Assessing the Efficacy of Whooping Cough Control Strategy using Mathematical Model

Bashir Sule, Muhammad M. Hussaini, & Yahaya Sambo
Department of Mathematics,
Aminu Saleh College of Education,
Azare, Bauchi State,
Nigeria.

Huzaifa Abba Usman
Department of Computer Education,
Aminu Saleh College of Education,
Azare, Bauchi State,
Nigeria.

Abstract
The study investigates the efficacy of Whooping cough control measures with the use of mathematical modelling application. The primary purpose of the study is to provide a vaccination model to carry out a stability analysis on control measures so as to evaluate their efficiency and effectiveness. This includes a maternally derived immunity, susceptible, infected and recovered compartmental model (MSIR). Data were collected on incidences of whooping cough from the Federal Medical Centres of the four states across the North Eastern states of Nigeria to obtain parameter values. Maple software was used for stability analysis. It was finally recommended that Immunization organisations in collaboration with government ought to reinforce routine DTaP vaccination system and effort should be intensified toward increasing the duration of efficacy of the vaccines utilized and decreasing the level of contact rate accordingly.

Keywords: Mathematical model, efficacy, Whooping cough, Vaccination, Stability, simulation

Introduction

One of the respiratory diseases which is most severe in infants is pertussis (Bordetella pertussis) popularly known as whooping cough. In 1950s, it was globally a major cause of infant mortality before massive vaccination was introduced (Fabricius *et al.* 2013). They also observed a noticeable drop in the disease incidence due to the implementation of the pertussis immunization programme.

Vaccination of pregnant women with Tdap protects the mother from becoming infected with pertussis and making her less likely to transmit pertussis to her infant. This is so because it stimulates the development of maternal antipertussis antibodies that pass through the placenta, providing the new born with protection against pertussis in early life (CDC, published data 2013). Crowcroft and Pebody (2006) argued that even though there is high vaccination coverage over decades whooping cough has not been eliminated in any country. They added that the number of deaths in young infants caused as a result of pertussis infection has been increased.

Although a new vaccine DTPads, which was a combination of cellular pertussis vaccines with diphtheria (DTx) and tetanus tox-oids (TTx), was recommended for routine immunization of infants, it was accused of causing CNS injury. But this was later disproved (Robbins et al.

2014). The vaccines reduced the incidence of pertussis significantly in the infants throughout the developed countries including the United States. It was also supported by Luz, Codeco, Werneck and Struchiner, (2006) who emphasize that against severe cases of whooping cough, vaccination of children is an effective preventive strategy. Contrarily, although there is high vaccination coverage of young infants for more than 50 years in many developed countries, pertussis is still classified as a re-emerging disease.

According to Munoz *et al.* (2014) there is a significant higher concentration of antibodies to all vaccine antigens in infants which was as a result of maternal immunization with Tdap from birth. This continues till introduction of immunization with DTap at the age of two months and did not alter infant responses considerably to DTaP. They also emphasized that to provide a conclusive evidence of the safety and efficacy of Tdap immunization at birth, further research is therefore needed which led to our current study.

According to Anderson and May 1991; Keeling and Rohani 2007 as in Andrew and Alan (2010) deterministic models have traditionally formed the basis of mathematical epidemiology. The susceptible-infected-recovered (SIR) and the susceptible-exposed-infected-recovered (SEIR) models are the typical methods. However in this study we proposed an MSIR epidemic mathematical model that has compartments; maternally derived immunity-susceptible-Infectious-Recovered.

Due to the continuous outburst of infectious diseases, epidemiological mathematics have been grown exponentially wider since the middle of the 20^{th} century (Shah and Gupta, 2013). The period of time between the generations of infected individuals determines the speed at which an epidemic spreads through a population (Keeling and Danon, 2009). A major goal in the treatment of individuals with chronic infectious diseases is to bring about an improvement in their functioning and well-being (Stewart, 1989). According to Abdulrazak, *et al*. (2012) when the incidence of an infection starts to increase, people and government think of how best to combat the outbreak. Vaccination campaigns may be costly and time consuming endeavour, so any tool that may enable the campaign to become directed or to predict the outcome is highly valuable.

Mathematical modelling is becoming an increasingly important branch of Mathematics as computers expand our ability to translate mathematical equations and formulations into concrete conclusions concerning the world, both natural and artificial that we live in. The mechanisms by which disease spread have been studied using the modelling of infectious diseases. The spread of infectious diseases has all the time remain a major threat to public health (WHO, 2008). Millions of people infected annually from the diseases and millions of them die as a result (Badshah, Porwal and Tiwari, 2013).

Several forms of control measures exist, all operate by reducing the average amount of transmission between infectious and susceptible individuals, selecting a suitable control strategy depends on the nature of the disease. This research is geared towards investigating the existing measures to find out how efficient and effective they are. Therefore, with the employment of Mathematical model, hopefully the problem will be addressed. This is a step forward to see total eradication of the disease.

Statement of the problem

In the twentieth century, pertussis was a standout amongst the most well-known adolescence infections and a significant reason for youth mortality in the United States. Prior to the

accessibility of pertussis vaccine in the 1940s, more than 200,000 instances of the pertussis were accounted for yearly. Since wide spread use of the vaccine began, a low incidence of more than 80% has been recorded compared with the pre-vaccine era.
According to World Health Organisation (2008), the disease remains a major health problem among children in developing countries, with an estimate of 195,000 deaths resulting from the disease. This problem is currently affecting children especially under the age of ten in both developed and developing countries. Report have shown a total of 48,277 cases of pertussis worldwide in 2012 and 28639 cases in 2013.

Aims and objectives
The general aim of this research work is to evaluate the efficacy of whooping cough control measures in Nigeria. To deal with this, it is necessary to address relevant and sufficient objectives which may include the following:

- **a.** To develop a vaccination model for the Pertussis.
- **b.** To carry out a stability analysis on control measures to evaluate their efficiency and effectiveness.

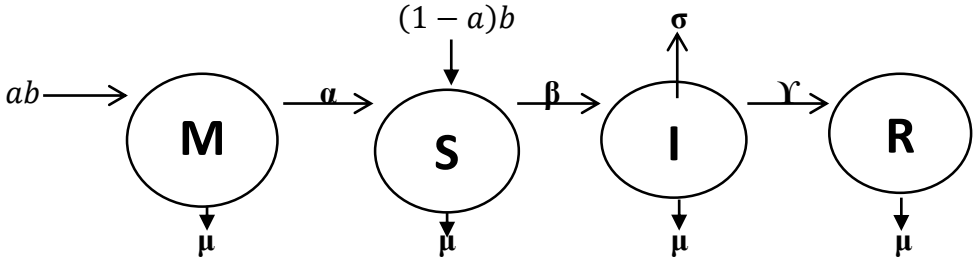
Methodology
This research work is a quantitative study that involves deterministic approach of differential equations and will utilise numerical techniques to perform simulation using Euler's method. The population covers all pertussis record cases for the past one year from four Federal Medical Centres in the North-Eastern States of Nigeria. The data collected will be used to obtain accurate simulation results. Stability analysis will also be carried out using Maple Software.

Model Formulation
For many infections, including pertussis, babies are not born into the susceptible compartment but are immune to the disease for the first few months of life due to protection from maternal antibodies. This new detail can be shown by including an M class, for maternally derived immunity at the beginning of the model.
Therefore, in this study we proposed an MSIR epidemic mathematical model that has compartments; maternally derived immunity, Susceptible, Infectious and Recovered. We prefer this compartment model over others because it generalise some of the models such as SIR and SIS as it takes care of the M class which is left in those models. It does not also make things too complicated as in the models with more and more compartments

Assumptions
Let us denote M, S, I, R be the population members of each class. Regarding the transmission and incubation period, the following assumptions are considered.

1. The population is mixing in a homogeneous manner. That is everyone has equal chances of contacting the disease that a proportion of the population of new-borns is immunized against pertussis infection through vaccination;
2. Expiration of duration of vaccine efficacy at constant rate;
3. Birth and death occur at constant rate;
4. That people in the compartment have equal natural death rate;
5. Recovery occurs at a constant rate;
6. That all new-borns are previously uninfected by pertussis and therefore join either the immunized or the susceptible compartment depending on whether they are vaccinated or not.

Epidemiological diagram of the model:



Definition of Parameters

$\boldsymbol{a}$ is the proportion of new birth given Diphtheria-Tetanus-acellular-Pertussis (DTaP) vaccines at birth to protect against infection;
$\boldsymbol{ab}$ is the proportion of incoming individuals immunized against infection;
$\boldsymbol{(1-a)b}$ represents population of individuals not immunized against infection;
μ represents natural death rate;
α represents the rate of vaccine efficacy;
β represents rate of transmission;
ϒ stands for the rate of successful cure of infections pertussis patients (removal rate);
σ represents death rate caused as a result of chronic pertussis infection.

Model Description

The population is partitioned into four compartments. A proportion a of new births were given DTaP vaccines at birth to protect them against infection. The immunized compartment changes due to the coming in of the immunized children into the population in which it was assumed that a proportion ab of the incoming individuals are immunized against infection. The susceptible population increases because of the coming in of new births that were not immunized against infection into the population at the rate $(1-a)b$, this compartment reduces because of the expiration of vaccine efficacy duration at the rate α and also by natural death at the rate μ and infection with incident rate of infection β.

Similarly, the population dynamic of the infectious class grows with the instantaneous incidence rate of infection β resulting from contacts of members of susceptible class with infectious class. This class also reduces by natural death rate μ, successful cure of infectious pertussis patients at the rate ϒ and death caused as a result of chronic pertussis infection at the rate σ. Finally, the dynamic of the recovered class increases with successful cure of infectious pertussis patients at the rate ϒ and decreases by natural death rate μ.

Model Equations

Using the assumptions stated above, the model will be of the form

$$\frac{dM}{dt} = ab - \alpha M - \mu M$$
$$\frac{dS}{dt} = (1-a)b + \alpha M - \beta SI - \mu S$$
$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I - \sigma I$$
$$\frac{dR}{dt} = \gamma I - \mu R$$

At steady states:

$$\frac{dM}{dt} = \frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

This gives;

$$ab - \alpha M - \mu M = 0 \quad \text{.....(1.1)}$$
$$(1-a)b + \alpha M - \beta SI - \mu S = 0 \quad \text{.....(1.2)}$$
$$\beta SI - \gamma I - \mu I - \sigma I = 0 \quad \text{.....(1.3)}$$
$$\gamma I - \mu R = 0 \quad \text{.....(1.4)}$$

From (1):

$$M = \frac{ab}{\alpha+\mu} \quad \text{... ... ... (1.5)}$$

From (3): $(\beta S - (\gamma + \mu + \sigma)I = 0$

$$Either \quad I = 0 \quad or \quad S = \frac{\gamma + \mu + \sigma}{\beta} \quad \text{... ... ... (1.6)}$$

From (4): $R = \frac{\gamma I}{\mu}$ ... ... (1.7)

$At \quad I = 0, \quad R = 0$

Substitute for $I = 0 \quad and \quad M = \frac{ab}{\alpha+\mu}$ in equation (1.2) to get:

$$(1-a)b + \frac{\alpha ab}{\alpha + \mu} = \mu S$$

$$S = \frac{\alpha b + (1-a)\mu b}{\mu(\alpha + \mu)} \quad \text{... ... ... (1.8)}$$

Hence the Disease-Free-Steady States are:

$$M^* = \frac{ab}{\alpha + \mu} \quad \text{... ... ... (1.9)}$$

$$S^* = \frac{\alpha b + (1-a)\mu b}{\mu(\alpha + \mu)} \quad \text{... ... ... (2.0)}$$
$$I^* = 0 \quad \text{... ... ... (2.1)}$$
$$R^* = 0 \quad \text{... ... ... (2.2)}$$

Substituting equations (1.5) and (1.6) in (1.2) gives

$$(1-a)b + \alpha\left(\frac{ab}{\alpha+\mu}\right) - \beta I\left(\frac{\gamma+\mu+\sigma}{\beta}\right) - \mu\left(\frac{\gamma+\mu+\sigma}{\beta}\right) = 0$$

$$\Rightarrow \quad I = \frac{(\alpha+\mu)[\beta b - \mu(\gamma+\mu+\sigma)] - \beta\mu ab}{\beta(\alpha+\mu)(\gamma+\mu+\sigma)} \quad \text{... ... ... (2.3)}$$

And also, substituting equation (9) in (4) gives:

$$R = \frac{\gamma[(\alpha+\mu)\{\beta b - \mu(\gamma+\mu+\sigma)\} - \beta\mu ab]}{\beta\mu(\alpha+\mu)(\gamma+\mu+\sigma)} \quad \text{... ... ... (2.4)}$$

Hence the epidemic steady states are:

$$M^* = \frac{ab}{\alpha+\mu} \quad \text{... ... ... (2.5)}$$
$$S^* = \frac{\gamma+\mu+\sigma}{\beta} \quad \text{... ... ... (2.6)}$$
$$I^* = \frac{(\alpha+\mu)[\beta b - \mu(\gamma+\mu+\sigma)] - \beta\mu ab}{\beta(\alpha+\mu)(\gamma+\mu+\sigma)} \quad \text{... ... ... (2.7)}$$
$$R^* = \frac{\gamma[(\alpha+\mu)\{\beta b - \mu(\gamma+\mu+\sigma)\} - \beta\mu ab]}{\beta\mu(\alpha+\mu)(\gamma+\mu+\sigma)} \quad \text{... ... ... (2.8)}$$

Basic Reproduction Number, R_0

In epidemiology, the basic reproduction number refers to the number of new cases of infection linked to a person infected shortly after the pathogen was introduced into population with no pre-existing immunity. Generally the higher the value of R_O, the harder it is to control the epidemic. When $R_o < 1$, the infection will die out in the long run. But when $R_o > 1$, the infection will invade.

The Threshold,

$$\frac{1}{R_O} = \frac{\gamma + \mu + \sigma}{\beta} \quad \dots\dots\dots\dots\dots\dots\dots\dots (2.9)$$

This implies, $R_O = \frac{\beta}{\gamma+\mu+\sigma}$ $\dots\dots\dots\dots\dots\dots\dots\dots (3.0)$

Numerical calculation of R_0

Take parameters in the model system as ϒ = 0.213, β = 0.235, μ = 0.0034, σ = 0.142

For these parameter values, the basic reproduction number for the Disease-Free Equilibrium (DFE) is $R_o = 0.6557 < 1$ .This shows that the infection is temporal and the disease dies out in time. If we keep the value of μ unchanged and let ϒ= 0.0213, β = 0.0651, σ = 0.0142, then the basic reproduction number is calculated as R_O = 1.6735 > 1 and the disease becomes endemic. In this situation, an average infectious individual is able to replace itself and the number of infected rises and an epidemic reveals.

Stability Analysis

Having obtained the equilibrium states, we now move forward to investigate the stability of the equilibrium states by examine the behaviour of the model near the equilibrium states. This is done by computing the Jacobian matrix as follows:

$$J = \begin{pmatrix} -(\alpha+\mu) & 0 & 0 & 0 \\ \alpha & -(\beta I+\mu) & -\beta S & 0 \\ 0 & \beta I & \beta S-(\gamma+\mu+\sigma) & 0 \\ 0 & 0 & \gamma & -\mu \end{pmatrix}$$

The characteristic equation of the Jacobian matrix is

$$|J-\lambda I| = \begin{vmatrix} -(\alpha+\mu+\lambda) & 0 & 0 & 0 \\ \alpha & -(\beta I+\mu+\lambda) & -\beta S & 0 \\ 0 & \beta I & \beta S-(\gamma+\mu+\sigma) & 0 \\ 0 & 0 & \gamma & -(\mu+\lambda) \end{vmatrix} = 0$$

$$(\alpha+\mu+\lambda)\{-(\beta I+\mu+\lambda)[-(\mu+\lambda)(-\gamma-\mu-\sigma+\beta S-\lambda)]\} = 0$$

Zero Equilibrium State

At the Disease-Free-Equilibrium (DFE);

$$\alpha+\mu+\lambda = 0$$

That is $\lambda_1 = -\alpha-\mu$

And $\beta I+\mu+\lambda = 0$

Putting $I = 0 \quad in \quad \beta I+\mu+\lambda = 0, \quad gives \quad \lambda_2 = -\mu$

Then $\mu+\lambda = 0 \quad \Rightarrow \quad \lambda_3 = -\mu$

Also, $-\gamma-\mu-\sigma+\beta S-\lambda = 0$

Putting $S = \frac{\alpha b+(1-a)\mu b}{\mu(\alpha+\mu)}$, $gives$

$$\lambda_4 = \frac{\alpha\beta b+(1-a)\beta\mu b}{\mu(\alpha+\mu)} - (\gamma+\mu+\sigma)$$

$\lambda_1, \lambda_2, \lambda_3$ are all negative and λ_4 should also be negative if

$\frac{\alpha\beta b+(1-a)\beta\mu b}{\mu(\alpha+\mu)} < (\gamma+\mu+\sigma)$

Non-Zero Equilibrium State

To investigate the stability of the endemic equilibrium state, we did apply the Bellman and Cooke's theorem as stated in Momoh, *et al.*, (2014).

Bellman and Cooke's theorem

$Let\ H(z) = P(z, e^x)\ where\ P(z,w)$ is a polynomial with principal term.

Suppose, $H(iy), y \in \Re$, is separated into its real and imaginary parts:

$H(iy) = F(y) + iG(y)$

If the zeros of $H(y)$ have negative real parts, then the zeros of $F(y)$ and $G(y)$are real, simple and alternate. Then

$$F(0)G'(0) - F'(0)G(0) > 0, \forall y \in \Re \quad \ldots\ldots\ldots\ldots\ldots (3.1)$$

Conversely, all zeros of $H(z)$ will be in the left half plane provided that either of the following condition is satisfied:

1. All the zeros of $F(y)$ and $G(y)$ are real, simple and alternate and the inequality (3.1) is satisfied at least for one y
2. All zeros of $F(y)$ are real and for each zero, the relation (3.1) is satisfied
3. All zeros of $G(y)$ are real and for each zero, the relation (3.1) is satisfied

Applying the theorem:

$$|J-\lambda I| = \lambda^4 + (4\mu - \beta S + \gamma + \sigma + \beta I + \alpha)\lambda^3 + (-3\beta S\mu + 3\gamma\mu + 6\mu^2 + 3\sigma\mu + 3\mu\beta I + 3\alpha\mu - \beta S\beta I - \alpha\beta S + \beta\gamma I + \alpha\gamma + \beta\sigma I + \alpha\sigma + \alpha\beta I)\lambda^2 + (3\gamma\mu^2 + 3\mu^2\sigma + 3\beta I\mu^2 + 3\alpha\mu^2 - 2\beta S\beta I\mu - 2\beta S\alpha\mu + 2\beta I\gamma\mu + 2\alpha\gamma\mu + 2\alpha\mu\sigma + 2\beta I\alpha\mu - \alpha\beta S\beta I + \alpha\gamma\beta I + \alpha\sigma\beta I - 3\mu^2\beta S + 4\mu^3)\lambda + \mu(\beta S + \gamma + \mu + \sigma)(\beta I + \mu)(\alpha + \mu) = 0$$

$$H(iq) = (iq)^4 + (4\mu - \beta S + \gamma + \sigma + \beta I + \alpha)(iq)^3 + (-3\beta S\mu + 3\gamma\mu + 6\mu^2 + 3\sigma\mu + 3\mu\beta I + 3\alpha\mu - \beta S\beta I - \alpha\beta S + \beta\gamma I + \alpha\gamma + \beta\sigma I + \alpha\sigma + \alpha\beta I)(iq)^2 + (3\gamma\mu^2 + 3\mu^2\sigma + 3\beta I\mu^2 + 3\alpha\mu^2 - 2\beta S\beta I\mu - 2\beta S\alpha\mu + 2\beta I\gamma\mu + 2\alpha\gamma\mu + 2\alpha\mu\sigma + 2\beta I\alpha\mu - \alpha\beta S\beta I + \alpha\gamma\beta I + \alpha\sigma\beta I - 3\mu^2\beta S + 4\mu^3)(iq) + \mu(\beta S + \gamma + \mu + \sigma)(\beta I + \mu)(\alpha + \mu) = 0$$

$H(iq) = F(q) + iG(q)$ Where:

$$F(q) = q^4 + (-3\mu\beta S + 3\mu\gamma + 6\mu^2 + 3\mu\sigma + 3\mu\beta I + 3\mu\alpha - \beta S\beta I - \alpha\beta S + \gamma\beta I + \alpha\gamma + \sigma\beta I + \alpha\sigma + \alpha\beta I)(q)^2 + \mu(-\beta S + \gamma + \mu + \sigma)(\beta I + \mu)(\alpha + \mu)$$

$$F(0) = \mu(\beta S + \gamma + \mu + \sigma)(\beta I + \mu)(\alpha + \mu)$$

And

$$F'(q) = 4q^3 + 2(-3\mu\beta S + 3\mu\gamma + 6\mu^2 + 3\mu\sigma + 3\mu\beta I + 3\mu\alpha - \beta S\beta I - \alpha\beta S + \gamma\beta I + \alpha\gamma + \sigma\beta I + \alpha\sigma + \alpha\beta I)q$$

$$F'(0) = 0$$

Also,

$$G(q) = (4\mu - \beta S + \alpha + \gamma + \sigma + \beta I)q^3 + (3\gamma\mu^2 + 3\mu^2\sigma + 3\mu^2\beta I + 3\alpha\mu^2 - 2\mu\beta S\beta I - 2\alpha\mu\beta S + 2\gamma\mu\beta I + 2\alpha\gamma\mu + 2\alpha\mu\sigma + 2\alpha\mu\beta I - \alpha\beta S\beta I + \alpha\gamma\beta I + \alpha\sigma\beta I - 3\mu^2\beta S + 4\mu^3)q$$

$$G(0) = 0$$

$$G'(q) = 3(4\mu - \beta S + \alpha + \gamma + \sigma + \beta I)q^2 + (3\gamma\mu^2 + 3\mu^2\sigma + 3\mu^2\beta I + 3\alpha\mu^2 - 2\mu\beta S\beta I - 2\alpha\mu\beta S + 2\gamma\mu\beta I + 2\alpha\gamma\mu + 2\alpha\mu\sigma + 2\alpha\mu\beta I - \alpha\beta S\beta I + \alpha\gamma\beta I + \alpha\sigma\beta I - 3\mu^2\beta S + 4\mu^3)$$

$$G'(0) = (3\gamma\mu^2 + 3\mu^2\sigma + 3\mu^2\beta I + 3\alpha\mu^2 - 2\mu\beta S\beta I - 2\alpha\mu\beta S + 2\gamma\mu\beta I + 2\alpha\gamma\mu + 2\alpha\mu\sigma + 2\alpha\mu\beta I - \alpha\beta S\beta I + \alpha\gamma\beta I + \alpha\sigma\beta I - 3\mu^2\beta S + 4\mu^3)$$

Therefore,

$$J = F(0)G'(0) > 0 \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots (3.2)$$

Substituting the expressions for $S^{*}and\ I^{*}$ in equation (3.2) above, we evaluate J using the parameter values with the Maple software and the result is presented in table 1.1 below:

Table 1.1

a	b	α	β	γ	μ	σ	J	*Remark*
0.30	0.90	0.10	0.00	0.00	0.01	0.00	0.00000000000000	Threshold
0.30	0.90	0.20	1.00	0.10	0.01	0.10	0.000001648565089	Stable
0.30	0.90	0.30	1.50	0.20	0.01	0.30	-0.00006251714876	Unstable
0.30	0.90	0.40	2.00	0.30	0.01	0.40	-0.00008066708356	Unstable
0.30	0.90	0.50	2.50	0.40	0.01	0.20	0.00009293714470	Stable
0.30	0.90	0.60	3.00	0.50	0.01	0.20	0.0002617861087	Stable
0.30	0.90	0.70	3.50	0.60	0.01	0.20	0.0005364792478	Stable
0.30	0.90	0.80	4.00	0.70	0.01	0.00	0.002377517469	Stable
0.30	0.90	0.90	4.50	0.80	0.01	0.10	0.0002754776803	Stable
0.30	0.90	1.00	5.00	0.90	0.01	1.00	-0.002089504255	Unstable

The following figures show the simulation results of the model using the parameter values collected from the four selected medical centres named as H_1, H_2, H_3, and H_4

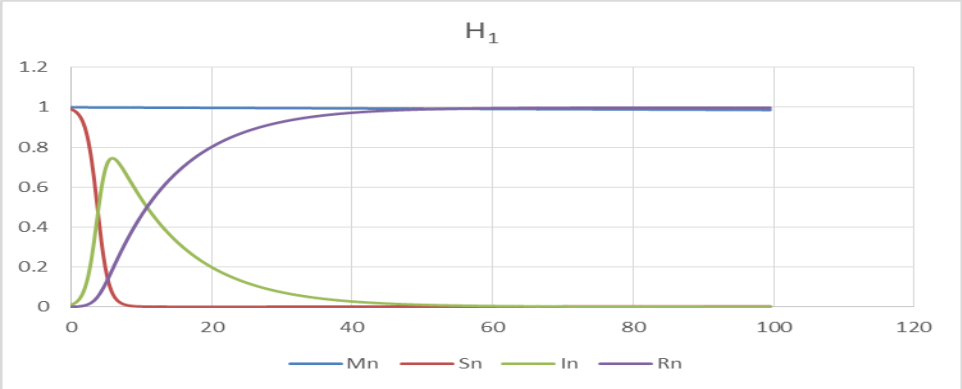


Figure 1.1 *Simulation result of* H_1

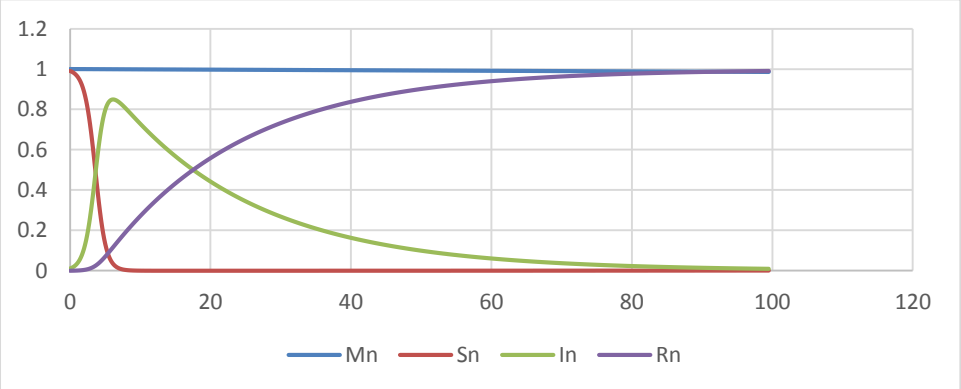


Figure 1.2 *Simulation result of* H_2

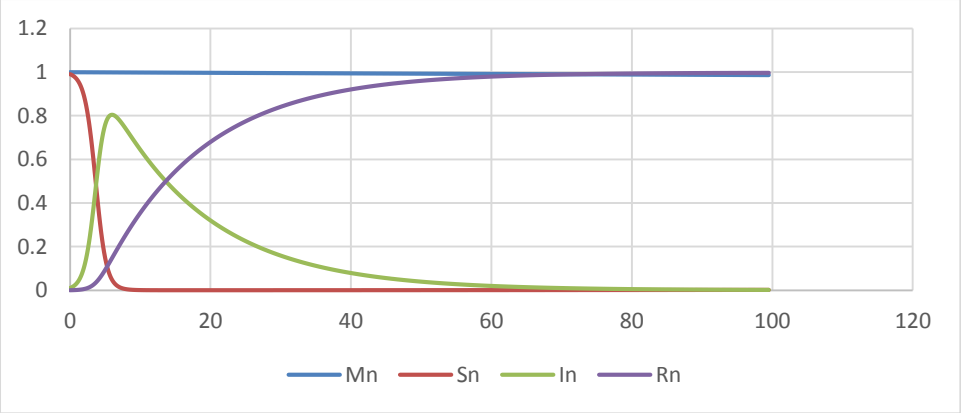


Figure 1.3 *Simulation result of* H_3

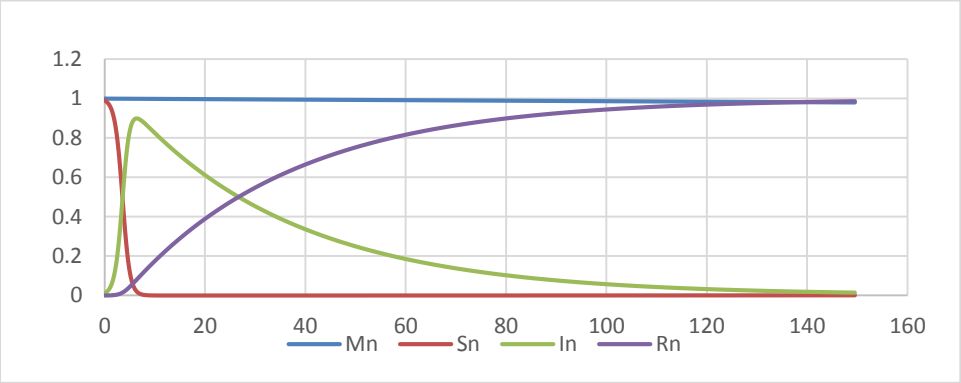


Figure 1.4 *Simulation result of* H_4

From figure 1.1, it is clearly seen that every new born babies were given protection from maternal antibodies. They were immunised within the first few months of life and pass through a maternally derived immunity. This compartment remained static because the birth and death occur at constant rate. Some of these individuals moved into the susceptible compartment where everyone has equal chance of becoming infected. As infection starts manifesting, the population in the susceptible class reduces because some babies became infected and moved to the infectious class while others die naturally. This process continues till vaccination campaign was implemented. Then individuals in the infectious compartment begin to recover from the disease.

According to figure 1.1, infected individual get recovered at around forty-five (45) days of infection. From figure 1.2, we observed that the infected individual can get recovered within eighty-five (85) days of infection. Figure 1.3 indicates that the infected individual can be recovered at around sixty-five (65) days of infection. Whereas figure 1.4 shows that infected individual can get recovered from the disease within hundred and fifty (150) days of infection. The disparity in the duration for every individual to recover from the disease as seen above, depends upon the type of vaccine used and the time of application.

Discussion

In this study, stability analysis of Whooping Cough have been investigated on assessing the efficacy of its measures of control. Bellman and Cooke's Theorem techniques was used in the analysis of this model to test for the stability. This fundamental theorem establishes the analysis of the stability of characteristic equation as stated by Momoh, *et al.*, (2014). The system (1.1) – (1.4) is locally stable around the epidemic equilibrium state. When the contact rate increases

from 1.0 to 1.5 then to 2.0, the system is unstable. However, with increase the vaccine efficacy, although the contact rate increases, the system around the epidemic equilibrium state remain stable up till when the contact rate is kept below 5.0 and otherwise the epidemic equilibrium state is unstable.

For the DFE to be stable, it is necessary that all the four eigenvalues are negative and this will be achieved by having

$$\frac{\alpha\beta b + (1-a)\beta\mu b}{\mu(\alpha+\mu)} < (\gamma + \mu + \sigma)$$

This indicates that at this state, Whooping Cough dies out in time. This was equally observed by Fabricius *et al.* (2013) who realised a noticeable drop in the disease incidence due to the implementation of the pertussis immunization programme. However, there is need for the persistence of Vaccination of pregnant women with DTap to protects the mother from becoming infected with pertussis and making her less likely to transmit pertussis to her infant.

Conclusion

In this research, stability analysis of pertussis has been investigated on the effect of DTaP vaccine to the prevention of whooping cough using an MSIR Mathematical model. It was successfully proved that provided the four eigenvalues found to be negative the disease free equilibrium state is stable. At this point, it is relevant to indicate that pertussis will be completely eradicated. However, it is important to note that for the population to be sustained, the recovery rate from infectious class must be greater or equal to the natural death rate combined with the death rate due to infection else the population will approach to extinction.

Recommendation

Immunization organisations in collaboration with government ought to reinforce routine DTap vaccination system and effort should be intensified toward increasing the duration of efficacy of the vaccines utilized and decreasing the level of contact rate accordingly. The model developed in this paper can be used in interactive workshops with health planners and other stakeholders in the analysis of pertussis control strategies so that participants could gain a better understanding of how vaccination campaign become much effective in controlling the disease.

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References

Abdulrazak A. J., Ibrahim M. O., Usman I. O. (2012). A SEIV Vaccination Model with General Non-Linear Incidence Rate & Waning Preventive Vaccine. IOSR *Journal of Mathematics*. 51-59

Badshah, Porwal and Tiwani, (2013) Mathematical Modelling and Role of Dynamics in Epidemiology. *International Journal of Computational and Mathematics.* **5**(1), 1-10

Black A. J. and McKane A. J. (2010) Stochasticity in staged models of epidemics: quantifying the dynamics of whooping cough. Journal of the royal society interface. **7**, 1219-1227 [Online]. Accessed on 1st May, 2018. Available at: http://rsif.royalsocietypublishing.org/

Crowcroft, N. S. and Pebody, R. G. (2006) Recent Developments in Pertussis. Immunization development, health protection agency centre for infections, London, Uk. 3(67) 1926-36

Centre for Disease Control and Prevention, (2013) adopted recommendations of the advisory committee on immunization practices (ACIP). MMWR, 62(7). Pp131-135.

Fabricius G. et al. (2013) Modelling pertussis transmission to evaluate the effectiveness of an adolescent booster in Argentina. *Epidemiol. Infect.* **141,** 718-734.

Keeling M. J. and Danon L. (2009) Mathematical Modelling of infectious diseases. *British Medical Bulletin*, **92**, 33-42

Luz P. M., Codeco C. T., Werneck G. L. and Struchiner C. J. (2006) A modelling analysis of pertussis transmission and vaccination in Rio de Janeiro, Brazil. *Epidemiol. Infect.* **134,** 850-862.

Momoh A. A. *et al*. (2014) Stability Analysis of Mathematical Model of Hepatitis B. Current Research Journal of Biological Sciences **4**(5) p. 534-537 [online]. [Accessed 14 Jan, 2014]. Available at: http://www.maxwellsci.com/print/crjbs/v4-534-537.pdf

Munoz, F. M. *et al.* (2014) Safety and Immunization of Tetanus Diphtheria and Accellular Pertussis (Tdap) Immunization During Pregnancy in Mothers and Infants. A Randomized Clinical Trial. JAMA; 311(17). Pp 1760-1769.

Robbins J. B. et al. (2014) Toward a new vaccine for pertussis. PNAS, 111(9). Pp 3213-3216.

Shah N. H. and Gupta J. (2013) SEIR Model and Simulation for Vector Borne Diseases. *Journal of Applied Mathematics*. **4**, 13-17.

Stewart A. L. *et al.* (1989) Functional Status and well-being of patients with chronic conditions: results from the medical outcomes study. *Journal of the American Medical Association,* **262**, 907-913.

World Health Organisation (2008) Recommended standards for surveillance of selected vaccines-preventable diseases (www.who.int/vaccines-documents). Accessed Nov, 2017.