

# A New Mathematical Model to Simulate Infectious Disease Dynamics in Rivers State, Nigeria – I

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## Abstract

We stretch existing knowledge on model formulation and develop an enhanced mathematical model to study the pattern of spread of infectious diseases in Rivers State. The SIR model formalism was used to compartmentalize the population and the resulting model equations were solved numerically. The disease free equilibrium and endemic equilibrium of the system were established and analyzed for stability. A graph representation of the sub groups is presented and discussed based on the results from simulation.

**Keywords:** *Mathematical model, Simulation, Endemic, equilibrium, Disease free*

## 1. Introduction

Disease is “a condition of the body, or some part or organ of the body, in which its functions are disturbed or deranged; a morbid physical condition; a departure from the state of health, especially when caused by structural change (Oxford Advanced Learners Dictionary, 2006).” This definition encompasses a wide range of ailments from AIDS to arthritis, from the common cold to cancer. It is classified as infectious, non infectious and notifiable. Our interest following therefrom is in Infectious classification and our emphasis in this paper is developing a mathematical model to study the pattern at which the infectious disease spread in Rivers State.

Infectious diseases remain a major cause of illness and death in the world today (WHO, 2009). In many countries of the developed World, infectious diseases of the childhood in particular have been generally conquered. This however, is far from being the case in developing countries, where they are responsible for 45% of all death (WHO, 2008). Coming down home, the data of infectious disease prepared by the Department of Health Planning and Research, Federal Ministry of Health at our disposal shows the same trend in Rivers State. This thus brings to fore the need to properly study and understand the pattern at which these diseases spread and provide timely

information to experts who will use it in guarding against any epidemic and combating the scourge. The leading killers are acute respiratory infections, HIV/AIDs, tuberculosis, malaria and measles (Lucas and Gilles 2003). These infectious diseases can be endemic or emerging; it is endemic when it is sustained in a population without the need for external input. This means on average, each infected person is infecting exactly one other person any more the number infected will grow exponentially and there will be an epidemic, any less the disease will die out, it is emerging.

These infections are caused by introduction into the body, micro organisms such as bacteria, viruses and fungi which are disease causing pathogens that reproduce and cause disease by directly damaging the cells or by releasing toxins. This normally provokes the immune system into responding, which accounts for many common symptoms. Infection can be localized within a particular area or tissue, as a boil or distributed as influenza (British Medical Association, 2007)

An infectious disease is a clinically evident illness resulting from the presence of pathogenic microbial agents, including pathogenic viruses, pathogenic bacteria, fungi, protozoa, multi-cellular parasites, and aberrant proteins known as prions. These pathogens are able to cause disease in animals and plants. Infectious pathologies are also called communicable diseases or transmissible diseases due to their potential of transmission from one person or species to another by a replicating agent (Dorlands medical dictionary 2007). Transmission of an infectious disease may occur through one or more of diverse pathways including physical contact with infected individuals. These infecting agents may also be transmitted through liquids, food, body fluids, contaminated objects, airborne inhalation, or through vector-borne spread (Ryan et al 2004). Transmissible diseases which occur through contact with an ill person or their secretions, or objects touched by them, are especially infective, and are sometimes referred to as contagious

diseases. Infectious diseases are Health problems, which is a departure from good state of Health, caused by structural changes in which the proper body function is impaired, making it difficult for the organs of the body to function properly (Carlo, 2006)

## 2. Related Work

The literature of mathematical models is well researched and over the last few decades, studies into the pattern at which disease spread in a particular location to help provide early information for experts has gained sufficient grounds especially in Western Nigeria. A model is an approximate representation of a given part of the material world, the study or understanding of whose part it facilitates (Oko, 1998). Models may be used in the research and development or design of systems prior to their construction, or to simulate conditions, processes or entities which otherwise will be impossible or infeasible to deal with directly. Models are also used to study and modify existing system and processes and as integral parts of control systems. On the other hand modeling is the process of model building and analysis that usually culminate in a valid decision making in respect of the modeled entity. There are many types of modeling used in every day human activities; they include linguistics, physical and mathematical modeling, etc. Most research works on infectious disease simulation are done with deterministic modeling. It requires less data, relatively easy to set up and a lot of user friendly software can be developed.

The earliest account of mathematical modeling of spread of disease was carried out in 1766 by Daniel Bernoulli. Trained as a physician, Bernoulli created a mathematical model to defend the practice of inoculating against smallpox. The calculations from this model showed that universal inoculation against smallpox would increase the life expectancy from 26years 7 months to 29 years 9 months (Bernoulli, 1760). Following Bernoulli, other physicians contributed to modern mathematical epidemiology. Among the most acclaimed of these were A. G. McKendrick and W. O. Kermack, whose paper, 'A contribution to the Mathematical Theory of Epidemics' was published in 1927.

The idea behind epidemiological model is compartmentalization in which the host population is divided into distinct classes, according to epidemiological status. One of such model is to classify individuals as susceptible to the disease (S), currently infected (I) and recovered (R). The total host population is  $N = S+I+R$ . This work explores the potentials of deterministic model in simulating infectious diseases in Rivers State.

A simple deterministic (compartmental) model was formulated in this paper and was successful in predicting the behavior of an epidemic very similar to that observed in many recorded epidemics following Kermack and Mc Kendrick, 1927. Hethcote et al, 1980, developed an integral equation models which was used to simulate Endemic infectious disease. It was found that the constant parameter models include vital dynamics (Birth and Death), immunization and distributed infection period. Okwa et al 2009, simulated the transmission dynamics of malaria in four selected ecological zones of Nigeria in the rainy season. They found that the most infected mosquitoes were seen in the rain forest and rainy season. Githeko and Ndegwa 2001, developed a mathematical model for predicting malaria epidemic and found that the rate of infection is dependent on the level of rainfall and temperature.

Koriko and Yusuf, 2008 developed a mathematical model for the tuberculosis disease population dynamics and posited that population dynamics depends more on the number of actively infected people in the population at the initial time and also on the disease incidence transmission rate at a given time. Also, it was shown that the disease – free equilibrium is stable while the endemic equilibrium may or may not be stable on the various values of the model parameters.

This work presents an enhanced and more efficient mathematical formulation to simulate infectious diseases dynamics in Rivers State, Nigeria using a modified SIR formalism

## 3. Research Methodology

A comprehensive epidemiological data was collected from Federal Ministry of Health, Podium Block, Port Harcourt, Rivers State. The data contained reported cases of diseases (communicable, non communicable and notable) prepared by the Department of Health Planning and Research, Federal Ministry of Health. These data were obtained from the different Primary Health Centers located across the Local Government Areas of Rivers State and covered the period of 10 years from the year 2000 to 2009.

**Table 1: 2006 census figure of the 23 Local Government Areas (LGAs) in Rivers state, collected from the Ministry of Local Government Affairs, Rivers State**

LGA Name	Area (sq.km)	Population	Administrative capital	Code
Port Harcourt	109	541,115	Port Harcourt	500
Obio-Akpor	260	464,789	Rumuokoro	500
Okrika	222	222,026	Okrika	500
Ogu/Bolo	89	74,683	Ogu	500
Eleme	138	190,884	Eleme	501
Tai	159	117,797	Sakpenwa	501
Gokana	126	228,828	Kpor	501
Khana	560	294,217	Bori	502
Oyigbo	248	122,687	Afam	502
Opobo/Nkoro	130	151,511	Opobo Town	503
Andoni	233	211,009	Ngo	503
Bonny	642	215,358	Bonny	503
Degema	1,011	249,773	Degema	504
Asari-Toru	113	220,100	Buguma	504
Akuku-Toru	1,443	156,006	Abonnema	504
Abua/Odual	704	282,988	Abua	510
Ahoada West	403	249,425	Akinima	510
Ahoada East	341	166,747	Ahoada	510
Ogba/Egbema/Ndoni	969	284,010	Omuku	510
Emohua	831	201,901	Emohua	511
Ikwerre	655	189,726	Isiokpo	511
Etche	805	249,454	Okehi	512
Omuma	170	100,366	Eberri	512



**Figure 1: Map of Rivers State showing all the Local Government Areas**

#### 4.0 Model/Analysis

A mathematical model is a set of equations, which are the mathematical translation of hypothesis (or assumptions) when interpreting model prediction. It is thus important to bear in mind the underlying assumptions. An assumption, by our definition, is an unverified proposition, tentatively accepted to explain certain facts or to provide a basis for further investigation.

In 2008, Koriko and Yusuf simulated tuberculosis disease population dynamics. In that model, which was based on the standard conventional theory of SIR, the assumption was that the increasing population was a constant rate. This enhanced model proposes that people affect the population randomly. This is comparable to a wave train approaching the shoreline from different directions than from the positive x-direction, say, Asor and Okeke (2000), Asor (2001), Asor and Okeke (2001). Following therefrom, we make the following proposition:

### 4.1 Proposition 1

Let P and N represent the Study area population and the new entrants into the population at random respectively. Then, P is as susceptible as  $\Sigma(P,N)$ . So,

$$S = \Sigma(P,N)$$

The proof of the proposition is quite simple and we design the following model parameters and their description to generate our sets of differential equations:

Let	
P	Population size
N	total number of new people coming in randomly
S	total number of people in the study area that are susceptible
$I_A$	total number of people that are actively infected by the disease
$I_L$	total number of people that are latently infected by the disease
$N_p$	Probability that a susceptible person is not vaccinated
E	Efficacy rate of vaccine
$T_L$	success rate of latent disease therapy
$T_A$	Active diseases treatment cure rate
R	Disease incidence rate per susceptible
d	Human natural death rate
p	Proportion of infection instantaneously degenerating in active condition
i	Disease induced death rate
$\beta_A$	Breakdown rate from latent to active condition

Based on the standard SIR model, we compartmentalize the model population into the susceptible (S) and the infected (I) which is further broken down into latently infected ( $I_L$ ) and actively infected ( $I_A$ ) while the recovered sub-population is ploughed back into the susceptible group due to the possibility of re-infection after successful treatment of the earlier infection. The model monitors the temporary dynamics in the population of susceptible people  $S(t)$ , disease latently infected people  $I_L(t)$  and disease actively infected people  $I_A(t)$  as captured in the model system of ordinary differential equations that follows:

$$\left. \begin{aligned} \frac{dS}{dt} &= \Sigma(P, N) - \alpha I_A S - \delta S + T_A I_A + T_L I_L \\ \frac{dI_L}{dt} &= (1-p)\alpha I_A S - \beta_A I_L - T_L I_L - \delta I_L \\ \frac{dI_A}{dt} &= p\alpha I_A S + \beta_A I_L - T_A I_A - \delta I_A - \epsilon I_A \\ S(0) &= N, I_L(0) \geq 0, I_A(0) \geq 0. \end{aligned} \right\} \text{Eq. (1)}$$

Changes in the susceptible population are accountable

- i. from new entrants into the study area randomly at a random rate N;
- ii. by the people who are cured from active disease;
- iii. from those successfully treated for latent disease;
- iv. from natural death at death rate, d and
- v. from infection with an incidence rate of infection.

The instantaneous incidence rate of infection, a, per susceptible will depend on number of contact with infection per susceptible people and the probability that the contact lead to infection. In the same way, the latently infected population dynamics depends on the proportion of the infection that results in latent infection  $((1-p)\alpha I_A S)$  and this is reduced by loss due to natural death, successful treatment of latent patients, and occasional breakdown of latent infection into active infection.

Finally, the change in the actively infected population is dependent on the proportion of disease infection of the susceptible sub-population degenerating instantaneously into active infection plus the number of latently infected people breaking down into active patients while this is diminished due to natural death, successful cure of active patients, and death caused as a result chronic active infection.

It is important to note that in this model, the assumption is that successfully treated latent patients and cured active patients become susceptible immediately after their treatments whenever they are exposed to infection again irrespective of their infection history. Realistically, this group may sometimes enjoy some temporary immunity to infection and whenever they are re-infected, they may require more intensive treatment as a result of some multi-drug resistance that must have been built over time.

### 5.0 Equilibrium Analysis

Modeling infectious diseases demands that we investigate whether the disease spread could attain a pandemic level or it could be wiped out. The equilibrium analysis helps to achieve this. Thus, we shall consider two equilibriums – the diseases-free equilibrium and endemic equilibrium.

At equilibrium,

$$\left. \begin{aligned} \frac{dS}{dt} &= 0, \\ \frac{dI_L}{dt} &= 0, \\ \frac{dI_A}{dt} &= 0 \end{aligned} \right\} \text{Eq. (2)}$$

Hence, the system of equation becomes:

$$\left. \begin{aligned} P + N - \alpha I_A S - \delta S + T_A I_A + T_L I_L &= 0 \\ (1 - P)\alpha I_A S - \delta S - \beta_A I_A - T_L I_L - \delta I_L &= 0 \\ p\alpha I_A S + \beta_A I_L - T_A I_A - \delta I_A - \epsilon I_A &= 0 \\ S(0) = N, I_L(0) \geq 0, I_A(0) \geq 0 \end{aligned} \right\} \text{Eq. (3)}$$

Obviously, at the disease-free situation,

$$I_L = 0, \text{ and } I_A = 0.$$

Thus, from equation (3) we have

$$P + N - \delta S = 0 \quad \text{Eq. (4)}$$

$$\Rightarrow S = \frac{P + N}{\delta} = N^0 \quad \text{Eq. (5)}$$

So, the disease-free equilibrium is  $(N^0, 0, 0)$ . However, for endemic equilibrium, we solve equation (3) for  $S, I_L$  and  $I_A$  respectively. This gives

$$S = \frac{P + N + T_A I_A + T_L I_L}{\delta + \alpha I_A} = S^*, \quad \text{Eq. (6)}$$

$$I_L = \frac{(1 - P)\alpha I_A S}{\beta_A + T_L + \delta} = I_L^*, \quad \text{Eq. (7)}$$

$$I_A = \frac{\beta_A I_L}{T_A + \epsilon + \delta - p\alpha S} = I_A^*, \quad \text{Eq. (8)}$$

Thus, we can take our endemic equilibrium as  $(S^*, I_L^*, I_A^*)$  Eq. (9)

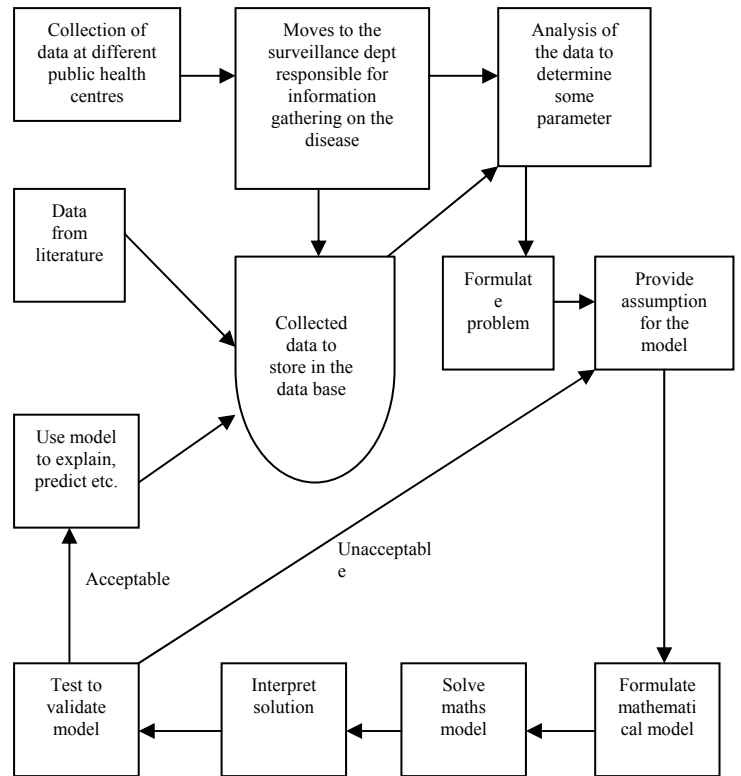


Figure 2: Data Flow Diagram for the proposed System

## 6.0 CONCLUSION

Following analysis, modeling infectious diseases demands that we investigate whether the disease spread could attain a pandemic level or it could be wiped out. The equilibrium analysis helps to achieve this.

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