of the individuals in Quarantine recover permanently and now move into a class known as Recovered R(t).

Some individuals who die as a result of Ebola Fever move from I(t) and Q(t) into a class known as Dead D(t). This class exists because they are capable of spreading Ebola Fever Virus through unsafe burial.

S(t) are individuals who have not had effective contact with Ebola virus but are prone (susceptible) to Ebola fever through contact with the I(t) and D(t) at the rate



 $\frac{\alpha_1}{N} + \frac{\alpha_2(1-\varsigma)}{N} \text{ where } \alpha_1 \text{ is the effective contact rate}$ between S(t) and $I(t) \cdot \alpha_2(1-\varsigma)$ the effective contact rate between S(t) and D(t). They are generated through a natural birthrate β from S(t), L(t) and R(t) and are reduced by a natural death rate μ .

L(t) are individuals who through contact with the I(t) and D(t), got infected at the rate $\frac{\alpha_1}{N} + \frac{\alpha_2(1-\zeta)}{N}$, where α_1 is the effective contact rate between S(t) and $I(t) \cdot \alpha_2(1-\zeta)$ is the effective contact rate between S(t) and D(t). They are still in incubation period since they have not yet manifested the symptoms of Ebola Fever. After the twenty one (21) days incubation period, they may become infectious if they do not possess strong immunity to fight off the disease, they then join I(t) at a progression rate γ . They are reduced at a death rate μ .

I(t) are the individuals that are infected with Ebola Fever. They are generated by β , where β is the natural birth rate of I(t) and through a progression rate γ from L(t) to I(t). They a reduction reduced due to μ , q and δ_1 , where μ is the natural death rate, q is the rate of quarantine and δ_1 , the disease induced death rate.

D(t) is the compartment for those who are dead through infection, and are generated from both classes I(t) and Q(t) through μ , δ_1 and δ_2 respectively, where μ is the natural death rate of I(t) and $Q(t) \cdot \delta_1$ and δ_2 , the disease induced death rate of I(t) and Q(t) respectively.

Q(t) are the individuals that are generated (quarantined) from I(t) through φ , where φ is the rate of quarantine. They are reduced through τ , μ and δ_2 , where τ is the treatment rate, μ is the natural death rate, and δ_2 is disease induced death rate of Q(t).

R(t) are the individuals that have recovered and have acquired permanent immunity through a treatment rate τ . They suffer a natural death rate μ .

The following assumptions were made to formulate the model:

1. The mixing of people is homogeneous, meaning that all individuals have

equal chance of getting infected if they come in adequate contact with infectious individuals.

2. Those in S(t) get infected through contact with I(t) and D(t).

3. L(t) are infected but not yet infectious, since they get infectious, only when they are symptomatic.

4. The isolation of I(t) to Q(t) cause the spread of Ebola Fever to be very low treatment rate τ .

5. $\delta_2 < \delta_1$ due to the treatment of Q(t) at the rate τ .

6. Offspring of I(t) and Q(t) are not taken into consideration because offspring from these classes die as soon as they are given birth to. Princess Christian Maternity Hospital, Freetown, Sierra Leone. Saturday, September 20th, 2014.

7. If Persons in Q(t) recover, they recover permanently due to the treatment rate τ .

8. The dead class D(t) is not the compartment for the total dead, but for the disease induced death from classes I(t) and Q(t)

The variables are defined as follows:

S(t) Susceptible class at time t

- L(t) Latent class at time t
- I(t) Infectious class at time t
- D(t) Dead from I(t)
- Q(t) Quarantined class at time t
- R(t) Recovered class at time t

The parameters are defined as follows:

- β birth rate
- μ death rate
- δ_1 disease induced death rate of I(t)
- δ_2 disease induced death rate of Q(t)
- α_1 effective contact rate between I(t) and S(t)

 $\alpha_2(1-\zeta)$ effective contact rate between D(t) and S(t)

- γ progression rate from L to I(t)
- φ rate of quarantine
- τ treatment rate
- ξ the rate of effectiveness of public campaign

 ς rate at which the dead is decontaminated and buried

 $(1-\xi)$ proportion that ignored public campaign who can still be infected with EBF.

Model Equations

The schematic diagram is described by a system of ordinary differential equations (1.0) - (1.5):

$$\frac{dS}{dt} = \beta(S+L+R) - \left(\frac{\alpha_1 I}{N} + \frac{\alpha_2(1-\zeta)D}{N}\right)(1-\zeta)S - \mu S$$
(1.0)

$$\frac{dL}{dt} = \left(\frac{\alpha_1 I}{N} + \frac{\alpha_2 (1 - \zeta) D}{N}\right) (1 - \zeta) S - (\gamma + \mu) L \tag{1.1}$$



dt

$$= \gamma L - (\varphi + \mu + \delta_1) I \tag{1.2}$$

$$\frac{dQ}{dt} = \varphi I - (\tau + \mu + \delta_2)Q \tag{1.3}$$

$$\frac{dR}{dt} = \tau Q - \mu R \tag{1.4}$$

$$\frac{dD}{dt} = (\mu + \delta_1)I + (\mu + \delta_2)Q - \zeta D \tag{1.5}$$

Where,

$$N(t) = S(t) + L(t) + I(t) + Q(t) + R(t)$$
(1.6)

So that the total population which is changing at the rate $\frac{dN(t)}{dt}$, is given by

$$\frac{dN(t)}{dt} = \beta N - \mu (S + L + R) \tag{1.7}$$

III. EQUILIBRIUM STATE OF THE MODEL

At equilibrium state, the rate of change of each variable is equal to zero.

i.e.
$$\frac{dS}{dt} = \frac{dL}{dt} = \frac{dI}{dt} = \frac{dQ}{dt} = \frac{dR}{dt} = \frac{dD}{dt} = 0$$
(1.8)

Let (S(t), L(t), I(t), Q(t), R(t), D(t)) = (r, v, w, x, y, z) (1.9)

$$N(t) = n \tag{1.10}$$

Where

$$n = r + v + w + x + y \tag{1.11}$$

Hence, equations (1.0) to (1.7) become

$$\beta(r+v+x) - \left(\frac{\alpha_1 w}{n} + \frac{\alpha_2(1-\zeta)z}{n}\right)(1-\zeta)r - \mu r = 0 \ (1.12)$$

$$\left(\frac{\alpha_1 w}{n} + \frac{\alpha_2 (1-\zeta)z}{n}\right)(1-\zeta)r - (\gamma+\mu)v = 0 \quad (1.13)$$

$$\mathcal{W} - (\varphi + \mu + \delta_1) \mathcal{W} = 0 \tag{1.14}$$

 $\varphi w - (\tau + \mu + \delta_2) x = 0 \tag{1.15}$

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$$\tau x - \mu y = 0 \tag{1.16}$$

$$(\mu + \delta_1)w + (\mu + \delta_2)x - \varsigma z = 0$$
 (1.17)

$$\beta n - \mu (r + v + y) = 0$$

The equilibrium states are then obtained by solving equations (1.13) to (1.18).

(1.18)

3.1 Disease-Free Equilibrium E_0

At the disease free equilibrium state $E_{\rm 0}$, there is absence of infection. Thus the infected classes are zero. The whole population comprise of the susceptible clas.

From (1.17) gives

$$z = \frac{(\mu + \delta_1)w + (\mu + \delta_2)x}{\varsigma}$$
(1.19)

$$v = \frac{(1-\xi)r}{(\gamma+\mu)n} \left(\alpha_1 w + \alpha_2 (1-\zeta)z\right)$$
(1.20)

Substituting (1.19) in (1.20) gives

$$v = \frac{(1-\xi)r}{\varsigma(\gamma+\mu)n} \begin{bmatrix} (\alpha_1\varsigma w + \alpha_2(1-\varsigma)(\mu+\delta_2))w \\ + (\mu+\delta_2)x \end{bmatrix}$$
(1.21)

From (1.15) gives

$$x = \frac{\varphi}{\left(\tau + \mu + \delta_2\right)} w \tag{1.22}$$

Substituting (1.22) in (1.21) gives

$$v = \frac{(1-\xi)r}{\varsigma(\gamma+\mu)n} (\alpha_1 \varsigma + \alpha_2 (1-\varsigma)(\mu+\delta_1)) w$$

+ $(\mu+\delta_2) \frac{\varphi}{(\tau+\mu+\delta_2)} w$ (1.23)

Substituting (1.23) in (1.13) gives

$$\begin{bmatrix} \frac{(1-\xi)r}{\varsigma(\gamma+\mu)n} \left(\alpha_1 \varsigma + \alpha_2 (1-\varsigma) (\mu+\delta_1) + \frac{\varphi(\mu+\delta_2)}{(\tau+\mu+\delta_2)} \right) \\ -(\varphi+\mu+\delta_1-\beta) \\ w = 0 \end{bmatrix} w = 0$$
(1.24)

or

$$\frac{\gamma(1-\xi)r}{(\gamma+\mu)n} \begin{pmatrix} (\alpha_1 \varsigma + \alpha_2(1-\varsigma)(\mu+\delta_2)) \\ + \frac{\varphi(\mu+\delta_2)}{(\tau+\mu+\delta_2)} \end{pmatrix}$$
(1.25)
$$-(\varphi+\mu+\delta_1) = 0$$

Substituting (1.24) in (1.22) gives
$$x = 0$$
 (1.26)

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Substituting (1.26) in (1.16) gives y = 0 (1.27) Substituting (1.24) and (1.26) in (1.19) gives z = 0 (1.28) Substituting (1.24) in (1.14) gives v = 0 (1.29)

Substituting (1.27) and (1.28) in (1.18) gives

 $r = \frac{\beta}{\mu} n$ (1.30) Therefore the disease free equilibrium state is given by: $E_{0} = (r, v, w, x, y, z) = \left(\frac{\beta}{\mu} n, 0, 0, 0, 0\right)$ (1.31)

From (1.0) to (1.6) we obtained the Jacobian matrix given by

$$J = \begin{pmatrix} (\beta - \mu) \\ -\left(\frac{\alpha_1 w}{n} + \frac{\alpha_2 (1 - \varsigma) z}{n}\right)(1 - \xi) & \beta & -\frac{\alpha_1 (1 - \xi) r}{n} & \beta & 0 & -\frac{\alpha_2 (1 - \varsigma) (1 - \xi) r}{n} \\ \left(\frac{\alpha_1 w}{n} + \frac{\alpha_2 (1 - \varsigma) z}{n}\right)(1 - \xi) & -(\gamma + \mu) & \frac{\alpha_1 (1 - \xi) r}{n} & \frac{\alpha_2 (1 - \varsigma) (1 - \xi) r}{n} & 0 & 0 \\ 0 & \gamma & (\phi - \mu - \delta_1) & 0 & 0 & 0 \\ 0 & 0 & \phi & -(\tau - \mu - \delta_2) & 0 & 0 \\ 0 & 0 & 0 & \tau & -\mu & 0 \\ 0 & 0 & 0 & (\mu + \delta_1) & (\mu + \delta_2) & 0 & -\varsigma \end{pmatrix} = 0 \quad (1.32)$$

3.2 Local stability of Disease-Free Equilibrium E_0

We used the Jacobian stability technique of determining the local stability of the system. Consider the Jacobian matrix of (1.32). At disease free equilibrium, E_0 is given by:

$$\begin{pmatrix} (\beta - \mu) & \beta & -\frac{\alpha_{1}(1 - \xi)r}{N} & 0 & \beta & -\frac{\alpha_{2}(1 - \zeta)(1 - \xi)r}{N} \\ 0 & -(\gamma + \mu) & \frac{\alpha_{1}(1 - \xi)r}{N} & 0 & 0 & \frac{\alpha_{2}(1 - \zeta)(1 - \xi)r}{N} \\ - & 0 & \gamma & (\phi - \mu - \delta_{1}) & 0 & 0 & 0 \\ 0 & 0 & \phi & -(\tau - \mu - \delta_{2}) & 0 & 0 \\ 0 & 0 & 0 & \tau & -\mu & 0 \\ 0 & 0 & (\mu + \delta_{1}) & (\mu + \delta_{2}) & 0 & \varsigma \end{pmatrix} = 0$$
(1.33)

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Using reduced row echelon form gives

$\begin{array}{cccccccccccccccccccccccccccccccccccc$	β-μ	β	- <i>c</i>	0	β	-d
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	0	$-\gamma - \mu$			0	h
0 0 0 τ -μ 0	0	γ	j	0		0
	0	0	φ	-1	0	0
$0 0 p r 0 \varsigma$	0	0	0	τ	-μ	0
	0	0	р	r	0	ς

where,

$$c = -\frac{\alpha_{1}(1-\xi)r}{N}, d = -\frac{\alpha_{2}(1-\zeta)(1-\xi)r}{N},$$

$$g = \frac{\alpha_{1}(1-\xi)r}{N}, h = \frac{\alpha_{2}(1-\zeta)(1-\xi)r}{N},$$

$$j = (\varphi - \mu - \delta_{1}), l = -(\tau - \mu - \delta_{2}), p = (\mu + \delta_{1}),$$

$$r = (\mu + \delta_{2})$$

Thus the characteristic equation of the row reduced echelon Jacobian matrix is given by:

$$\begin{bmatrix} \beta - \mu & \beta & -c & 0 & \beta & -d \\ 0 & -\gamma - \mu & g & 0 & 0 & h \\ 0 & 0 & \frac{g\gamma + \gamma j + j\mu}{\gamma + \mu} & 0 & 0 & \frac{\gamma h}{\gamma + \mu} \\ 0 & 0 & 0 & -l & 0 & -\frac{\varphi \gamma h}{g\gamma + \gamma j + j\mu} \\ 0 & 0 & 0 & 0 & -\mu & -\frac{\varphi \gamma h}{l(g\gamma + \gamma j + j\mu)} \\ 0 & 0 & 0 & 0 & 0 & \frac{g\gamma l \varsigma - \gamma h l p - \gamma h r \varphi + \gamma j l \varsigma + j l \mu \varsigma}{l(g\gamma + \gamma j + j\mu)} \end{bmatrix} = 0 \quad (1.34)$$

Thus the eigen values are:

 $\lambda_1 = \beta - \mu < 0 \tag{1.35}$

$$\lambda_2 = -\gamma - \mu < 0 \tag{1.36}$$

$$\lambda_3 = \frac{g\gamma + j\gamma + j\mu}{\gamma + \mu} < 0 \tag{1.37}$$

$$\lambda_4 = -(\tau - \mu - \delta_2) < 0 \tag{1.38}$$

$$\lambda_5 = -\mu < 0 \tag{1.39}$$

$$\lambda_{6} = \frac{g\gamma l\varsigma - \gamma h lp - \phi\gamma hr + \gamma j l\varsigma + j l\mu\varsigma}{l(g\gamma + j\gamma + j\mu)} < 0$$
(1.40)

IV. RESULT

The condition for stability is that $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6$ must be negative.

Therefore λ_1 is negative if $\beta < \mu$ λ_2 is negative since $\lambda_2 = -\gamma - \mu < 0$ λ_4 is negative if $\tau > \mu + \delta_2$ λ_5 is negative since $\lambda_5 = -\mu < 0$ For λ_3 to be negative, $g\gamma + j\gamma + j\mu$ must be negative and so from

$$\lambda_3 = \frac{g\gamma + j\gamma + j\mu}{\gamma + \mu} < 0$$
 we must have

$$g\gamma + j\gamma + j\mu < 0$$
Also for
$$\lambda_6 = \frac{g\gamma l\zeta - \gamma hlp - \varphi\gamma hr + \gamma jl\zeta + jl\mu\zeta}{l(g\gamma + j\gamma + j\mu)} < 0 \text{ to be}$$

negative

$$g \not l \varsigma - \not h l p - \varphi \not h r + \gamma j l \varsigma + j l \mu \varsigma < 0$$

from
$$g \not l \varsigma - \not h l p - \varphi \not h r + \gamma j l \varsigma + j l \mu \varsigma < 0$$

$$l = -(\tau - \mu - \delta_2) < 0$$
 implies that $\tau > \mu + \delta_2$,

V. DISCUSSION OF RESULT

 $\tau > \mu + \delta_2$ implies that the inequality (1.40) will hold and so λ_6 is negative and so we take the condition for the stability of the disease free equilibrium state, to be locally asymptotically stable.

Meaning that τ treatment rate must be greater than both μ natural death rate and δ_2 disease induced death rate,



for the disease free equilibrium state to be locally asymptotically stabile and so the disease will die out.

If otherwise $\tau < \mu + \delta_2$ that is τ treatment rate is less

than both μ the natural death rate and δ_2 disease induced death rate, the disease free equilibrium state will be unstable and this could result in an outbreak of epidemics.

VI. CONCLUSION

The current epidemic of Ebola Fever disease has shown to the world that in absence of a strong public health care delivery system even a rare disease can risk the lives of millions of people. The crux of this epidemic is that a large scale and coordinated international response is the need of the hour to support affected and at-risk nations in intensifying their response activities and strengthening of national capacities.

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REFERENCES

- Akinwande, N.I. "Local Stability Analysis of Equilibrium State of A Mathematical Model of Yellow Fever Epidemics" J. Nig. Math Soc Vol. 14 1995, pp 73-79
- [2] Baize S, Pannetier D, Oestereich L, Rieger T, Koivogui L, Magassouba N, et al. Emergence of Zaire Ebola Virus Diseasein Guinea - Preliminary Report. N Engl J Med. 2014.
- [3] World Health Organization (WHO). Ebola virus disease, West Africa – update on 27 July 2014. Geneva: WHO; 27 Jul 2014.[Accessed 23 Aug 2014]. Available from: http://www.who.int/csr/don/2014_07_27_ebola/en/
- [4] World Health Organization (WHO). WHO Statement on the Meeting of the International Health Regulations Emergency Committee Regarding the 2014 Ebola Outbreak in WestAfrica. Geneva: WHO; 8 Aug 2014. [Accessed 10 Sep 2014].Available from:http://www.who.int/mediacentre/news/statements/2014/ebo la-20140808/en/
- [5] Centre for Disease Control (CDC). Interim Guidance for Managing Patients with Suspected Viral Hemorrhagic Fever in U.S.Hospitals (2005). Available athttp://www.cdc.gov
- [6] Centers for Disease Control (CDC), 2003a.Ebola hemorrhagic fever(http://www.cdc.gov/ncidod/dvrd/spb/mnpages/dispages/eb ola.htm), accessed on August 24, 2003.
- [7] Chowell G, Hengartner NW, Castillo-Chavez C, Fenimore PW, Hyman JM. The basic reproductive number of Ebola and the effects of public health measures: the cases of Congo and Uganda. J Theor Biol. 2004 Jul 7;229(1):119-26.
- [8] World Health Organization (WHO), 2003a.Ebola hemorrhagic fever: disease outbreaks. (http://www.who.int/disease-outbreaknews/disease/A98.4.htm), accessed on October 17, 2003.
- [9] Althaus CL. Estimating the reproduction number of Ebola virus (EBOV) during the 2014 outbreak in West Africa. PLOS Currents Outbreaks. 2014 Sep 2. Edition 1.
- [10] Wikipedia, "Timeline of reported cases and deaths, Ebola virus epidemic in West Africa," 2014.
- [11] Merler S, Ajelli M, Fumanelli L, Gomes MF, Piontti AP, Rossi L, Chao DL, Longini IM Jr, Halloran ME, Vespignani A. Spatiotemporal spread of the 2014 outbreak of Ebola virus disease in Liberia and the effectiveness of non-pharmaceutical interventions: a computational modeling analysis. Lancet Infect Dis. 2015 Jan 6.

- [12] Lekone PE, Finkenstädt BF. Statistical inference in a stochastic epidemic SEIR model with control intervention: Ebola as a case study. Biometrics. 2006 Dec;62(4):1170-7.
- [13] Nishiura H, Chowell G. Early transmission dynamics of Ebola virus disease (EVD), West Africa, March to August 2014. Euro Surveill. 2014;19(36):pii=20894. Available online: http:// www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20894
- [14] Jaime Astacio, DelMar Briere, &Milton Guillen, Mathematical Models to Study the Outbreaks of Ebola. Ebola Virus Disease (Ebola Hemorrhagic Fever), Research, Viral Diseases, Infectious Diseases, Zaire, Africa, 1996.

AUTHOR'S PROFILE

Mrs. Roseline Toyin Abah

Assistant Lecturer, Department of Mathematics, University of Abuja, Abuja, Nigeria. Email: roseabah@yahoo.com

Prof. Ninuola Ifeoluwa Akinwande,

Department of Mathematics and Statistics, Federal University of Technology, Minna, Nigeria.

Dr. Idris Abdullahi Enagi,

Senior Lecturer, Department of Mathematics and Statistics, Federal University of Technology, Minna, Nigeria.

Dr. Farouk A. Kuta,

Senior Lecturer, Department of Microbiology, Federal University of Technology, Minna, Nigeria.

Dr. Sirajo Abdulrahaman,

Senior Lecturer, Department of Mathematics and Statistics, Federal University of Technology, Minna, Nigeria. Email: sirajo.abdul@futminna.edu.ng

Mr. Samuel Abu Somma,

Assistant Lecturer, Department of Mathematics and Statistics, Federal University of Technology, Minna, Nigeria. Email: sam.abu@futminna.edu.ng

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