International Journal on Recent Trends in Life Science and Mathematics (IJLSM)
ISSN: 2349-7955 Volume: 9 Issue: 3
***Article Received**: 05 July 2022 **Revised**: 25 August 2022 **Accepted**: 30 August 2022 **Publication**: 09 September 2022*

Mathematical Model for the Public Campaign on Typhoid Fever Transmission and Control

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Abstract
In this paper, we examined the dynamics of the typhoid fever model; in order to validate our model formulations and governing equations, we first established the disease-free equilibrium (DFE) of the state as well as the endemic equilibrium (EE). This paper further performed the local stability of the disease-free equilibrium. The basic reproductive number, R_0, is determined using the next generation matrix approach. finally, the paper demonstrated that, when $R_0 < 1$ is less than one, the disease-free equilibrium is considered to be globally asymptotically stable, and it guarantees that the disease will eventually be eradicated.

Keyword: mathematical model, public campaign, typhoid fever, transmission, and control

1.0 Introduction

Typhoid fever is a highly contagious, human-only illness largely caused by the salmonella typhi bacterium [17]. The occurrence of typhoid fever is a serious global danger, with over 20 million infections and about a quarter million deaths per year. The illness is especially frequent in impoverished and undeveloped countries with poor sanitation, particularly portions of south Asia [8].

Nearly 80% of cases and fatalities occur in Asia, with the remaining happening predominantly in Africa and Latin America [16, 18]. The prevalence of typhoid fever has declined globally during the past two decades [26]. Multi-year outbreaks of antibiotic bacteria, on the other hand, threaten to reverse this progress [14]. Salmonella enteric serovar Typhi is the causative agent of enteric fever (S.Typhi). With early diagnosis and medical knowledge, the severity of illnesses can be reduced, consequently lowering the death rate. Nevertheless, traditional procedures like the Widal test, although sensitivity and specificity are not very high [21], are commonly employed to identify illnesses. In addition, additional variables of time, labour, and expense limit the availability of the testing, particularly in underdeveloped nations.

Salmonella enterica, Serovar Typhi (S.Typhi) is a human-only infection spread by faeces that contaminate food and water [24]. Among these variables are inadequate sanitation, interaction with carriers, living near stagnant bodies of water, meteorological circumstances, and socioeconomic level settings (such as literacy rates, food and water consumption habits) [7,9]. While changes to water and sanitation resulted to the eradication of typhoid in most industrialised nations throughout the 20th century, the worldwide burden of typhoid fever is estimated to be between 13.5 to 26.9 million illnesses and 190,000 to 216,000 yearly fatalities.

Traditionally, the incubation period lasts between one and two weeks, and the disease lasts between four and six weeks. The symptoms include anorexia, stomach discomfort, headache, pains and weakness, a high temperature that commonly reaches 104 degrees Fahrenheit, intestinal haemorrhage, cough, diarrhoea, and constipation. People with typhoid fever often develop a persistent temperature between 103 and 104 degrees Fahrenheit (39 and 40 degrees Celsius) [11]. In many poor nations, it may be challenging to fulfil the public health goals that can help prevent and limit the spread of typhoid fever illness by providing clean drinking water, better sanitation, and enough medical treatment. Health education is essential for raising public awareness and influencing behaviour change [27].

Early in the 20th century, public health professionals argued the appropriate techniques for measuring the efficiency of typhoid vaccines and if immunisation constituted a diversion from sanitation and hygiene improvements. These remain contemporary policy issues for ministries of health and other health partners who may be considering programmatic anti-typhoid initiatives, such as significant improvement to income distributions, sanitation, water supplies, and hand

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washing with soap (after defecation and before preparing food at home or for sale on the street), as well as identification and management of carriage.

In much of Africa, including Nigeria, the Widal agglutination test is the most used diagnostic method for typhoid fever due to its inexpensive cost, convenience of use, and minimum training and equipment [20]. Nevertheless, a number of medical experts have regularly expressed worry over the 'high prevalence' of typhoid fever in Nigeria and other African healthcare institutions [10, 19]. In addition, the World Health Organization has advised immunisation of high-risk people in endemic regions and the use of vaccinations to suppress epidemics [29].

Typhoid fever remains a significant problem in Nigeria due to factors such as increased urbanisation, inadequate storage facilities for potable water, regional movement of large numbers of immigrant workers, inadequate facilities for processing human waste, overburdened healthcare delivery, and overuse of antibiotics that contribute to the expansion and spread of S. Typhi resistant to antibiotics [2,25]. Due to the lack of a planned epidemiological monitoring network, it is impossible to estimate the exact prevalence of typhoid fever in Nigeria.

Several mathematical models [1,12,13, 16] have been constructed to represent the illness's dynamics, but none of them include public health education campaigns, immunisation, and therapy as disease management methods.

A mathematical model is a depiction of a system that employs mathematical concepts and terminology; mathematical modelling is the process of creating a mathematical model. Most mathematical models are utilised by physicists, engineers, statisticians, operation research analysts, and economists. A model may be used to describe a system, assess the influence of its many components, and anticipate its behaviour.

Mathematical models have become critical management tools for epidemiologists, illuminating the mechanisms underlying observed dynamics and quantitatively forecasting the efficiency of various control strategies. The mathematical epidemiology literature and evolution are exhaustively documented ([3–5]).

[15] developed a system for estimating the reduction of typhoid cases in poor countries without raising government investment. According to their results, the magnitude of herd protection effects has a substantial effect on the overall number of cases averted and the amount of money saved on public rehabilitation costs.

In Nigeria, typhoid is frequent in general. Regarding typhoid prevention, a great deal of information exists. Numerous people continue to be unaware of typhoid therapy and its impact on the incidence rate. This experiment reveals the influence of awareness creation (treatment and education), which is particularly essential considering that public awareness currently plays a key role in the spread of typhoid.

2.0 Methodology

2.1 Formulation of Model

For the purpose study the total human population was categorized into 5 compartments: vaccinated (V), susceptible (S), infected (A), treated (T), and recovered (R) Individual.

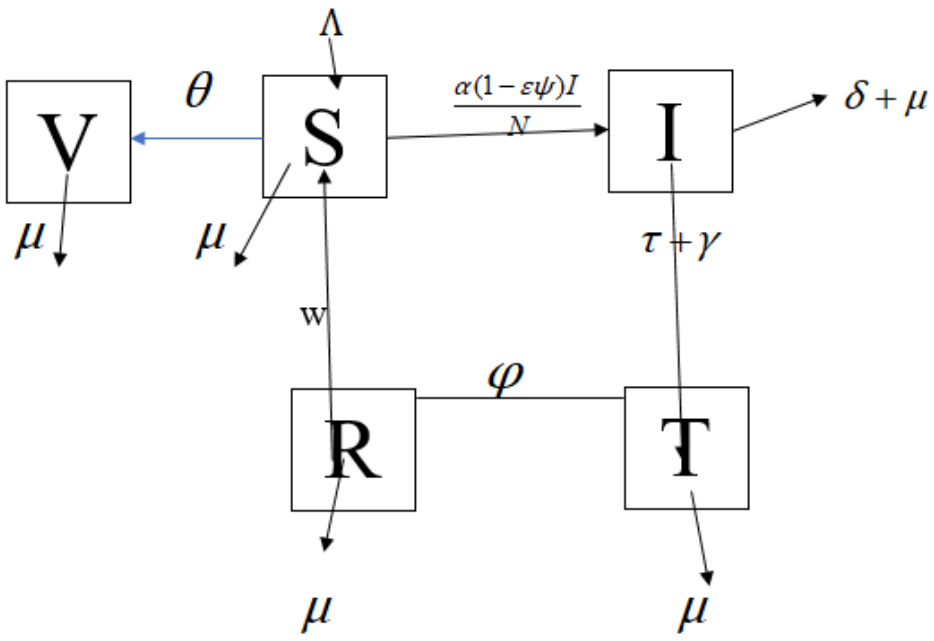

Figure 1 Schematic Diagram of Typhoid Fever Transmission Dynamics

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Using systems of ordinary differential equations (1) through (5), the model's equivalent mathematical equations can be determined.

$$\frac{dV}{dt} = \theta S - \mu \mathrm{V} \quad (1)$$

$$\frac{dS}{dt} = \Lambda - \theta S - \frac{\alpha(1-\varepsilon\psi)SI}{N} - \mu S + \omega R \quad (2)$$

$$\frac{dI}{dt} = \frac{\alpha(1-\varepsilon\psi)S\,\mathrm{I}}{N} - (\delta + \tau + \gamma + \mu)\mathrm{I} \quad (3)$$

$$\frac{dT}{dt} = \tau I - (\mu + \varphi)T \quad (4)$$

$$\frac{dR}{dt} = (\varphi + \gamma)T - (\mu + \omega)R \quad (5)$$

$V(t)$ the vaccinated compartment is generated when the susceptible individuals receive vaccine at the rate θ and decreases due to natural mortality at the rate μ. The susceptible individuals $S(t)$ is generated as a result of constant recruitment at the rate Λ into the population either by birth or through immigration. It decreases when some of them receives vaccine at the rate θ and are no longer prone to the disease, it further reduces when there is an effective contact between the susceptible individual at the rate α and an infected individual, the susceptible population, also there is a reduction in the susceptible population due to natural death μ and finally there is an addition to the susceptible population due to waning of drugs at the rate ω.

The infected class $I(t)$ is generated when there is an effective contact between the susceptible individual at the rate α and an infected individual, it reduces due to treatment, natural recovery, natural mortality and disease induced death at the rate τ, γ, μ and δ respectively. The treated compartment $T(t)$ are generated when drugs are being administered to the infected individuals at the rate τ, it decreases due recovery of the patient measured in (φ) and in addition decreases due to natural mortality measured in (μ). Finally, the recovered class is generated from the recovery of the treated individuals at the rate φ and decreases due to waning of drugs and natural mortality at the rate ω and μ respectively. ψ is the level of compliance to the public awareness programs as control parameter and ε is the effectiveness of the public awareness programs.

2.2 Existence of Equilibrium

At equilibrium, there is no external force acting within the population. Hence, the rate of change of each variable is null.

given;

$$\frac{dV}{dt} = \frac{dS}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = \frac{dR}{dt} = 0 \quad (6)$$

It is then assumed that;

$$(\mathrm{V, S, I, T, R}) = (V^*, S^*, \mathrm{I}^*, T^*, R^*) \quad (7)$$

Equations (1) to (5) becomes,

$$\theta S^* - \mu V^* = 0 \quad (8)$$

$$\Lambda - \theta S^* - \frac{\alpha(1-\varepsilon\psi)S^*I^*}{N} - \mu S^* + \omega R^* = 0 \quad (9)$$

$$\frac{\alpha(1-\varepsilon\psi)S^*\,\mathrm{I}^*}{N} - A_1\,\mathrm{I}^* = 0 \quad (10)$$

$$\tau I^* - A_2 T^* = 0 \quad (11)$$

$$A_4 T^* - A_3 R^* = 0 \quad (12)$$

$$\delta + \tau + \gamma + \mu = A_1 \quad (13)$$

$$\mu + \varphi = A_2 \quad (14)$$

$$\mu + \omega = A_3 \quad (15)$$

$$\varphi + \gamma = A_4 \quad (16)$$

From (8)

$$V^* = \frac{\theta S^*}{\mu} \quad (17)$$

From (9)

$$(\theta + \frac{\alpha(1-\varepsilon\psi)I^*}{N} + \mu)S^* = \Lambda + \omega R^* \quad (20)$$

$$(\frac{\theta N + \alpha(1-\varepsilon\psi)I^* + \mu N}{N})S^* = \Lambda + \omega R^* \quad (21)$$

$$S^* = \frac{N(\Lambda + \omega R^*)}{\theta N + \alpha(1-\varepsilon\psi)I^* + \mu N} \quad (22)$$

Also, from (10)

$$\left(\frac{\alpha(1-\varepsilon\psi)S^*}{N} - A_1\right)\mathrm{I}^* = 0 \quad (23)$$

(23) results in two equilibria. that is,

$$I^* = 0 \quad (24)$$

or

$$\left(\frac{\alpha(1-\varepsilon\psi)S^*}{N} - A_1\right) = 0 \quad (25)$$

2.3 Disease Free Equilibrium (DFE) E^o

At this point, the entire population reduces to the susceptible and the vacinated individuals. There is a total absence of disease.

Lemma 3.1: At each point there is disease-free equilibrium (DFE)

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$$E^o = (V^o, S^o, I^o, T^o, R^o) = \left(\frac{\theta\Lambda}{\mu(\theta+\mu)}, \frac{\Lambda}{\theta+\mu}, 0, 0, 0\right) \quad (26)$$

Proof:

From (24),

$$I^o = 0 \quad (27)$$

Substitute equation (27) into equation (11)

$$T^o = 0 \quad (28)$$

Substitute equation (28) into equation (12)

$$R^o = 0 \quad (29)$$

Substitute equation (27) and (29) into equation (22)

$$S^o = \frac{\Lambda}{\theta+\mu} \quad (30)$$

Substituting equation (30) into (17)

$$V^o = \frac{\theta\Lambda}{\mu(\theta+\mu)} \quad (31)$$

The lemma is proved

2.4 Endemic Equilibrium (EE) E^{**}

At endemic equilibrium, the disease is not totally wiped out from the population, but it persists.

Lemma 2: There exist an endemic equilibrium of the model at:

$$E^{**} = \left(V^{**}, S^{**}, I^{**}, T^{**}, R^{**}\right) =$$

$$\left(\begin{array}{c} \frac{A_1\theta N}{\alpha(1-\varepsilon\psi)\mu}, \frac{A_1 N}{\alpha(1-\varepsilon\psi)}, \frac{\left((\theta+\mu)S^{**}+\Lambda\right)(NA_2A_3)}{A_2A_3\alpha(1-\varepsilon\psi)S^{**}+N\omega A_4\tau}, \\ \frac{\tau\left((\theta+\mu)A_1 N+\Lambda(1-\varepsilon\psi)\right)(NA_2A_3)}{A_2(1-\varepsilon\psi)\left(A_1A_2A_3\alpha N+N\omega A_4\tau\right)}, \\ \frac{A_4\tau\left((\theta+\mu)A_1 N+\Lambda(1-\varepsilon\psi)\right)(NA_2A_3)}{A_3A_2(1-\varepsilon\psi)\left(A_1A_2A_3\alpha N+N\omega A_4\tau\right)} \end{array}\right) \quad (44)$$

Proof:

From (25)

$$S^{**} = \frac{A_1 N}{\alpha(1-\varepsilon\psi)} \quad (33)$$

substituting (33) in (17)

$$V^{**} = \frac{A_1\theta N}{\alpha(1-\varepsilon\psi)\mu} \quad (34)$$

From (12)

$$R^{**} = \frac{\tau I^{**}}{A_2} \quad (35)$$

Substituting (35) into (9)

$$I^{**} = \frac{\left((\theta+\mu)S^{**}+\Lambda\right)(NA_2A_3)}{A_2A_3\alpha(1-\varepsilon\psi)S^{**}+N\omega A_4\tau} \quad (36)$$

Substituting (33) into (36)

$$I^{**} = \frac{\left((\theta+\mu)A_1 N+\Lambda(1-\varepsilon\psi)\right)(NA_2A_3)}{(1-\varepsilon\psi)\left(A_1A_2A_3\alpha N+N\omega A_4\tau\right)} \quad (37)$$

Substituting (37) into (11)

$$T^{**} = \frac{\tau\left((\theta+\mu)A_1 N+\Lambda(1-\varepsilon\psi)\right)(NA_2A_3)}{A_2(1-\varepsilon\psi)\left(A_1A_2A_3\alpha N+N\omega A_4\tau\right)} \quad (38)$$

Substituting (38) into (12)

$$R^{**} = \frac{A_4\tau\left((\theta+\mu)A_1 N+\Lambda(1-\varepsilon\psi)\right)(NA_2A_3)}{A_3A_2(1-\varepsilon\psi)\left(A_1A_2A_3\alpha N+N\omega A_4\tau\right)} \quad (39)$$

The theorem is proved

2.4.1 Number of Effective Reproduction, R_C

Using the next generation operator approach outlined by (Diekmann & Heesterbeek, 2000) and then analysed by (Van de Driesches & Watmough, 2002). The effective reproduction number, R_C which is the spectral radius of the matrix of the following generation, was determined.

Using the next generation operator strategy provided by Diekmann and Heesterbeek (2000) and assessed by (Van de Driesches & Watmough, 2002). The effective reproduction number R_C and spectral radius of the matrix FV^{-1} of the subsequent generation were calculated.

given

$$R_C = \rho FV^{-1} \quad (40)$$

where

ρ is the spectral radius

F is matrix of infection term at disease free equilibrium

V is matrix of transmission term at disease free equilibrium

$$FV^{-1} = \left(\frac{\partial F_i(E^0)}{\partial x_i}\right)\left(\frac{\partial V_i(E^0)}{\partial x_i}\right)^{-1} \quad (41)$$

$$F = \begin{pmatrix} \frac{\alpha(1-\varepsilon\psi)IS}{N} \\ 0 \end{pmatrix} \quad (42)$$

$$V = \begin{pmatrix} A_1 I \\ A_2 T \end{pmatrix} \quad (43)$$

$$F = \begin{pmatrix} \frac{\alpha(1-\varepsilon\psi)S}{N} & 0 \\ 0 & 0 \end{pmatrix} \quad (44)$$

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$$V = \begin{pmatrix} A_1 & 0 \\ 0 & A_2 \end{pmatrix} \quad (45)$$

$$V^{-1} = \frac{1}{A_1 A_2}\begin{pmatrix} A_2 & 0 \\ 0 & A_1 \end{pmatrix} \quad (46)$$

$$V^{-1} = \begin{pmatrix} \frac{1}{A_1} & 0 \\ 0 & \frac{1}{A_2} \end{pmatrix} \quad (47)$$

$$FV^{-1} = \begin{pmatrix} \frac{\alpha(1-\varepsilon\psi)\mathrm{S}}{N} & 0 \\ \tau & 0 \end{pmatrix}.\begin{pmatrix} \frac{1}{A_2} & 0 \\ 0 & \frac{1}{A_3} \end{pmatrix} \quad (48)$$

$$R_0 = \frac{\alpha(1-\varepsilon\psi)\mathrm{S}}{A_1 N} \quad (65)$$

2.5 Local Stability of Disease Free Equilibrium State (E^0)

The derived fundamental reproduction number was utilised to analyse the equilibrium point's stability; the resulting value is:

Theorem 1: Locally asymptotically stability for Disease-free state E_0 is attained if and only if: $R_0 < 1$

Proof: After evaluating the disease-free equilibrium point E^0, the Jacobian matrix of the system is obtained as follows:

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial V} & \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial T} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial V} & \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial T} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial V} & \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial I} & \frac{\partial f_1}{\partial T} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_4}{\partial V} & \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial T} & \frac{\partial f_4}{\partial R} \\ \frac{\partial f_5}{\partial V} & \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial T} & \frac{\partial f_5}{\partial R} \end{pmatrix} \quad (66)$$

$$J = \begin{pmatrix} -\mu & \theta & 0 & 0 & 0 \\ 0 & -(\theta+\mu) & \frac{\alpha(1-\psi)\mathrm{S}}{N} & 0 & \omega \\ 0 & 0 & \frac{\alpha(1-\psi)\mathrm{S}}{N} - A_1 & 0 & 0 \\ 0 & 0 & \tau & -A_2 & 0 \\ 0 & 0 & 0 & A_4 & -A_3 \end{pmatrix} \quad (67)$$

Reducing to upper triangular factor, using maple 18

$$J = \begin{pmatrix} -\mu & \theta & 0 & 0 & 0 \\ 0 & -(\theta+\mu) & -\frac{\alpha(1-\varepsilon\psi)\mathrm{S}}{N} & 0 & \omega \\ 0 & 0 & \frac{\alpha(1-\varepsilon\psi)\mathrm{S}}{N} - A_1 N & 0 & 0 \\ 0 & 0 & \tau & -A_2 & 0 \\ 0 & 0 & 0 & A_4 & -A_3 \end{pmatrix} \quad (68)$$

$$\left|J_{E_0} - \lambda I\right| = \begin{pmatrix} -\mu-\lambda & \theta & 0 & 0 & 0 \\ 0 & -(\theta+\mu)-\lambda & -\frac{\alpha(1-\varepsilon\psi)\mathrm{S}}{N} & 0 & \omega \\ 0 & 0 & \frac{\alpha(1-\varepsilon\psi)\mathrm{S}}{N} - A_1 N - \lambda & 0 & 0 \\ 0 & 0 & \tau & -A_2-\lambda & 0 \\ 0 & 0 & 0 & 0 & -A_3-\lambda \end{pmatrix} = 0 \quad (69)$$

$$\lambda_1 = -\mu \quad (70)$$

$$\lambda_2 = -(\theta+\mu) \quad (71)$$

$$\lambda_3 = \frac{\alpha(1-\varepsilon\psi)\mathrm{S}}{N} - A_1 N \quad (72)$$

$$\lambda_4 = -A_2 \quad (86)$$

$$\lambda_5 = -A_3 \quad (87)$$

$R_C < 1$ if $\lambda_3 < 0$

i.e,

$$\frac{\alpha(1-\varepsilon\psi)\mathrm{S}}{N} - A_1 N < 0 \quad (88)$$

$$NA_1\left(\frac{\alpha(1-\varepsilon\psi)\mathrm{S}}{NA_1} - 1\right) < 0 \quad (89)$$

$$\frac{\alpha(1-\varepsilon\psi)\mathrm{S}}{NA_1} - 1 < 0 \quad (90)$$

$$R_C - 1 < 0 \quad (91)$$

$$R_C < 1 \quad (92)$$

Therefore, Disease-free equilibrium is therefore locally asymptotically stable.

2.6 Global Disease-Free Equilibrium Stability State

Global equilibrium stability eliminated the restriction on the first condition of the model variable. In global asymptotic stability, methods perspective equilibrium for all initial conditions. Castilo-Chavez *et al.* global's stability theorem and the Lyapunov theorem are examples of methods for demonstrating the disease-free equilibrium of global stability; however, the Lyapunov theorem was utilised in this study.

Theorem 2: The disease-free state E_0 is asymptotically stable globally if $R_c \leq 1$

Proof: Consideration is given to the Lyaponuv function in order to ascertain the global stability of the disease-free equilibrium.

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$$L = NI \quad (93)$$

$$L^1 = N\frac{dI}{dt} \quad (94)$$

$$L^1 = N(\frac{\alpha(1-\varepsilon\psi)SI}{N} - A_1 I) \quad (95)$$

$$L^1 = NI(\frac{\alpha(1-\varepsilon\psi)S}{N} - A_1) \quad (96)$$

$$L^1 = NI(\frac{\alpha(1-\varepsilon\psi)S}{NA_1} - 1) \quad (97)$$

$but \;\; N^0 \le S^0$

$$and \;\; R_C = \frac{\alpha(1-\varepsilon\psi)\mathrm{S}}{NA_1} \quad (98)$$

$$L^1 = NI(R_C - 1) \le 0 \quad (99)$$

$$R_C - 1 \le 0 \quad (100)$$

$$R_C \le 1 \quad (101)$$

Hence the DFE is globally Asymptotically stable.

3.0 Results

Variable Values Estimation

To investigate the impact of the proposed correction assessments, simulations of the model were performed using the numbers of the variables and parameters listed in Tables 1 and 2, respectively. Our findings were presented at the conclusion of this work.

Table 1 The initial values for variables of the typhoid model in Nigeria as at year 2021.

S/No	Variables	Values	Source
1	N	170127200	Assumed
2	V	1000	Assumed
3	S	170123700	[28]
4	I	1000	Assumed
5	T	800	Assumed
6	R	700	Assumed

Parameters Value Estimation

Correspondingly, based on the provided data and predominantly typhoid epidemiology, we have partly referenced and assumed values of model restrictions in Table 2.

Table 2 Baseline values for the typhoid model's parameters

S/NO	Parameters	Values	Source
1	θ	0.02	Assumed
2	Λ	15000	Assumed
3	α	0.2	Assumed
4	μ	0.018	[230]
5	γ	0.1	Assumed
6	δ	0.008	Assumed
7	τ	Varies	Assumed
8	φ	0.4	Assumed
9	ψ	Varies	Assumed
10	ε	Varies	Assumed
11	ω	0.02	Assumed

Figure 2 – 4 present the graphical simulation of the model

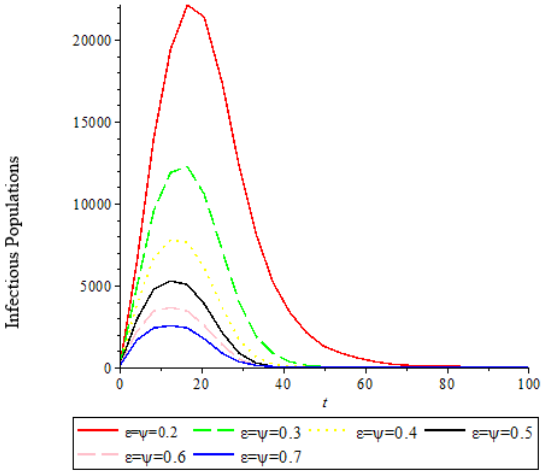

Figure 1. Graph of Link Between Public Sensitization and Infectious Population

The Figure 4.1 shows that if there are enough public sensitization and people are complying with it. There will few typhoid Infected individuals in the population. That is, the higher the public sensitization and people are compliance level, the lower the infectious population. This suggests the need for more awareness in the area of hygiene in order to prevent typhoid.

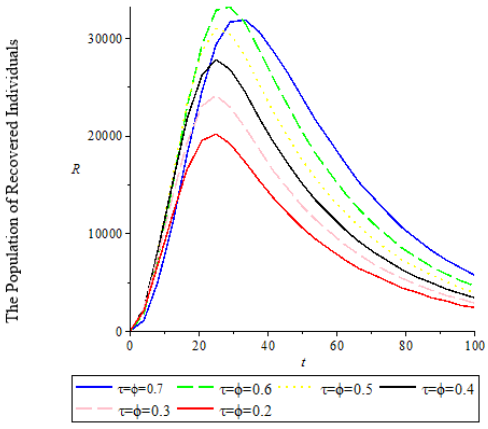

Figure 2. Graph of Link Between Treatment and Recovered Individual

Figure 2 demonstrates that the recovered class increases more rapidly when the treatment rate and rate of recovery class development rise. After a period of time, the population decreases owing to the fading of the medications or treatments delivered, and the population returns to the vulnerable compartment.

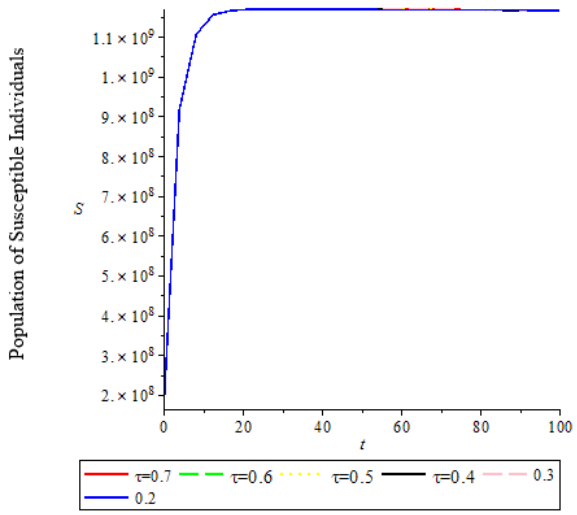

Figure 3. **Graph of Link Between Treatment and Susceptible Individual**

Figure 3 demonstrates that the treatment amount has no substantial influence on the susceptible group, since the effectiveness of the therapy diminishes with time and more people become infected everyday..

4.0 Conclusion and Recommendations

Using systems of ordinary differential equations, a mathematical model of the influence of a public sensitization programme on typhoid disease control was developed in this study. The local stability study of the Disease-Free Equilibrium State (DFE) of the model may be judged to be stable if $R_C < 1$ is stable and globally stable if $R_C \leq 1$.

A mathematical model for the effect of public sensitization program in controlling typhoid fever was formulated. The work has demonstrated the conditions for the stability of the model equilibrium point free of illness. The research has demonstrated the impact of the public sensitization campaign on the infectious and recovery compartments of the model. The model demonstrates that the transmission of typhoid illness is highly dependent on public sensitization; hence, health professionals should place a greater emphasis on adhering to the high level of cleanliness taught during public awareness programmes. Treatment of infected patients as soon as possible is strongly advised.

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