



Optimal Solution of Mathematical and Statistical Modelling for The Study of Spread, Transmission and Control of Tuberculosis (TB) in Damaturu, Nguru and Potiskum Major Cities of Yobe State

Umar Yusuf Madaki*, Abdul Aziz B. M. Hameed & Ahmed Audu Daya

Department of Mathematics and Statistics, Faculty of Science, Yobe State University, Damaturu, NIGERIA

*Corresponding author email: uymadaki84@gmail.com

Available online 29 December 2021

ABSTRACT:

Background: Tuberculosis (TB) is a contagious bacterial infection caused by mycobacterium tuberculosis. It usually affects the lungs (pulmonary tuberculosis). It can also affect the central nervous system, the lymphatic system, the brain, spine and the kidneys.

Problem: Only peoples who have pulmonary TB infectious. one third of the world population is currently infected with the TB bacillus and new infectious are occurring at the rate of one per second.

Objectives: Tuberculosis was among the top ten causes of death worldwide in 2015 when 10.4 million peoples become ill from TB of which 1.8 million people died from TB. The disease is airborne and so its primary transmitted through the respiratory route.

Results: When people, who are infected with the disease cough, sneeze spit or talks, the propel TB germs in mucus droplets, known as bacilli, into the air.

Conclusion: A previously uninfected person need only a small number of these germs to be infected.

Introduction

Tuberculosis usually attacks the lungs but can also attack other parts of the body lie the kidney, spine, brain, bones, joints etc. the classic symptoms of TB of the lungs are a chronic cough which may result in blood – tinged sputum, fever, nigh, sweats, loss of appetite, weight loss and fatigue. Infection of other organs causes a wide range of symptoms, pneumonia, and lung collapse and enlarge lymph nodes may also occur. Two forms of tuberculosis that become life – threatening are:

1. Military TB, which means the bacteria have spread throughout the lungs and into the bloodstream.
2. Tb meningitis (infection of the covering of the spinal cord and/ or brain by TB bacteria)

Diagnosis relies on radiology (commonly chest X- ray), a tuberculin skin test, blood tests, as well as microscopic examination and microbiological culture of bodily fluids (such as sputum). Prevention relies on screening programmed and vaccination, usually with bacillus Chalmette-Guerin (BCG) vaccine given to infants. The directly observed treatment short (DOTS) course is the internally recommended strategy for the control and cure for TB treatment for tuberculosis uses antibiotics to kill the bacteria.

Problem Statement/Justification

Despite numerous management and control strategies of tuberculosis currently in Yobe state which we will consider the three (3) main towns of the state as case studies (Damaturu, Potiskum and Nguru) specialist hospital and federal medical centers respectively. Tuberculosis continues to cause great health effect worldwide (WHO, 2016). A number of studies over the past two decades have shown that tuberculosis contributes too may illness including cardiovascular disease, cancer and endocrine disease such as tuberculosis Colin and Jane (2015). The mathematical model for tuberculosis transmission will be formulated.

In 2016, tuberculosis (TB) is still a major cause of death and suffering worldwide. Its control is a global public health issue and therefore needs to be conceived and carried out along with the basic principles of equity, human right to health and social protection. As marginalized people are always greatly exposed to health problems and often face difficulties in accessing care, social and economic determinants of ill health must be appropriately addressed together with adequate implementation of the specific interventions available today to combat those diseases that disproportionately affect the poorest.

Jama (2017), Tuberculosis (TB) mortality rate has declined by 37% worldwide since 2000, but the disease remains 1 of the top 10 causes of death, according to the most recent Global TB Report 2017, released by the World Health Organization. Greater political commitment is needed to address the burden of the disease and meet the goal of ending the TB epidemic by 2030.

Tuberculosis was the leading cause of death from a single infectious agent in 2016, ranking above HIV/AIDS. Worldwide, 10.4 million new TB infections were estimated in 2016—10% of which occurred in people with HIV—and about 1.7 million people died of the disease. More than 600 000 new cases of TB with resistance to the most effective first-line drug, rifampicin, were reported, including 490 000 multidrug-resistant TB infections.

Motivation of this Research

The motivations of this research are to:

Model the transmission dynamics of TB for computational mathematical researchers.

To optimize a model for reducing TB cases in Damaturu, Potiskum and Nguru cities without increasing public spending.

Objectives of the Study

Research Data collection in three (3) case studies (Damaturu, Potiskum and Nguru) specialist hospital and federal medical centers respectively.

This work is aim at formulating mathematical model for tuberculosis dynamics incorporating treatment. The following objectives are to be achieved

1. To formulated and analysis a mathematical model on the tuberculosis dynamic for treatment of the disease.
2. To determine the stability analyses of the equilibrium points among people of Yobe State.
3. To obtain the basic reproduction number of determine equilibrium point and study on the strategies of addressing TB on how to reduce it among of TB infected individuals.
4. To explain which mathematical and statistical models are widely used to examine, explain and predict the dynamics of infectious disease transmission and models of specific diseases of global importance have played important role in developing public health strategies for control and prevention of infectious disease.

Significance of the Study

The significances of the study include the following:

- a) The model will help to understand the dynamic and treatment TB.
- b) The study will also act as a base for further research on the tuberculosis dynamic and treatment and other related diseases.
- c) The study intends to contribute on strategies of addressing TB and how to reduce it among of TB infected individual.

Scope and Limitation

This work centered on formulating a mathematical model for tuberculosis dynamics and its analysis by obtaining the equilibrium solution state and stability of such state. This study limited to only tuberculosis disease, however, it can be extended to other epidemic with little modification.

Literature Review

A review on earlier works on the tuberculosis provides the prospective of the proposed study. Many mathematical models have been developed to address tuberculosis transmission dynamic and control.

Enagi (2015), considers a deterministic compartmental model of tuberculosis control strategy adopted by national tuberculosis and leprosy control program. He established the disease free and the endemic equilibrium state and carried out the stability analysis of the disease free and the endemic equilibrium state.

He carried out numerical simulations of the model using maple mathematical software to have an insight into the dynamics of the model. He found out that the disease free equilibrium state is stable. The numerical simulation showed that it will be very difficult to complete eradicate tuberculosis from Nigeria using this method adopted by national tuberculosis and leprosy control program.

Mugisha et al. (2015), formulated mathematical models for the dynamics of tuberculosis in density population required to minimize and therefore eradicate tuberculosis. Both numerical and qualitative analyses was done and the effect of various in the area size and recruitment rates was investigated. His work suggested that characteristic area could as well be looked at as environmental stressor that can lead to tuberculosis.

Methodology

Model Formulation

Susceptible Infectious Recovered (SIR) Model:

SIR model is considered as a basic epidemic model. Usually, diseases caused by a virus such as influenza and measles, typhoid are of SIR type. Kermack and Mckendrick proposed this model in 1927. Many epidemiological diseases could be described by SIR model. Consider the flow of SIR model with constant vaccination strategy.

The basic idea behind the SIR model (Susceptible - Infectious - Recovered) of communicable disease outbreaks is that there are three groups (also called compartments) of individuals:

- S: those who are healthy but susceptible to the disease (i.e., at risk of being contaminated). At the start of the pandemic, S is the entire population since no one is immune to the virus.
- I: the infectious (and thus, infected) people
- R: individuals who were contaminated but who have either recovered or died. They are not infectious anymore.
- These groups evolve over time as the virus progresses in the population:
- S decreases when individuals are contaminated and move to the infectious group I
- As people recover or die, they go from the infected group I to the recovered group R

To model the dynamics of the outbreak we need three differential equations to describe the rates of change in each group, parameterized by:

- β , the infection rate, which controls the transition between S and I
- γ , the removal or recovery rate, which controls the transition between I and R

Formally, this gives:

$$\frac{dS}{dt} = -\frac{\beta IS}{N} \dots \dots \dots (1)$$

$$\frac{dI}{dt} = \frac{\beta IS}{N} - \gamma I \dots \dots \dots (2)$$

$$\frac{dR}{dt} = \gamma I \dots \dots \dots (3)$$

for of susceptible individuals (S) decreases with the number of newly infected individuals, where new infected cases are the result of the infection rate (β) multiplied by the number of susceptible individuals (S) who had a contact with infectious individuals (I).

The second equation (Eq. 2) states that the number of infectious individuals (I) increases with the newly infected individuals (βIS), minus the previously infected people who recovered (i.e., γI which is the removal rate γ multiplied by the infectious individuals I).

Finally, the last equation (Eq. 3) states that the recovered group (R) increases with the number of individuals who were infectious and who either recovered or died (γI). An epidemic develops as follows:

- . Before the start of the disease outbreak, S equals the entire population as no one has anti-bodies.
- . At the beginning of the outbreak, as soon as the first individual is infected, S decreases by 1 and I increases by 1 as well.
- . This first infectious individual contaminates (before recovering or dying) other individuals who were susceptible.
- . The dynamic continues, with recently contaminated individuals who in turn infect other susceptible people before they recover.

Visually, we have:

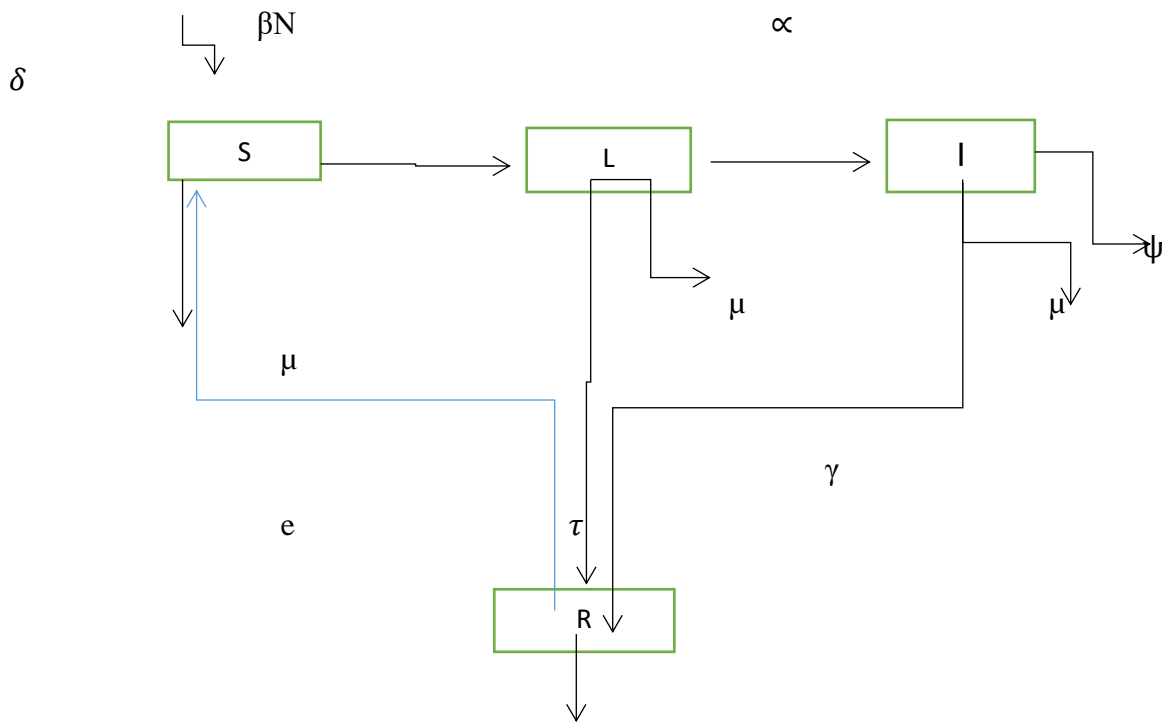


SIR model

Before fitting the SIR model to the data, the first step is to express these differential equations as an R function, with respect to time t.

In this study, we formulated a deterministic, compartment model to investigate the transmission dynamics between infected and susceptible individual in a population. The progression of tuberculosis within the total population can be simplified using four different equations representing four different groups of people; the susceptible (S) the intent (L) the infective (I). Thus, the diagram for the deterministic model as follows:

The schematics diagram of the model is giving below;



Where μ , β , γ and π are considered as positive parameters. Furthermore, we assumed that vaccination is 100% effective and the natural death rates μ and birth rate π are not same, this cause N to be not constant. A susceptible will move to I-compartment when encounters an infected individual, an infected individual move to R compartment after recovery. Vaccinated individuals are also coming into R-compartment. Now SIR model can be formulated as

$$\frac{ds}{dt} = (1 - p)\pi N - \beta \frac{SI}{N} - \mu S \dots \dots \dots (4)$$

SIR Model with constant vaccination where

- S = Susceptible individuals
- I = Infected
- R = Recovered people with permanent immunity
- μ = Natural death rate
- β = average contact rate
- γ = Recover rate
- π = Birth rate
- P = new born vaccinated each year ($0 < P < 1$)

$$\frac{dl}{dt} = \beta \frac{SI}{N} - (\gamma + \mu)I \dots \dots \dots (5)$$

$$\frac{dR}{dt} = P\pi N + \gamma I - \mu R \dots \dots \dots (6)$$

Solution of a Vaccination Based SIR Epidemic Model by Homotopy Analysis Method
 We know $N = S + I + R$

Adding (1) to (3), we have

$$\frac{dN}{dt} = (\pi - \mu)N \dots\dots\dots(7)$$

We have a case of varying total population.

Dimensionless Transformation

We want to convert the varying total population into a constant total population, for this we have to choose new variables

$$s = \frac{S}{N} \quad i = \frac{I}{N} \quad r = \frac{R}{N}$$

Now total population is constant i.e., $0 = dt \, dN$, from equation (4), we have birth rate equal to death rate $\pi = \mu$
 Putting respective values in (1), (2) & (3), new system is

$$\frac{ds}{dt} = (1 - p)\pi - \beta si - \pi s \dots\dots\dots(8)$$

$$\frac{di}{dt} = \beta si - (\gamma + \pi)i \dots\dots\dots(9)$$

$$\frac{dr}{dt} = P\pi + \gamma i - \pi r \dots\dots\dots(10)$$

Qualitative Analysis

We will analyze system in two categories
 1. Infection free equilibrium ($i = 0$)
 2. Endemic equilibrium ($i \neq 0$)

Subsystems in the closed set form are

$$\Gamma = \{(s, i) \in R^+ | 0 \leq s \leq 1\}$$

To find fixed points, from eq (8) & (9)

$$0 = (1 - p)\pi - \beta si - \pi s \dots\dots\dots(11)$$

$$0 = \beta si - (\gamma + \pi)i \dots\dots\dots(12)$$

Existance of Disease Free - Equilibrium (DFE) State

The disease free equilibrium is a steady state solution of the tuberculosis dynamic model with all infected population equal to zero. The stability of the disease free equilibrium state is extremely important because it help us to investigate the long-term behavior of the system. It can determine whether or the bacterial are capable of invading a population.

Case I. Infection free equilibrium

When disease dies out naturally then from eq (9)

$$(\beta s - \gamma - \pi) \neq 0 \quad \& \quad i = 0$$

From eq (8); $s = (1 - p)$

The solution comes on an infection free equilibrium E_0 asymptotically

$$E_0 = (1 - P, 0)$$

Reproduction number and basic reproduction numbers are:

$$R_0 = \frac{\beta}{\gamma + \pi} \quad \& \quad R_v = \frac{\beta(1 - p)}{\gamma + \pi} \quad \text{Respectively}$$

This is a threshold which determines the stability of equilibrium.

Case II. Endemic Equilibrium

An unstable disease free equilibrium i.e., $R_v > 1$ give rise to endemic equilibrium E_u . Again from eq (9);

$$\begin{aligned} 0 &= (\beta s - \gamma - \pi)i \\ (\beta s - \gamma - \pi) &= 0 \quad \& \quad i = 0 \\ \Rightarrow s &= \frac{(1 - p)}{R_v} \end{aligned}$$

From of eq (11);

$$i = \frac{\pi}{\beta} \left(\frac{(1 - P)}{s} - 1 \right) \quad \text{or} \quad i = \frac{\pi}{\beta} (R_v - 1)$$

So, we have endemic equilibrium of the form

$$E_u = \left(\frac{(1 - p)}{R_v}, \frac{\pi}{\beta} (R_v - 1) \right)$$

Stability Analysis

The infection free equilibrium E_0 is locally stable if $R_v < 1$ and endemic equilibrium E_u is unstable. Conversely for $R_v > 1$, endemic equilibrium E_u is stable and infection free equilibrium E_0 is unstable. In both cases local stability of equilibrium give rise to Global stability in the particular domain of s and i . An examination of local stability of the model's equilibria reveals that there is a critical vaccination proportion

$$P_c = 1 - \frac{1}{R_0} \Rightarrow P_c = \frac{\beta - \gamma - \pi}{\beta}$$

P_c governs the system as follow

1. For relatively large vaccination level i.e., $P_c > P$ infection free equilibrium is locally stable with the coordinates

$$s = 1 - p \quad \& \quad i = 0$$

While endemic equilibrium is unstable.

2. For relatively weak vaccination i.e., $P_c < P$, endemic equilibrium is locally stable with the coordinates

$$s = \frac{(1 - p)}{Rv} \quad \& \quad \frac{\pi}{\beta}(Rv - 1)$$

The Jacobian matrix at Endemic equilibrium E_u .

$$J = \begin{pmatrix} -\pi Rv & -(\gamma + \pi) \\ \pi(Rv - 1) & 0 \end{pmatrix}$$

$$trcJ = -\pi Rv$$

$$\det J = \pi Rv(\gamma + \pi) - \pi(\gamma + \pi)$$

As we know

$$trcJ \pm \frac{\sqrt{(trcJ)^2 - 4(\det J)}}{2}$$

On putting values, we have

$$\lambda_{1,2} = \frac{-\pi Rv \pm \sqrt{(-\pi Rv)^2 - 4[\pi Rv(\gamma + \pi) - \pi(\gamma + \pi)]}}{2}$$

For small values of π & γ we neglect the last term under the square root sign

$$\lambda_{1,2} \approx -\frac{\pi}{2} Rv \pm \frac{1}{2} \sqrt{\pi^2 Rv^2 - 4Rv\pi(\gamma + \pi)}$$

For asymptotically stable, value under square root will be negative i.e.

$$Rv \leq \frac{4(\gamma + \pi)}{\pi}$$

The endemic equilibrium E_u is locally asymptotically stable if

$$1 < Rv \leq \frac{4(\gamma + \pi)}{\pi}$$

We have complex eigenvalues with negative real part. So E_u can be treated as a spiral sink. This can be explained as initially susceptible are increasing and we have few infected. Then infection starts spreading and susceptible start to decrease. Disease spread more rapidly than increment in susceptible. As a result we are left with too small number of individuals who are susceptible to disease, the outbreaks ends and susceptible begins to increase again.

Before going on with sir model it is important to understand how epidemics set up in a population for disease conferring long lasting immunity infection. E.g measles. The number of susceptible (s) decrease with time. Before the outbreak of a first measles case the population of susceptible (s) is 100% in the population because everyone is susceptible, the proportion of expose (E), infected (I) and recovered (R) is zero (0), when epidemic start to spread susceptible decrease while immune and infection increase until everyone get immunized.

The potential of infected person in a population depend on the basic in a population depends on the basic reproduction number (R) is define as the average number of parson directly infected by an infection disease during

his/her entire infection period when he/she enters a totally susceptible persons. The development and the size of infection are determined by (R) that relies on.

1. HAM is different than all perturbation and non-perturbation techniques because of the following facts.
2. 1. Large or small parameters are of no significance in HAM
3. Solution of a Vaccination Based SIR Epidemic Model by Homogony Analysis Method
4. 2. Convergence of solution can be ensured in a very simple way.
5. 3. We are free to choose base function.
6. The attack rate (risk of transmission per contact)
7. The number of potentially infected contact that is the average person in a population has per unit time.
8. The duration has per the infecting period.

If at any time (R) gets smaller than one that is the disease eventually disappears from the population because on average each infected person cannot ensure transmission of the infection agent to one susceptible. This result in a new waves of infected been lesser amplitude then the proceeding one and finally to disease elimination on other hand it (R₀=1) the disease remain endemic as one infection agent to one infections agent to one susceptible on the average. Lastly if (R₀=1) en epidemic build up. This effort has been established by Kermak and Mc Kendrick and expands body the introduction of infections individual into a community of susceptible does not automatically give rise to an epidemic outbreak.

THE FIRST RESULT AND DISCUSSION

In this section we are used the model formulated in chapter three to compute the result at various time and the result obtained can be discuss to enable used depict the epidemic or otherwise from equation .

Table 1.0 the initial values and parameters are presented of percentage (%)

Case	S_0	i_0	r_0	β	γ	π	P
1	0.0	0	0	0.9	0.04	0.2	1.98
2	0.1	2	0	0.8	0.04	0.2	1.84
3	0.2	8	0	0.7	0.04	0.2	1.75
4	0.3	14	0	0.9	0.04	0.2	1.56
5	0.4	18	0	0.8	0.04	0.2	1.45
6	0.5	22	0	0.9	0.04	0.2	1.30
7	0.6	26	0	0.6	0.04	0.2	1.20

THE EXPECTED RESULT IN DAMATURU LOCAL GOVERNMENT

The models will help to understand the dynamic and treatment TB. The study will also act as a base for further research on the tuberculosis dynamic and treatment and other related diseases. The model will serve as a benchmark in mathematical modelling on epidemic diseases that the disease-free equilibrium is asymptotically stable at threshold parameter less than unity and unstable at threshold parameter greater than unity. The existence of the unique endemic equilibrium is also determined under certain conditions. Numerical simulations will be carried out to confirm the analytic results and explore the possible behavior of the formulated model.

Table 1.1 to shows the Susceptible, Infected and Infectious, Recovered people, Vaccinated, Birth rate and Time Period

1	Time/period	0	1	2	3	4	5	6
2	Susceptible	0	100	200	300	400	500	600
3	Infection	100	92	84	72	54	46	34
4	Natural Death rate	0.04	0.04	0.04	0.04	0.04	0.04	0.04
5	Average contact rate	0.9	0.8	0.7	0.9	0.8	0.9	0.6
6	Birth rate	0.2	0.2	0.2	0.2	0.2	0.2	0.2
7	New born vaccinated each year	0.0	0.2	0.3	0.4	0.5	0.6	0.7
8	Recovered people with permanent immunity	0	125	180	200	210	220	230
9	Recover rate	0	130	140	148	158	169	178

$$R_v = \frac{\beta(1-P)}{\gamma + \pi}$$

$$= \frac{0.9(1-1.98)}{0.04 + 0.2} = \frac{-0.98}{0.24}$$

$$= -3.675$$

$R_v < 1, E_0$

For $t=0$

$$\frac{ds}{dt} = (1-P)\pi - \beta s_0 i_0 - \pi s_0 - \frac{\beta(1-P)}{\gamma + \pi}$$

$$= (1-2.70) 0.2 - 0.9(0.0) - 0.2(0.0) - (-3.675)$$

$$= -0.735 + 3.675$$

$$= 2.94 \Rightarrow s = 2.94 = s_1 > 1$$

$$s(t) = 34 - 2.94t + 1.30 \times 10^{-1}t^2 - 1.45 \times 10^{-2}t^3 + 1.56 \times 10^{-3}t^4 - 1.78 \times 10^{-4}t^5 - 1.84 \times 10^{-5}t^6 - 1.98 \times 10^{-6}t^7$$

When $t=0$

$$s(0) = 34 - 2.94t + 1.30 \times 10^{-1}t^2 - 1.45 \times 10^{-2}t^3 + 1.56 \times 10^{-3}t^4 - 1.78 \times 10^{-4}t^5 - 1.84 \times 10^{-5}t^6 - 1.98 \times 10^{-6}t^7$$

$$s(0) = 34 \qquad \qquad \qquad \text{First year}$$

When $t=1$

$$s(1) = 46 - 2.94t + 1.30 \times 10^{-1}(1)^2 - 1.45 \times 10^{-2}(1)^3 + 1.56 \times 10^{-3}t^4 - 1.78 \times 10^{-4}(1)^5 - 1.84 \times 10^{-5}(1)^6 - 1.98 \times 10^{-6}(1)^7$$

$$s(1) = 46 - 2.94 + 2.53 + 1.94 + 1.24 - 0.75 - 0.0016 + 0.617$$

$$s(1) = 46 - 10.015$$

$$s(1) = 35.98$$

$$\approx 36 \qquad \qquad \qquad \text{Approximation} \qquad \qquad \qquad \text{Second year}$$

When $t=2$

$$s(2) = 150 - 2.94t + 1.30 \times 10^{-1}(2)^2 - 1.45 \times 10^{-2}(2)^3 + 1.56 \times 10^{-3}(2)^4 - 1.78 \times 10^{-4}(2)^5 - 1.84 \times 10^{-5}(2)^6 - 1.98 \times 10^{-6}(2)^7$$

$$s(2) = 150 - 5.94 + 10.12 + 15.52 + 19.84 - 24 - 0.1024 + 78.976$$

$$s(2) = 150 - 106.294$$

$$s(2) = 43.76$$

$$\approx 44 \qquad \qquad \qquad \text{Approximation} \qquad \qquad \qquad \text{Third year}$$

$i(t) = 0$

$$r(t) = 2.94t + 1.30 \times 10^{-1}t^2 - 1.45 \times 10^{-2}t^3 + 1.56 \times 10^{-3}t^4 + 1.78 \times 10^{-4}t^5 - 1.84 \times 10^{-5}t^6 - 1.98 \times 10^{-6}t^7$$

$$\frac{dr}{dt_{t=0}} = P\pi + -\gamma i_0 - \pi r_0$$

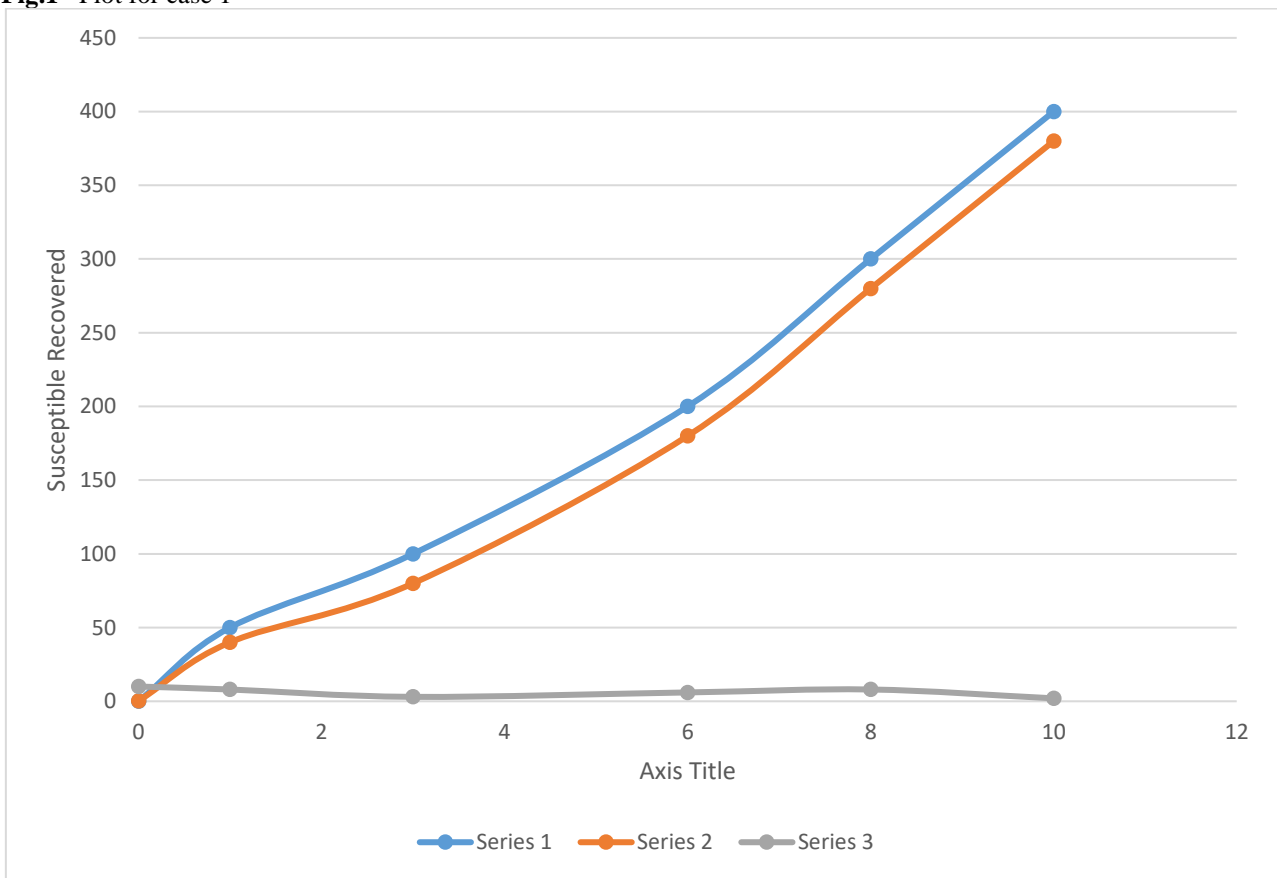
$$\Rightarrow r = 2.94t = r1$$

For

s2, s3, s41.....i2, i3, i43 and r2, r3, r4.....

Time Population Fraction
 Infected Recovered
 Susceptible Recovered

Fig.1 Plot for case 1



From **table**, based on **figure1**, there is a decline in the number of non-infected person in the society of Damatudu Local Government Yobe State host, although, the number of non-infected decline steady and latter reduces sharply showing a threat to the community.

Time Population Fraction

Susceptible
 Infected
 Recovered




Fig.2 Plot for case 1

Table 1 Infections People of Damaturu

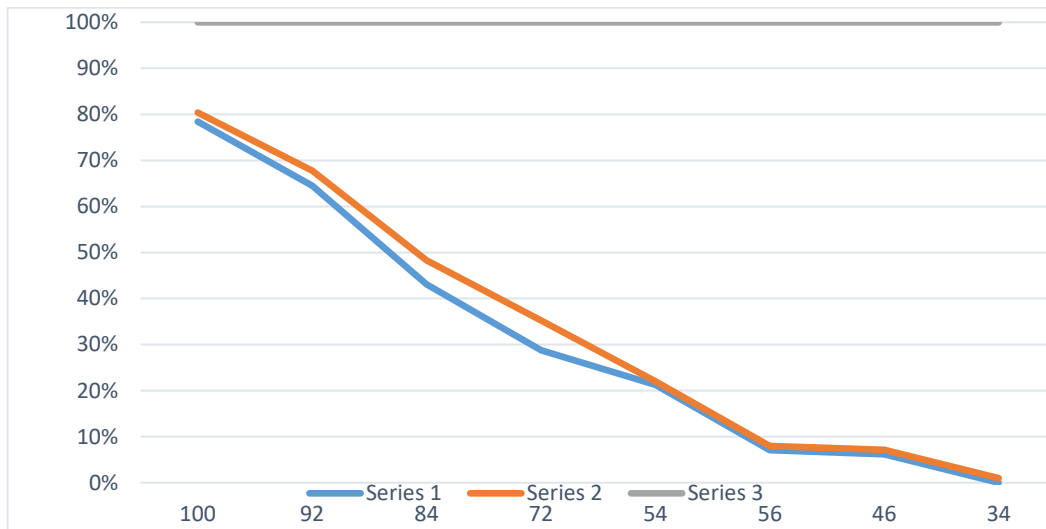


Figure 1 Infections

However, the **table 2** and the above **figure 1** depicts that an epidemics has set up and the level of the epidemic is totally of the final stayed affecting on average of 286. People in just four days, that is it infected positive is steady from the onset and letter it exponentially increase causing every large damage to the society. Also the graph below depicted the non-infected people against the time in the same society of host at that period in time.

Solution of a Vaccination Based SIR Epidemic Model by Homotopic Analysis Method

We know $N = S + I + R$

Adding (4) to (6), we have

THE SECOND RESULT AND DISCUSSION

In this section we are used the model formulated in chapter three to compute the result at various time and the result obtained can be discuss to enable used depict the epidemic or otherwise from equation .

Table 1.0 the initial values and parameters are presented of percentage (%)

Case	S_0	i_0	r_0	β	γ	π	P
1	0.0	0	0	0.8	0.04	0.2	0.84
2	0.1	2	0	0.9	0.04	0.2	0.78
3	0.2	4	0	0.7	0.04	0.2	0.65
4	0.3	8	0	0.9	0.04	0.2	0.56
5	0.4	12	0	0.8	0.04	0.2	0.45
6	0.5	14	0	0.9	0.04	0.2	0.36
7	0.6	20	0	0.9	0.04	0.2	0.20

THE EXPECTED RESULT IN NGURU LOCAL GOVERNMENT

The models will help to understand the dynamic and treatment TB. The study will also act as a base for further research on the tuberculosis dynamic and treatment and other related diseases. The model will serve as a benchmark in mathematical modelling on epidemic diseases that the disease-free equilibrium is asymptotically stable at threshold parameter less than unity and unstable at threshold parameter greater than unity. The existence of the unique endemic equilibrium is also determined under certain conditions. Numerical simulations will be carried out to confirm the analytic results and explore the possible behavior of the formulated model.

Table 1.1 to shows the Susceptible, Infected and Infectious, Recovered people, Vaccinated, Birth rate and Time Period

1	Time/period	0	1	2	3	4	5	6
2	Susceptible	0	50	150	200	250	300	350
3	Infection	80	76	68	54	44	36	24
4	Natural Death rate	0.04	0.04	0.04	0.04	0.04	0.04	0.04
5	Average contact rate	0.9	0.8	0.9	0.9	0.8	0.9	0.6
6	Birth rate	0.2	0.2	0.2	0.2	0.2	0.2	0.2
7	New born vaccinated each year	0.0	0.2	0.3	0.4	0.5	0.6	0.7
8	Recovered people with permanent immunity	0	50	75	95	150	175	200
9	Recover rate	0	40	70	90	120	140	175

$$R_v = \frac{\beta(1-P)}{\gamma + \pi}$$

$$= \frac{0.9(1-0.84)}{0.04 + 0.2} = \frac{0.16}{0.24}$$

$$= 2.5$$

$R_v < 1, E_0$

For $t=0$

$$\frac{ds}{dt} = (1-P)\pi - \beta s_0 i_0 - \pi s_0 - \frac{\beta(1-P)}{\gamma + \pi}$$

$$= (1-0.84)0.2 - 0.9(0.0) - 0.2(0.0) - (-2.5)$$

$$= -0.16 + 2.5$$

$$= 2.66 \Rightarrow s = 2.66 = s_1 > 1$$

$$s(t) = 26 - 2.5t + 0.84 \times 10^{-1}t^2 - 0.74 \times 10^{-2}t^3 + 0.65 \times 10^{-3}t^4 - 0.45 \times 10^{-4}t^5 - 0.30 \times 10^{-5}t^6 - 0.20 \times 10^{-6}t^7$$

When $t=0$

$$s(0) = 26 - 2.5t + 0.84 \times 10^{-1}t^2 - 0.74 \times 10^{-2}t^3 + 0.65 \times 10^{-3}t^4 - 0.45 \times 10^{-4}t^5 - 0.30 \times 10^{-5}t^6 - 0.20 \times 10^{-6}t^7$$

$$s(0) = 26$$

First year

When $t=1$

$$s(1) = 26 - 2.5(1) + 0.84 \times 10^{-1}(1)^2 - 0.74 \times 10^{-2}(1)^3 + 0.65 \times 10^{-3}(1)^4 - 0.45 \times 10^{-4}(1)^5 - 0.30 \times 10^{-5}(1)^6 - 0.20 \times 10^{-6}(1)^7$$

$$s(1) = 26 - 2.5 + 1.253 - 0.011 - 1.233 + 2.776 + 5.184 + 5.456$$

$$s(1) = 26 + 6.877$$

$$s(1) = 32.877$$

$$\approx 33$$

Approximation

Second year

When $t=2$

$$s(2) = 65 - 2.5(2) + 0.84 \times 10^{-1}(2)^2 - 0.74 \times 10^{-2}(2)^3 + 0.65 \times 10^{-3}(2)^4 - 0.45 \times 10^{-4}(2)^5 - 0.30 \times 10^{-5}(2)^6 - 0.20 \times 10^{-6}(2)^7$$

$$s(2) = 65 - 5 + 1.28 - 0.011 - 1.23 - 2.77 - 4.18 - 6.54$$

$$s(2) = 65 - 18.451$$

$$s(2) = 46.545$$

$$\approx 46$$

Approximation

Third year

$$i(t) = 0$$

$$r(t) = 2.5t + 0.84 \times 10^{-1}t^2 - 0.74 \times 10^{-2}t^3 + 0.65 \times 10^{-3}t^4 - 0.45 \times 10^{-4}t^5 - 0.30 \times 10^{-5}t^6 - 0.20 \times 10^{-6}t^7$$

$$\frac{dr}{dt_{t=0}} = P\pi + -\gamma i_0 - \pi r_0$$

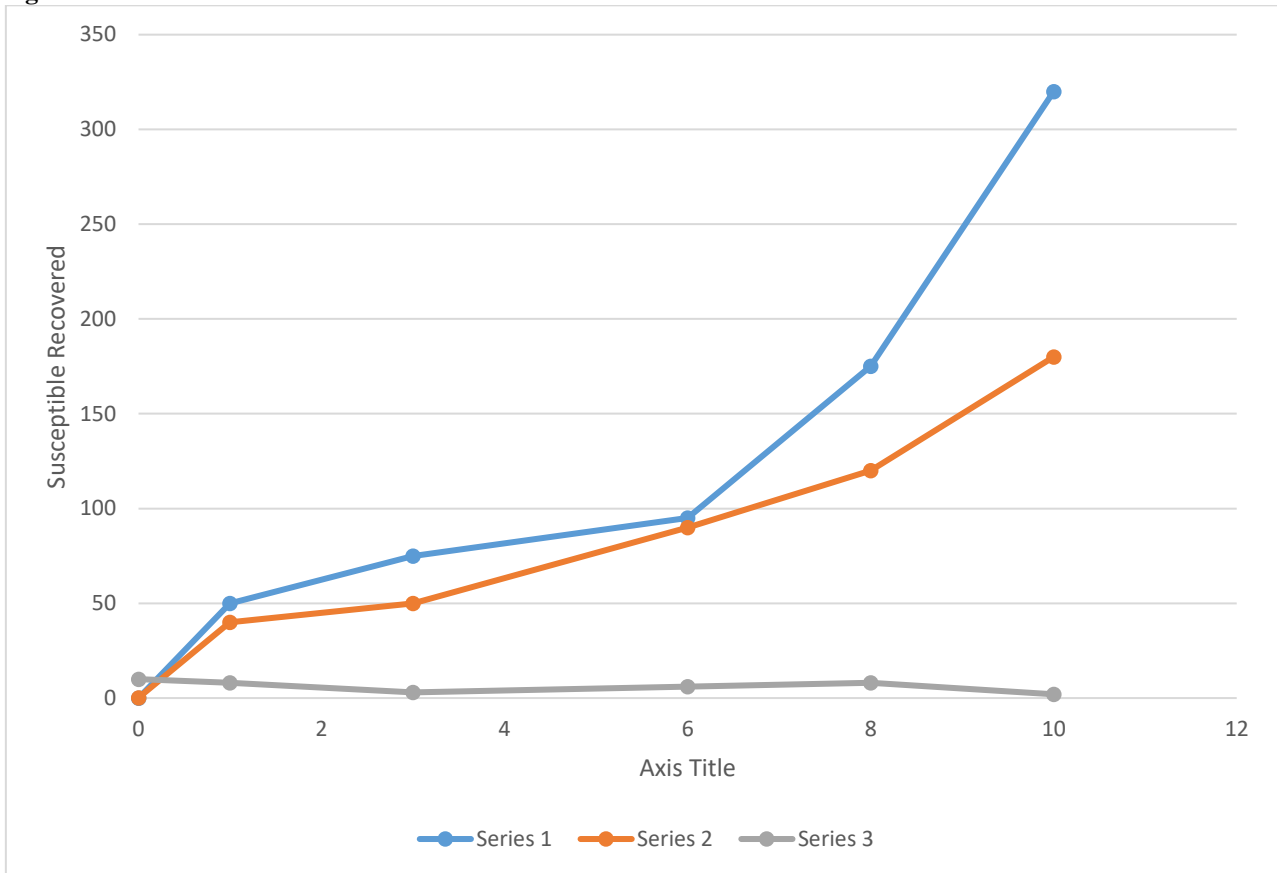
$$\Rightarrow r = 2.5t = r1$$

For

s₂, s₃, s₄.....i₂, i₃, i₄ and r₂, r₃, r₄.....

Time Population Fraction
 Infected Recovered
 Susceptible Recovered

Fig.1 Plot for case 1



From **table**, based on **figure1**, there is a decline in the number of non-infected person in the society of Ngru Local Government host, although, the number of non-infected decline steady and latter reduces sharply showing a threat to the community.

Time Population Fraction

Susceptible
 Infected
 Recovered




Fig.2 Plot for case 2

Table 2 Infections People of NGRU

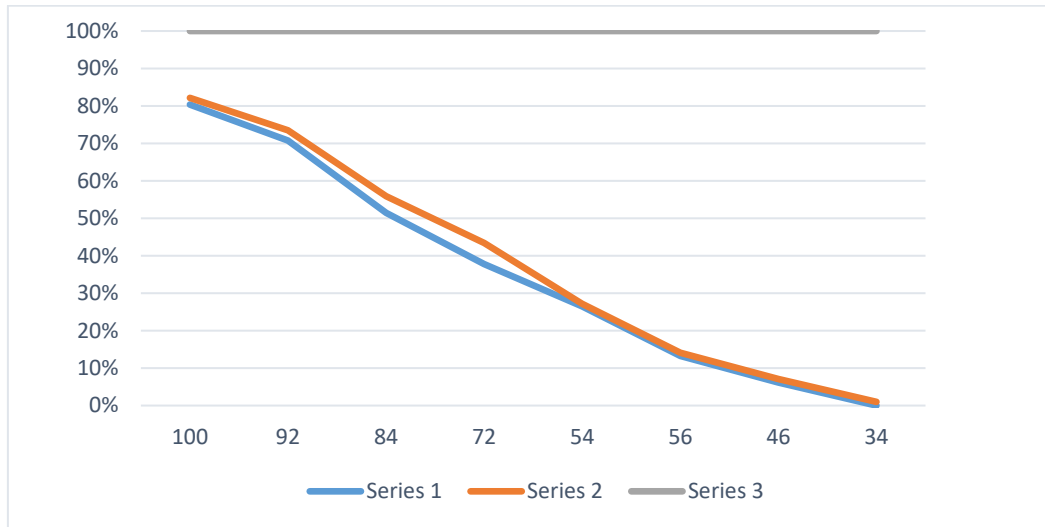


Figure 2 Infections

However, the **table 2** and the above **figure 1** depicts that an epidemics has set up and the level of the epidemic is totally of the final stayed affecting on average of 286. People in just four days, that is it infected positive is steady from the onset and letter it exponentially increase causing every large damage to the society. Also the graph below depicted the non-infected people against the time in the same society of host at that period in time.

Solution of a Vaccination Based SIR Epidemic Model by Homotopic Analysis Method

We know $N = S + I + R$

Adding (4) to (6), we have

THE THIRD RESULT AND DISCUSSION

In this section we are used the model formulated in chapter three to compute the result at various time and the result obtained can be discuss to enable used depict the epidemic or otherwise from equation .

Table 1.0 the initial values and parameters are presented of percentage (%)

Case	S_0	i_0	r_0	β	γ	π	P
1	0.0	0	0	0.9	0.04	0.2	0.90
2	0.1	2	0	0.9	0.04	0.2	0.88
3	0.2	4	0	0.9	0.04	0.2	0.78
4	0.3	8	0	0.9	0.04	0.2	0.67
5	0.4	12	0	0.9	0.04	0.2	1.56
6	0.5	14	0	0.9	0.04	0.2	1.45
7	0.6	20	0	0.9	0.04	0.2	1.30

THE EXPECTED RESULT IN POTISKUM LOCAL GOVERNMENT

The models will help to understand the dynamic and treatment TB. The study will also act as a base for further research on the tuberculosis dynamic and treatment and other related diseases. The model will serve as a benchmark in mathematical modelling on epidemic diseases that the disease-free equilibrium is asymptotically stable at threshold parameter less than unity and unstable at threshold parameter greater than unity. The existence of the unique endemic

equilibrium is also determined under certain conditions. Numerical simulations will be carried out to confirm the analytic results and explore the possible behavior of the formulated model.

Table 1.1 to shows the Susceptible, Infected and Infectious, Recovered people, Vaccinated, Birth rate and Time Period

1	Time/period	0	1	2	3	4	5	6
2	Susceptible	0	100	150	200	250	300	350
3	Infection	120	86	78	64	54	46	37
4	Natural Death rate	0.04	0.04	0.04	0.04	0.04	0.04	0.04
5	Average contact rate	0.9	0.8	0.9	0.9	0.8	0.9	0.6
6	Birth rate	0.2	0.2	0.2	0.2	0.2	0.2	0.2
7	New born vaccinated each year	0.0	0.2	0.3	0.4	0.5	0.6	0.7
8	Recovered people with permanent immunity	0	150	175	195	250	275	300
9	Recover rate	0	100	170	190	220	240	275

$$R_v = \frac{\beta(1-P)}{\gamma + \pi}$$

$$= \frac{0.9(1-0.90)}{0.04 + 0.2} = \frac{0.19}{0.24}$$

= -2.375

$R_v < 1, E_0$

For $t=0$

$$\frac{ds}{dt} = (1-P)\pi - \beta s_0 i_0 - \pi s_0 \frac{-\beta(1-P)}{\gamma + \pi}$$

$$= (1-0.90)0.2 - 0.9(0.0) - 0.2(0.0) - (-2.375)$$

$$= -0.82 + 2.375$$

$$= 3.195 \Rightarrow s = 3.2 = s_1 > 1$$

$$s(t) = 37 - 3.2t + 0.90 \times 10^{-1}t^2 - 0.88 \times 10^{-2}t^3 + 0.78 \times 10^{-3}t^4 - 0.67 \times 10^{-4}t^5 - 1.56 \times 10^{-5}t^6 - 1.45 \times 10^{-6}t^7$$

When $t=0$

$$s(0) = 37 - 3.2t + 0.90 \times 10^{-1}t^2 - 0.88 \times 10^{-2}t^3 + 0.78 \times 10^{-3}t^4 - 0.67 \times 10^{-4}t^5 - 1.56 \times 10^{-5}t^6 - 1.45 \times 10^{-6}t^7$$

$$s(0) = 37$$

First year

When $t=1$

$$s(1) = 37 - 3.2(1) + 0.90 \times 10^{-1}(1)^2 - 0.88 \times 10^{-2}(1)^3 + 0.78 \times 10^{-3}(1)^4 - 0.67 \times 10^{-4}(1)^5 - 1.56 \times 10^{-5}(1)^6 - 1.45 \times 10^{-6}(1)^7$$

$$s(1) = 37 - 3.2 + 1.45 - 0.39 - 0.879 - 2.178 - 2.129 - 9.94$$

$$s(1) = 37 - 17.266$$

$$s(1) = 19.734$$

$$\approx 18$$

Approximation

Second year

When $t=2$

$$s(1) = 37 - 3.2(2) + 0.90 \times 10^{-1}(2)^2 - 0.88 \times 10^{-2}(2)^3 + 0.78 \times 10^{-3}(2)^4 - 0.67 \times 10^{-4}(2)^5 - 1.56 \times 10^{-5}(2)^6 - 1.45 \times 10^{-6}(2)^7$$

$$s(2) = 36 - 6.4 + 5.8 - 3.12 - 14.06 - 69.39 - 135.68 - 1272.32$$

$$s(2) = 1500 - 1492.3$$

$$s(2) = 7.67$$

$$\approx 8$$

Approximation

Third year

$$i(t) = 0$$

$$r(t) = 3.2t + 0.90 \times 10^{-1}t^2 - 0.88 \times 10^{-2}t^3 + 0.78 \times 10^{-3}t^4 - 0.67 \times 10^{-4}t^5 - 1.56 \times 10^{-5}t^6 - 1.45 \times 10^{-6}t^7$$

$$\frac{dr}{dt_{t=0}} = P\pi + -\gamma i_0 - \pi r_0$$

$$\Rightarrow r = 3.2t = r1$$

For

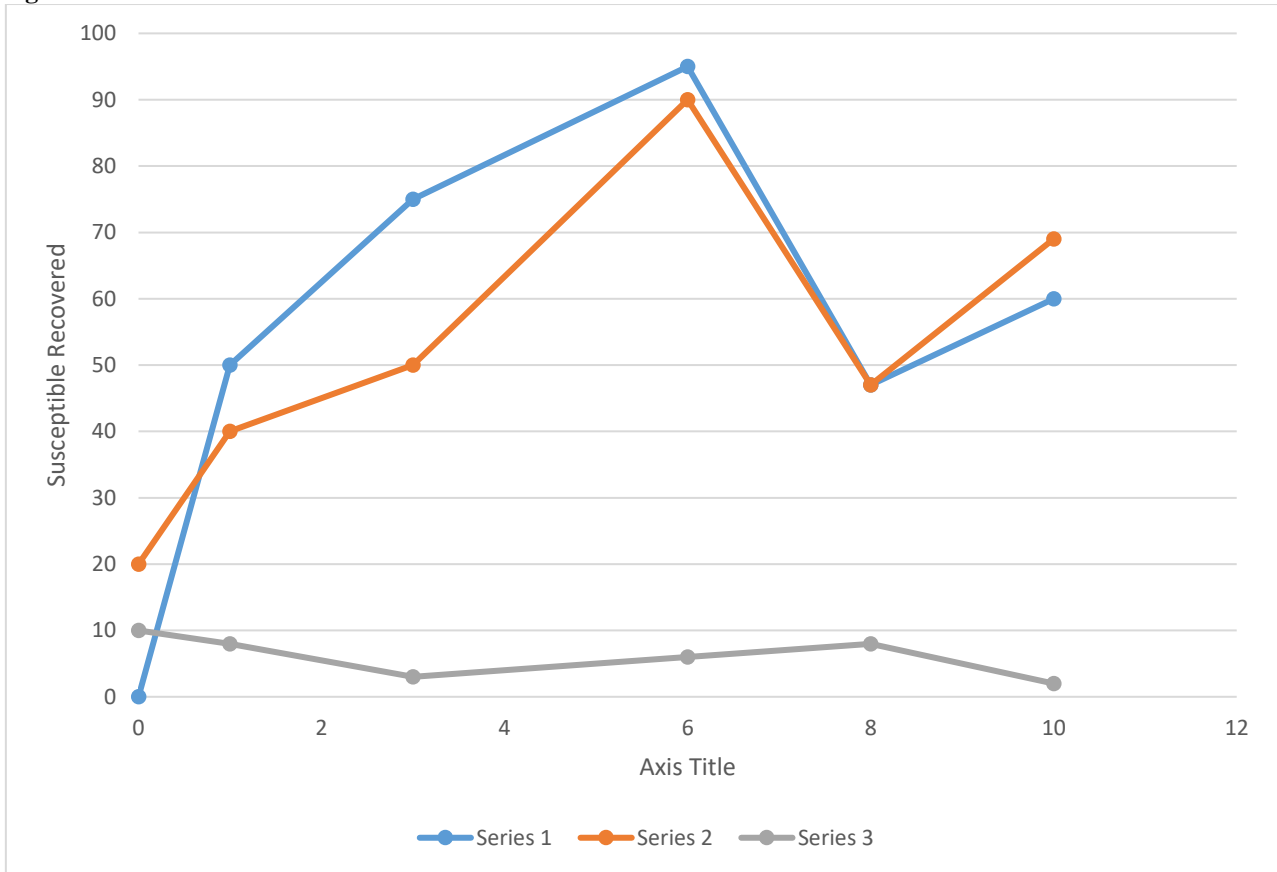
s₂, s₃, s₄.....i₂, i₃, i₄ and r₂, r₃, r₄.....

Time Population Fraction

Infected Recovered

Susceptible Recovered

Fig.1 Plot for case 1



From **table**, based on **figure1**, there is a decline in the number of non-infected person in the society of Potiskum Local Government host, although, the number of non-infected decline steady and latter reduces sharply showing a threat to the community.

Time Population Fraction

Susceptible

Infected

Recovered



Fig.2 Plot for case 1

Table 3 Infections People of NGRU



Figure 3 Infections

However, the **table 2** and the above **figure 1** depicts that an epidemics has set up and the level of the epidemic is totally of the final stayed affecting on average of 186. People in just four days, that is it infected positive is steady from the onset and letter it exponentially increase causing every large damage to the society. Also the graph below depicted the non-infected people against the time in the same society of host at that period in time.

CONCLUSION

Based on the findings so far, SIR generates series which converge speedily after some iteration. The epidemic has set up affecting the positive fraction of the community in Damaturu, Nguru, Potiskum host with as average of 20% infected person daily. The epidemic level was slow in the first place but later it increases exponentially indicating a sign of danger to the said community of host.

RECOMMENDATION

Sequel to the finding obtained in this study it is obvious that the results obtained have depicted the number of people likely to be infected over a period of time and make a reasonable for best of how many people to be infected in a certain time to enable a proper decision and supply in case of an outbreak Tuberculosis (TB) is a contagious bacterial like this in a given society of host with known population number.

Although the study has not make it to our consumption the death that occurred since inception of the epidemic the number of recovered people and the inclining and declining, in the trend of their immune system during Tuberculosis when on treatment. Further study could be conducted by any interested candidate to make clarification of these not known parameter enlisted above by the study.

A study of this nature need data from the host societies which were normally monitored and checked by health agencies difficulties is often encountered while trying to collect the data for the paper. In conclusion I would like to say that since there is an outbreak in the said society of host a special care unit be build by the government in at least every unit of local government so that we can get proper health care.

REFERENCES

- [1] Enagi, A. I (2013). A “deterministic compartmental model of tuberculosis control strategy adopted by the national tuberculosis and leprosy control program in Nigeria.” *Specific journal of science and technology*.
- [2] Asamoah Cynthia Sekubia and Ebenezer Kofi Mensah. *Analysis of Typhoid Fever Surveillance Data*, Cape Coast Metropolis, 2016. *Acta Scientific Medical Sciences*. Volume 3 Issue 2 February 2019. PP.
- [3] *Annual Report on Typhoid Fever*, World Health Organization (WHO), 2017.
- [4]. Transmission dynamics Adebisi A. F, Peter O. J , Ayoola T. A, Oguntolu F. A, Ishola C. Y. Approximate Solution of Typhoid Fever Model by Variational Iteration Method. *ATBU, Journal of Science, Technology & Education (JOSTE)*; Vol. 6 (3), September 2018, PP.254-265.
- [5] Moffat Cuchi Nyaboe, Mose Ong’au Fred and Johana Kibet Sigey. Modeling the Endemic Equilibrium of Typhoid Fever. *The SIJ Transactions on Computer Science Engineering & its Applications (CSEA)*, Vol. 5, No. 5, November 2017. PP.104-111.
- [6] World Health Organization (WHO), https://en.wikipedia.org/wiki/Paratyphoid_fever. 2017.
- [7] Gerald Gwinji. Guidelines for the management of typhoid fever. World Health Organization (WHO). July 2011. PP.1-39.
- [8] Agresti, A. (2002). *Categorical data analysis*: New York: John Wiley and Sons.
- [9] David W. Hosmer, Jr., Stanley Lemeshow, Rodney X. Sturdivant, *Applied logistic regression (3rd edition.)* March 2013. New York: Wiley ISBN: 978-0-470-58247-3.
- [10] Menard, S. (1995). *Applied logistic regression analysis (Sage University Paper Series on Quantitative Applications in the Social Sciences,07–106)*. Thousand Oaks, CA: Sage.
- [11] Press, S. J., & Wilson, S. (1978). Choosing between logistic regression and discriminant analysis. *Journal of the American Statistical Association*, 73, 699–705.
- [12] Barron, I.H. (2014) can stress cause disease? Revisiting tuberculosis research oh Thomas holmes. *Ann intern med*: volume 124 ISSN 1949 – 1961, PP 673 – 680.
- [13] Vimalkumar V K, C.R. Anand Moses, Padmanaban S. Prevalence and Risk Factors of Nephropathy in Type 2 Diabetic Patients *International Journal of Collaborative Research on Internal Medicine and Public Health*, 2011 - Vol. 3 No. 8.
- [14] Maurya V.N., Singh V.V. and Yusuf Madaki Umar, Statistical analysis on the rate of kidney (renal) failure, *American Journal of Applied Mathematics and Statistics*, Special Issue: Application and Future Scope of Fundamental Mathematical and Computational Sciences in Engineering and Technology, Science & Education Publishing, USA, Vol. 2, No. 6A, pp. 6-12, 2014.
- [15] Menard, S. (2000). Coefficients of determination for multiple logistic regression analysis. *The American Statistician*, 54(1), 17–24.
- [16] Steady Mushayabasa, Claver P. Bhunu and Ngoni A. Mhlanga. Modeling the Transmission Dynamics of Typhoid in Malaria Endemic Settings. *Applications and Applied Mathematics: An International Journal*, Vol.9, Issue 1(June 2014), pp.121-140.