Mathematical Theory and Modeling
ISSN 2224-5804 (Paper) ISSN 2225-0522 (Online)
Vol.5, No.8, 2015

ON THE EXISTENCE OF A STOCHASTIC MODEL OF TYPHOID FEVER

Andrew Omame, Reuben Andrew Umana, Nneka Onyinyechi Iheonu and Simeon Chioma Inyama
Department of Mathematics, Federal University of Technology, Owerri, Imo State, Nigeria

Abstract

In this work a stochastic model is developed and analyzed for the dynamics of Typhoid fever. The model includes susceptible, vaccinated, infected, carrier and recovered individuals. The model used in this work is based on a deterministic model. The deterministic model is transformed into a stochastic model and solved numerically using MATLAB. It is shown that the model satisfies the conditions for existence and uniqueness of solution. The simulation result also shows that increased vaccination rate will lead to Typhoid fever reduction and possible extinction.

Keywords and phrases: Stochastic model, typhoid fever, transition probability, Wiener process, vaccination.

1.0 INTRODUCTION

Typhoid is endemic in many developing countries and remains a substantial public health problem despite recent progress in water and sanitation coverage (Lauria *et al*, 2009). Globally, it is estimated that typhoid fever causes more than 16 million cases of illness each year, resulting in more than 600,000 deaths (Kariuki *et al*, 2004). Typhoid fever is a communicable disease found only in human and occurs due to systemic infection mainly by salmonella typhi organism. It is an acute generalized infection of the intestinal lymphoid tissue and the gall bladder. Incubation period, usually 10-14 days but it may be as short as 3 days or as long as 21 days. The epidemic is transmitted by feco-oral route or urine-oral route either directly through hands soiled with faeces or urine of cases or carriers or indirectly by ingestion of contaminated water, milk, food or through flies (Singh, 2001).

The literature and development of mathematical epidemiology are well documented and can be found in Anderson (1991), Bailey (1975), Brauer and Castillo-Chavez (2000). Modeling and transmission dynamics of typhoid is an important topic for a lot of researchers; Lauria et al (2009) developed an optimization model for reducing typhoid cases in developing countries without increasing public spending. Their work suggested that the magnitude of herd protection effects greatly influences the total number of cases avoided and the value of public treatment cost savings. Also, Kalajdzievska and Li (2011) developed a mathematical model for assessing the effects of carriers on the transmission dynamics of infectious diseases such as typhoid. They concluded that carriers play a significant role in the transmission of infectious diseases. Mushayabasa et al (2013) studied an epidemiological model for direct and indirect transmission of typhoid fever. Sensitivity analysis of the basic reproduction number suggested that indirect typhoid transmission has more impact on determining typhoid prevalence compared to direct transmission.

Other researchers on infectious diseases include Kalu and Inyama (2012), Omame and Inyama (2014), to mention but a few.

These models are mostly deterministic, and assume that all input variables are deterministic functions of time, ignoring completely the randomness of these variables. Because the

fundamental biological processes involved are stochastic, ignoring their inherent randomness may lead to misleading and erroneous results.
In this work, we overcome these limitations by extending the work of Mushayabasa (2011) and converting it to a stochastic model. Numerical simulations are carried out and analyzed with the aid of MATLAB.

2.0 MODEL FORMULATION

2.1 The Deterministic model equations

Let $S(t)$, $I(t)$, $C(t)$, $V(t)$ and $R(t)$ denote the susceptible, infected, carrier, vaccinated and recovered individuals at time t.

The flow diagram for the deterministic model is given by

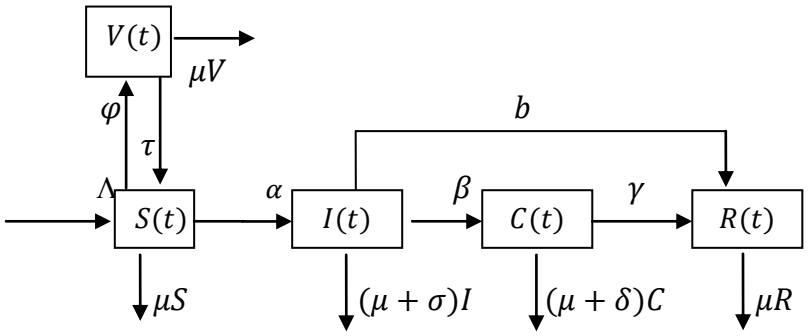


Fig. 2.1

Λ : Recruitment rate

α: Per capita infection rate

β: Rate at which infected becomes carrier

γ: Rate of recovery for carriers

b: Recovery rate for infectious individuals

σ: Per capita disease-induced mortality rate

φ: Rate at which susceptible individuals are vaccinated

μ : Natural mortality rate

τ : Rate at which the vaccine wanes

δ: Per capita carrier-induced mortality rate

Susceptibles $S(t)$: The number of individuals who can be infected but may have not yet contracted the *salmonella typhi* but may contract it if exposed to any mode of its transmission.
Infectives $I(t)$: the number of individuals who have contracted the *salmonella typhi* and are capable of transmitting it.

Carriers $C(t)$: the number of individuals (treated or not) who, although apparently healthy themselves, continue to shed bacteria in their faeces and are capable of transmitting the infection.

Recovered $R(t)$: the number of individuals who are recovered after treatment and are immune to the disease. The model assumes that there is drug efficacy.

Vaccinated $V(t)$: the number of individuals who have been vaccinated and can return to the susceptible class due to waning vaccination rate.

The differential equations for the deterministic model are as follows:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda - \alpha SI - (\varphi + \mu)S + \tau V \\
\frac{dV}{dt} &= \varphi S - (\tau + \mu)V \\
\frac{dI}{dt} &= \alpha SI - (\beta + b + \sigma + \mu)I \\
\frac{dC}{dt} &= \beta I - (\gamma + \delta + \mu)C \\
\frac{dR}{dt} &= \gamma C + bI - \mu R
\end{aligned}\right\} \quad \text{.....(2.1)}$$

The transition probabilities are shown on Table 2.1

Table 2.1 TRANSITION PROBABILITIES

Change	Probability	Event
$[1\ 0\ 0\ 0\ 0]^T$	$P_1 = \Lambda \Delta t$	Birth of a susceptible
$[-1\ 1\ 0\ 0\ 0]^T$	$P_2 = \varphi S \Delta t$	Susceptible becomes vaccinated
$[-1\ 0\ 1\ 0\ 0]^T$	$P_3 = \alpha SI \Delta t$	Susceptible becomes infected
$[-1\ 0\ 0\ 0\ 0]^T$	$P_4 = \mu S \Delta t$	Susceptible dies natural death
$[1\ -1\ 0\ 0\ 0\ ]^T$	$P_5 = \tau V \Delta t$	Vaccinated becomes susceptible
$[0\ -1\ 0\ 0\ 0]^T$	$P_6 = \mu V \Delta t$	Vaccinated dies natural death
$[0\ 0\ -1\ 1\ 0]^T$	$P_7 = \beta I \Delta t$	Infected becomes a carrier
$[0\ 0\ -1\ 0\ 1]^T$	$P_8 = bI \Delta t$	Infected recovers
$[0\ 0\ -1\ 0\ 0]^T$	$P_9 = (\mu + \sigma) I \Delta t$	Infected dies
$[0\ 0\ 0\ -1\ 1]^T$	$P_{10} = \gamma C \Delta t$	Carrier recovers
$[0\ 0\ 0\ -1\ 0]^T$	$P_{11} = (\mu + \delta) C \Delta t$	Carrier dies
$[0\ 0\ 0\ 0\ -1]^T$	$P_{12} = \mu R \Delta t$	Recovered dies naturally

2.2 The Stochastic Model Equations (SDEs)

Using the second modelling procedure developed by Allen, *et al* (2008), the stochastic model equations are given by

$$\left.\begin{aligned}
d\vec{X} &= \vec{f}(t, \vec{X}(t))dt + G(t, \vec{X}(t))d\vec{W}(t) \\
\vec{X}(0) &= \left[X_1(0), X_2(0), X_3(0), X_4(0)\right]^T
\end{aligned}\right\} \quad \text{.....(2.2)}$$

The drift vector is defined as

$$\vec{f} = \sum_{j=1}^{12} p_j \vec{\lambda_j}$$

where $\vec{\lambda_j}$ and p_j are the random changes and transition probabilities respectively (see Table 2.1)

The drift vector $\vec{f}$ is given by

$$\vec{f} = \begin{pmatrix} \Lambda - \alpha SI - (\varphi + \mu)S + \tau V \\ \varphi S - (\tau + \mu)V \\ \alpha SI - (\beta + b + \sigma + \mu)I \\ \beta I - (\gamma + \delta + \mu)C \\ \gamma C + bI - \mu R \end{pmatrix}$$

The diffusion matrix G has the entries

$$\lambda_{i,j} p_j^{1/2}$$

where $\lambda_{i,j}$ and p_j ($i = 1,...,5, j = 1,...,12$) are the components of the random changes and transition probabilities respectively.

The diffusion matrix G is given by:

$$G = \begin{pmatrix} (\Lambda)^{1/2} & -(\varphi S)^{1/2} & -(\alpha SI)^{1/2} & -(\mu S)^{1/2} & (\tau V)^{1/2} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & (\varphi S)^{1/2} & 0 & 0 & -(\tau V)^{1/2} & -(\mu V)^{1/2} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (\alpha SI)^{1/2} & 0 & 0 & 0 & -(\beta I)^{1/2} & -(bI)^{1/2} & -(\mu I + \sigma I)^{1/2} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & (\beta I)^{1/2} & 0 & 0 & -(\gamma C)^{1/2} & -(\mu C + \delta C)^{1/2} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & (bI)^{1/2} & 0 & (\gamma C)^{1/2} & 0 & -(\mu R)^{1/2} \end{pmatrix}$$

Also, where

$$\vec{W}(t) = [W_1(t), W_2(t), W_3(t), W_4(t), W_5(t), W_6(t), W_7(t), W_8(t), W_9(t), W_{10}(t), W_{11}(t), W_{12}(t)]^T$$

is a vector of twelve independent Wiener processes.

The probability distribution $p(t,x)$ of the solution that solves the above stochastic differential equation satisfies the forward kolmogorov equation given below:

$$\frac{\partial p(t,x)}{\partial t} = -\sum_{i=1}^{5} \frac{\partial [p(t,x) f_i(t,x)]}{\partial x_i} + \frac{1}{2} \sum_{i=1}^{5} \sum_{j=1}^{12} \sum_{l=1}^{12} \frac{\partial^2 [p(t,x) g_{i,l}(t,x) g_{j,l}(t,x)]}{\partial x_i \partial x_j} \quad \text{.....(2.3)}$$

2.3 Existence and Uniqueness theorem for SDEs (Ito, 1951)

Assume that the coefficients in the following system of stochastic differential equations

$$dX_t^i = a_i(t, X_t)dt + \sum_{i=1}^{n} \sum_{j=1}^{m} b_{ij}(t, X_t) dW_t^j \Big\} \quad \text{.....(2.4)}$$

where

$X_t = (X_t^1, X_t^2, ...X_t^n)^T$

$W_t = (W_t^1, W_t^2, ...W_t^m)^T$

$a_i(t, X_t)$ is an $n-$dimensional vector with entries $a_i(t, x)$

$b_{ij}(t, X_t)$ is an $n \times m$ matrix with entries $b_{ij}(t, x)$

satisfy the following Lipschitz and growth conditions in (2.5) for some constant $k < \infty$, and

for all $t \in R$ and $x, y \in R^n$

$$\left.\begin{aligned} \|a_i(t,x) - a_i(t,y)\| &\le k\|x-y\| \\ \|b_{ij}(t,x) - b_{ij}(t,y)\| &\le k\|x-y\| \\ \|a_i(t,x)\| &\le k\|x\| \\ \|b_{ij}(t,x)\| &\le k\|x\| \end{aligned}\right\} \quad \text{.....(2.5)}$$

$$\|b\| = \sqrt{\sum_{i=1}^{n}\sum_{j=1}^{m} b_{ij}(x)^2},$$

$$\|a\| = \sqrt{\sum_{i=1}^{n} a_i(x)^2}$$

Then for each $x_0 \in R^n$ there is a unique solution to the system of stochastic differential equations (2.4) such that $X_0 = x_0$.

Now consider equation (2.2) with the following:

$$X(0) = [S(0), V(0), I(0), C(0), R(0)] = [70, 54, 64, 28, 10]$$

$$f_1 = \Lambda - \alpha SI - (\varphi + \mu)S + \tau V$$
$$f_2 = \varphi S - (\tau + \mu)V$$
$$f_3 = \alpha SI - (\beta + b + \sigma + \mu)I$$
$$f_4 = \beta I - (\gamma + \delta + \mu)C$$
$$f_5 = \gamma C + bI - \mu R$$

Then there exists a constant $M > 0$ such that

$$\left|\frac{\partial f_1}{\partial S}\right| = |\alpha I - (\varphi + \mu)| \le M, \quad \left|\frac{\partial f_1}{\partial V}\right| = |\tau| \le M, \quad \left|\frac{\partial f_1}{\partial I}\right| = |\alpha S| \le M \quad \left|\frac{\partial f_1}{\partial C}\right| = \left|\frac{\partial f_1}{\partial R}\right| = 0$$

$$\left|\frac{\partial f_2}{\partial S}\right| = |\varphi| \le M, \quad \left|\frac{\partial f_2}{\partial V}\right| = |\tau + \mu| \le M, \quad \left|\frac{\partial f_2}{\partial I}\right| = \left|\frac{\partial f_2}{\partial C}\right| = \left|\frac{\partial f_2}{\partial R}\right| = 0$$

$$\left|\frac{\partial f_3}{\partial S}\right| = |\alpha I| \le M\,,\ \left|\frac{\partial f_3}{\partial I}\right| = |\beta + b + \sigma + \mu| \le M,\ \left|\frac{\partial f_3}{\partial V}\right| = \left|\frac{\partial f_3}{\partial C}\right| = \left|\frac{\partial f_3}{\partial R}\right| = 0,$$

$$\left|\frac{\partial f_4}{\partial I}\right| = |\beta| \le M,\ \left|\frac{\partial f_4}{\partial C}\right| = |\gamma + \delta + \mu| \le M \quad \left|\frac{\partial f_4}{\partial S}\right| = \left|\frac{\partial f_4}{\partial V}\right| = \left|\frac{\partial f_4}{\partial R}\right| = 0\,,$$

$$\left|\frac{\partial f_5}{\partial I}\right| = |b| \le M,\ \left|\frac{\partial f_5}{\partial C}\right| = |\gamma| \le M,\ \left|\frac{\partial f_5}{\partial R}\right| = |\mu| \le M,\ \left|\frac{\partial f_5}{\partial S}\right| = \left|\frac{\partial f_5}{\partial V}\right| = 0\,,$$

The elements of the diffusion matrix are continuously differentiable.

Also, for the system (2.2)

$$\|f\| = \sqrt{\sum_{i=1}^{5} f_i(x)^2} \quad \text{and} \quad \|G\| = \sqrt{\sum_{i=1}^{5}\sum_{j=1}^{12} g_{ij}(x)^2}$$

where

$$\|f\| = \sqrt{[\Lambda + \alpha SI - (\varphi + \mu)S + \tau V]^2 + [\varphi S - (\tau + \mu)V]^2 + [\alpha SI - (\beta + b + \sigma + \mu)I]^2 + [\beta SI - (\gamma + \delta + \mu)C]^2 + [\gamma C + bI - \mu R]^2}$$

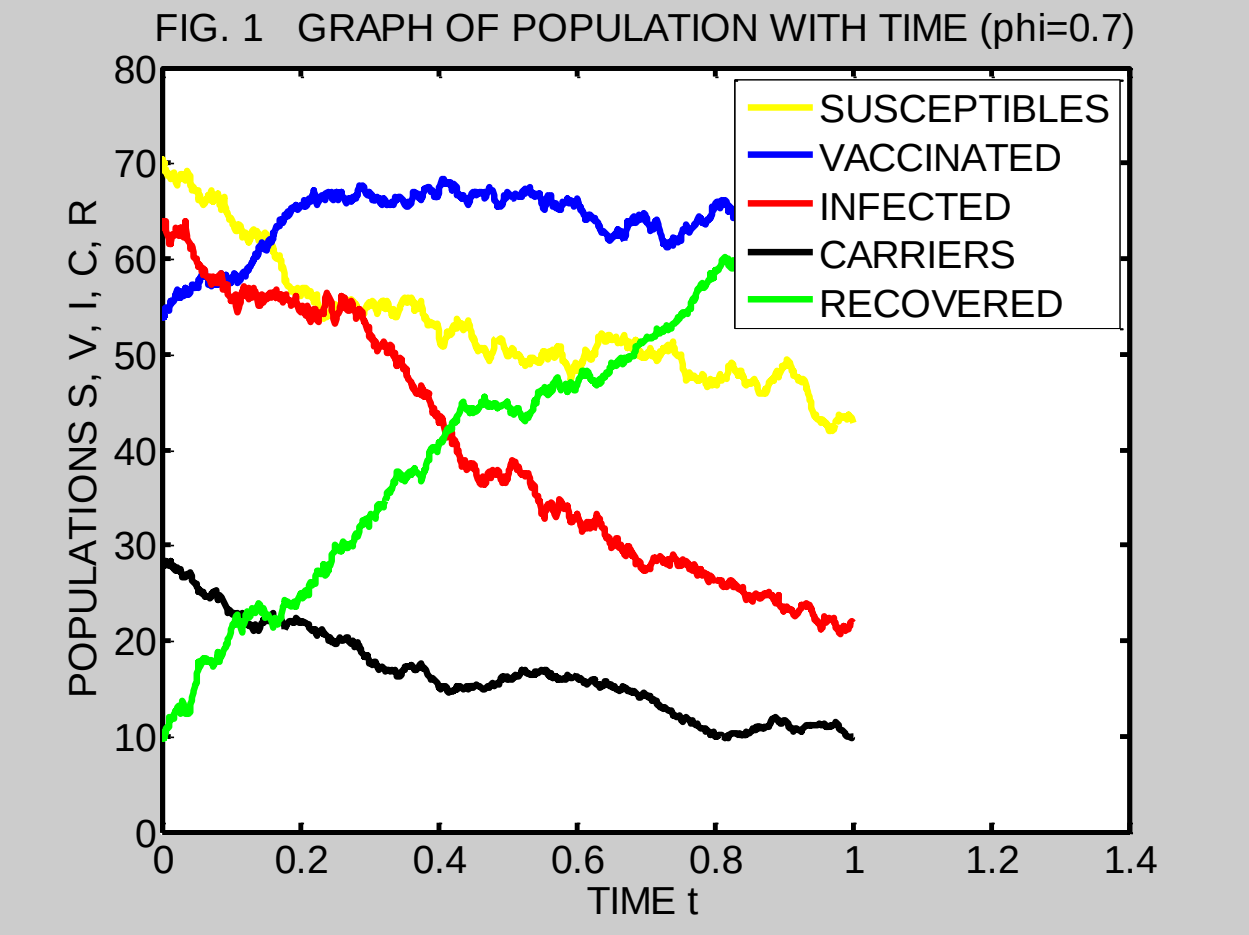
$$\|G\| = \sqrt{\Lambda + 2\varphi S + 2\alpha SI + \mu S + 2\tau V + \mu V + 2\beta I + 2bI + (\mu I + \sigma I) + 2\gamma C + (\mu C + \delta C) + \mu R}$$

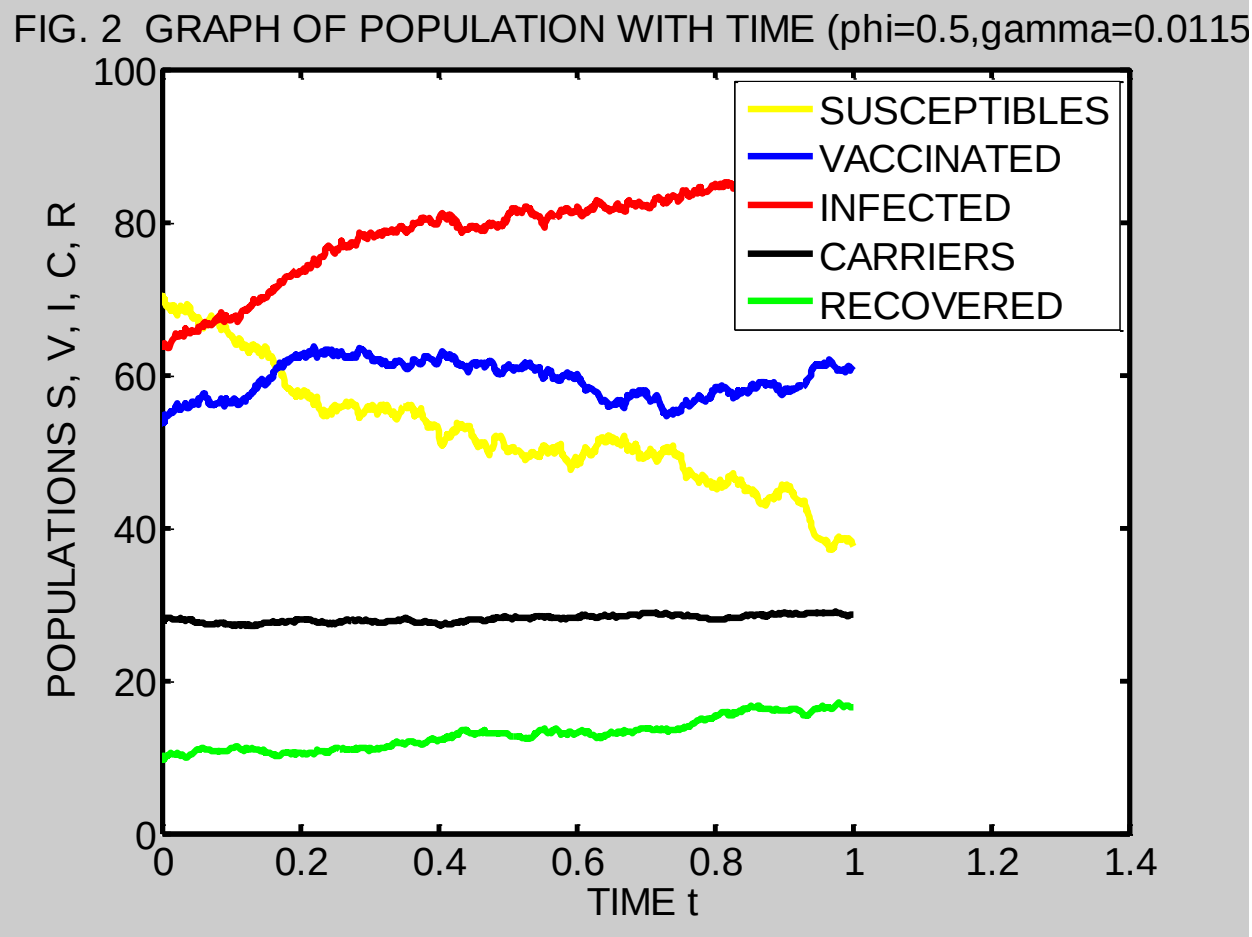
Both f_i and g_{ij} are continuously differentiable at $[S(0), V(0), I(0), C(0), R(0)]$ and hence satisfy the Lipschitz condition (by the Mean value theorem for calculus). Since the norms exist, they are bounded. They drift and the diffusion matrices are therefore bounded. Hence, they satisfy the conditions for existence and uniqueness of solution.

3.0 Numerical Simulation

We set year as a unit of time. The natural mortality rate μ is postulated to be equal to the inverse of the life expectancy at birth (Santrock, 2007). It is about 49 years in Nigeria (UNAIDS-WHO, 2004); that is $\mu = 1/49 = 0.02041 yr^{-1}$. The recruitment rate Λ controls the total population size because $N \sim \Lambda/\mu$. We set $\Lambda = \mu \times 41 yr^{-1}$(Song, *et al 2002*). The per capita infectious rate α is taken to be $0.0072\ yr^{-1}$. The per capita Typhoid-induced mortality rate σ is $0.9\ yr^{-1}$ (Adetunde, 2008). For this simulation we set the carrier-induced mortality rate δ to be $0.013\ yr^{-1}$(Adetunde, 2008). Following Lauria *et al* (2009), φ is the rate at which susceptible humans are vaccinated and is taken to be $0.9\ yr^{-1}$. τ is the rate at which vaccination wanes and is taken to be $0.33\ yr^{-1}$ (Lauria *et al*, 2009). We take the recovery rate as the inversion of an average period between the typhoid activation and the moment of recovery. According to Adetunde (2008), rate of recovery for carriers γ is taken

to be 0.115 yr^{-1} while the rate of recovery for infectious individuals b is set to be 0.096 yr^{-1}. The initial populations which were taken to be $S(0)$= 70, $V(0)$= 54, $I(0)$= 64, $C(0)$=28, $R(0)$= 10, were based on the examination of monthly medical record at the Usman Danfodio University Teaching Hospital, Sokoto in 2004 (Ameh and Opara, 2004)





4.0 Results

The figures above show the graphs of the different populations: susceptible, vaccinated, infected, carrier, and recovered individuals against time. In figure 1, the susceptibles, infected, and carrier populations decrease drastically with time while the vaccinated and recovered populations increase with time. This is as a result of high vaccination rate of 0.70 yr^{-1}. If the vaccination rate increases, more people become immune to the typhoid disease. Figure 2 shows the recovered and vaccinated populations decreasing with time whereas there is an increase in the infected, susceptible and carrier populations. This is as a result of low vaccination rate of 0.5 yr^{-1} as well as low recovery rate of 0.0115 yr^{-1} for carriers. When the vaccination rate decreases, and no proper treatment for carriers and infected populations, the disease persists in the population. In the result of Adetunde (2008), the higher the population density, the greater the risk of instability of the permanent immunity equilibrium, which implies the possibility of an epidemic in the population. He also concluded that an increase in the rate of infection with time decreases the susceptible population to almost zero level. Also, in the result of Mushayabasa (2011), the infective population rose to a peak at the initial stage of the epidemic and then decreased sharply with time as the vaccination rate is

increased. However, in our result as the vaccination rate is increased with time, the infective population falls but with fluctuations before reaching a low level. As the time increases with increasing vaccination rate, half of the population becomes susceptible. The paths are not smooth as in the deterministic approach of previous results. This is as a result of the fact that the deterministic model is insensitive to stochastic variations which can occur in actual population.

5.0 CONCLUSION AND FURTHER RESEARCH

In this work a stochastic differential equation model is developed and analyzed for the prevalence of typhoid disease. The model, which is a multidimensional diffusion process, includes susceptible, carrier, infected, vaccinated and treated or recovered individuals. The result shows that increased vaccination rate will lead to typhoid disease extinction while low vaccination rate as well as improper treatment for carriers will produce an epidemic.

Further research may include analytical solution of the present model, as well as applications of typhoid modelling in nationwide-scale study and implicate the proposed model to other contagious diseases of childhood such as chickenpox, malaria, cholera, etc.

ACKNOWLEDGEMENT

The authors are grateful to Usman Danfodio University Teaching Hospital, Sokoto, Nigeria for providing the data used for our simulation. The authors are also grateful to Prof. G.C.E. Mbah whose constructive criticism led to the improvement of this work.

REFERENCES

Adetunde, I.A. (2008): The mathematical models of the dynamical behaviour of tuberculosis disease in the upper region of the northern part of Ghana: a case study of Bawku. *Current Research in Tuberculosis*, 1(1): 1-6

Ameh, I.G. and Opara, W.E.K.(2004): "Typhoid: A record of cases in Sokoto, Nigeria". *Pakistan Journal of Biological Sciences*

Allen E.J. Allen L.J.S.,Arciniega A., Greenwood P. E. (2008) "Construction of Equivalent Stochastic Differential Equation Models " *Stoch. Anal. Appl*. 26(2): 274-297.

Anderson, R.M.(1991): "Infectious disease of humans". *Dynamics and control*, Oxford University Press, USA.

Bailey, N. (1975): "The mathematical theory of infectious diseases". *Charles Griffin*, London

Brauer, F and Castillo-Chavez, C. (2000): "Mathematical models in population biology and epidemiology". Springer, USA.

Ito, K. (1951): "On Stochastic Differential Equations", *Memoirs of American Mathematical Society* (4):1-51

Kalajdzievska, D and Li, M.Y. (2011): "Modelling the effects of carriers on transmission dynamics of infectious diseases". *Mathematical Biosciences and Engineering.* Vol. 8, no. 3: 711-722

Kalu, A.U., and Inyama, S.C.(2012): Mathematical model of the role of vaccination and treatment on the transmission dynamics of tuberculosis. *Gen. Math. Notes*, 11(1): 10-23

Kariuki, S., C. Gilks, G. Revathi and C.A. Hart (2000): "characterization of multidrug-resistant typhoid outbreaks in Kenya". *J. Clin. Microbiol*. 42: 1477-1482

Lauria, D.T., B. Maskery, C. Poulos and D, Whittington (2009): "An optimization model for reducing typhoid cases in developing countries without increasing public spending". *Vaccine.* 27:1609-1621

Mushayabasa, S. (2011): "Impact of vaccines on controlling typhoid fever in kassena-Nankana District of Upper East Regionj of Ghana: insights from a mathematical model". *J. of Modern math. and Stat.*

Omame, A., and Inyama, S.C.(2014): Stochastic model and simulation of the prevalence of measles. *Int. J. of Math. Sci. & Eng. Appls*, 8(1): 311-323

Santrock, John (2007): Life Expectancy. A total approach to Lifespan development. New York, The McGraw-Hill Companies, Inc: 128-132

Singh, B. (2001): "Typhoid fever epidemiology" *J. of Indian Acad. of Clinical Med.* Vol 2, no. 1

Song, B., C. Castillo-Chavez and J.P. Aparicio (2002): Tuberculosis models with fast and slow dynamics: The role of close and casual contacts. *Math. Biosc.* 180: 187-205

UNAIDS-WHO(2004): UNAIDS/WHO Epidemiological Fact sheet.

The IISTE is a pioneer in the Open-Access hosting service and academic event management. The aim of the firm is Accelerating Global Knowledge Sharing.

More information about the firm can be found on the homepage:
http://www.iiste.org

CALL FOR JOURNAL PAPERS

There are more than 30 peer-reviewed academic journals hosted under the hosting platform.

Prospective authors of journals can find the submission instruction on the following page: http://www.iiste.org/journals/ All the journals articles are available online to the readers all over the world without financial, legal, or technical barriers other than those inseparable from gaining access to the internet itself. Paper version of the journals is also available upon request of readers and authors.

MORE RESOURCES

Book publication information: http://www.iiste.org/book/

Academic conference: http://www.iiste.org/conference/upcoming-conferences-call-for-paper/

IISTE Knowledge Sharing Partners

EBSCO, Index Copernicus, Ulrich's Periodicals Directory, JournalTOCS, PKP Open Archives Harvester, Bielefeld Academic Search Engine, Elektronische Zeitschriftenbibliothek EZB, Open J-Gate, OCLC WorldCat, Universe Digtial Library , NewJour, Google Scholar