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Mathematical Modeling for Mitigating Pertussis Resurgence in the Post-COVID-19 Era: A Sensitivity Analysis and Intervention Strategies

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Abstract: **Pertussis (Whooping Cough) resurgence possess a formidable threat to public health systems, particularly due to disruptions in basic immunization programs induced by the recent COVID-19 Pandemic. Whooping cough predominantly affects infants and young children even in some developed countries with complete immunization programs. Therefore, reemergence of this disease stands as a double burden to developing countries with poor immunization scheme.**

In this Paper, we present a comprehensive five non-linear mathematical model to undersee the dynamics of pertussis transmission. The developed model puts into account the impact of vaccination during pregnancy (maternally derived immunity), vaccinated individuals, treatment of caregivers, adolescents and young adults who are usually disease reservoir (asymptomatic carriers. The stability of the equilibrium point was analyzed and the Basic reproduction number derived using the next generation matrix method. Furthermore, the normalized forward sensitivity analysis index was performed to pinpoint the most crucial parameter for targeted control and intervention strategies.

Numerical simulations were done using MATLAB 2021a to validate theoretical findings, relevant results are displayed.

Keywords: **Basic Reproduction Number, Pertussis, Resurgence, Sensitivity Analysis, Steady State, Treatment, Post-Covid-19, Vaccination.**

I. INTRODUCTION

Recently, Wales in the first few weeks of January, 2024 recorded rapid rise in Whooping cough cases with 135 notifications compared to 200 in the whole of 2023. Public Health Wales (PHW) expert said [1] *"With rates suppressed during the lockdowns of the pandemic we are naturally seeing a resurgence this year."* The consultant epidemiologist said "*whooping cough has waves of increased infection every three to four years".* Urging pregnant women, parents, children and all care givers to get vaccinated as quickly as possible.

Whooping cough (Pertussis) is an airborne respiratory infectious disease caused by the bacteria *Bordetella pertussis* or *B. Parapertussis*, which transmits bacteria through droplets from an infected person to a susceptible person and vaccine preventable [2]. Despite The vaccine wide coverage globally, the disease can be severe among adults because of long

immunity gap, major cause of death in infants, neonates and children less than 5 years old. Even though Vaccines against disease have significantly reduced mortality and morbidity for more than five decades globally [3]. According to World health Organization (WHO) estimations in 2013, whooping cough caused about 63,000 deaths in children less than 5 years old [4].

Recently, across the globe, the COVID-19 outbreak disrupted basic routine immunizations programs [1]. For instance, The WHO and UNICEF published large reductions in immunization coverage during COVID-19 pandemic, with approximately 25 million children missing out of lifesaving vaccines [5]. The agency recorded that the number of children who received zero dose vaccine of Diphtheria, Tetanus toxoids and Pertussis DTP [6] increased drastically from 13 to 18 million during COVID-19 pandemic era. The monumental decrease in vaccine coverage left a disproportionate number of children in developing countries most vulnerable to vaccine-preventable diseases which connotes that the risk of a huge outbreak is imminent with recent immunity gaps. Already, measles is reported in Africa and Eastern Mediterranean, wild polio virus 1 (WPV1) detected outside the endemic countries in Asia [7], Diphtheria outbreak in Nigeria and so on. [8] refereed to the opportunistic outbreak of diphtheria in developing countries like Nigeria as due to poor vaccination scheme during COVID-19 outbreak and therefore underscored the significance of vaccination and herd immunity. Pertussis disease usually thrives undetected, which implies that the reservoir or source of infection transmission to the susceptible infants is the adolescents and adults (who might be asymptotically infected) [9], and [10] attributed this instance to wanning immunity or the carrier is adapted to the pathogen (colonization). Pertussis in adults is underreported [11], the study focused on the comparative effect of pertussis cases underestimation in adults older than fifty years of age (50 years) in Brazil, Argentina, Mexico, Peru, Chile, of pertussis cases in adults aged ≥ 50 years in five Latin American countries. And concluded that pertussis in adult greater than fifty years old supersedes the once recorded by surveillance. Since the infants and neonates is more threatened by this deadly bacterium, [1,12] stressed the importance of vaccination during pregnancy which will help prevent fetus infection as well as passively transfer immunity against pertussis, tetanus toxoid from mother to infant for the first three 3 months of the new born.

Recently, there was an outbreak of pertussis in Jerusalem between January-July 2023, where about 257 cases was recorded with 1 fatality of an unvaccinated ten weeks (10) child whose mother was not vaccinated during pregnancy [13].

However, early diagnosis of whooping cough and treatment may reduce the spread of bacteria to others according to Centers for Disease Control and Prevention CDC [14]. To tackle pertussis disease, Mathematicians around the globe has formulated several models to this effect. For example, [15] considered the Analytical and Numerical study of whooping cough (pertussis) using the SEIR model and concluded that the implicit numerical integration scheme is best fit for studying pertussis epidemic. Also [16] considered pertussis resurgence despite huge vaccination interventions and formulated a model for the transmission behavior of pertussis with maternal derived immunity. The transmission dynamics of pertussis is influenced by vaccine wanning and natural booster of pertussis immunity, [17] developed a model SIVRWS (Susceptible-Infected-Vaccinated-Recovered-Waned-Susceptible) in a stationary homogeneous population setting. [18] proposed an SEIRQ model for diphtheria spread putting into consideration the exposed individual's natural immunity rate. [19,20] used the Normalized forward sensitivity index with regards to the giving parameter in the reproduction number to check compute parameters hierarchal influence on model.

Now, merging pertussis (whooping cough) reemergence possibility [1], poor vaccination programs in developing countries, and the global warning by the WHO [22] head cautioning that post-covid-19 effect still pose a deadlier threat on global health. We therefore propose a five (5) non-linear compartmental model to tackle resurgence of whooping cough in the next section.

II. PROPOSED MATHEMATICAL MODEL FORMULATION

In this study, a mathematical model of the transmission dynamics of pertussis disease is formulated, analyzed and modified to gain informed control strategy in low vaccine coverage settings through forward sensitivity index.

To undersee the spread of Pertussis disease in an erratic vaccine environment, we subdivide the total number of population N into five (5) groups, namely the Susceptible (S), Exposed (E), Infected (I), Treatment (T) and Recovered (R) respectively. That is

$$N(t) = S(t) + E(t) + I(t) + T(t) + R(t) \quad (1)$$

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The Susceptible group increases by the proportion $(1-\nu)bN$ and ϕ E exposed individual with strong immunity against pertussis, who are not vulnerable to disease even after interaction with an infected individual due to maternal, vaccine or other local immunity. The Susceptible group decreases by the infection force $\frac{\beta SI}{N}$ after interaction with the infected individual at rate β and natural death rate μ.

$$\frac{dS}{dt} = (1-\nu)bN - \frac{\beta SI}{N} + \phi E - \mu S \tag{2}$$

The exposed group gains $\frac{\beta SI}{N}$, decreases by the transmission rate α, proportion of individuals with strong immune ϕ and natural death μ.

$$\frac{dE}{dt} = \frac{\beta SI}{N} - (\alpha + \phi + \mu)E \tag{3}$$

The infected group increases by individuals who is already manifesting the clinical symptoms of pertussis in exposed class i.e., after incubation period. Also, the infected class decrease with natural death μ, pertussis induced death ω and treated individuals who yield self η.

$$\frac{dI}{dt} = \alpha E - (\eta + \omega + \mu) I \tag{4}$$

Treatment class increases by a portion of infected individuals who yield self for treatment, also decreases by individuals who have completed medications and are been discharged or decreases by natural death μ.

$$\frac{dT}{dt} = \eta I - (\Omega + \mu) T \tag{5}$$

The recovered class also increase by the proportion νbN of the total population and the number of discharged individuals after treatment and reduces by natural death rate μ.

$$\frac{dR}{dt} = \nu bN + \Omega T - \mu R \tag{6}$$

The Equations (1)-(6) of pertussis transmission dynamics is summarized by the non-linear differential equations below.

$$\begin{aligned}
\frac{dS}{dt} &= (1-\nu)bN - \frac{\beta SI}{N} + \phi E - \mu S \\
\frac{dE}{dt} &= \frac{\beta SI}{N} - (\alpha + \phi + \mu)E \\
\frac{dI}{dt} &= \alpha E - (\eta + \omega + \mu) I \\
\frac{dT}{dt} &= \eta I - (\Omega + \mu) T \\
\frac{dR}{dt} &= \nu bN + \Omega T - \mu R
\end{aligned} \tag{7}$$

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The diagram below shows transmission dynamics of pertussis disease,

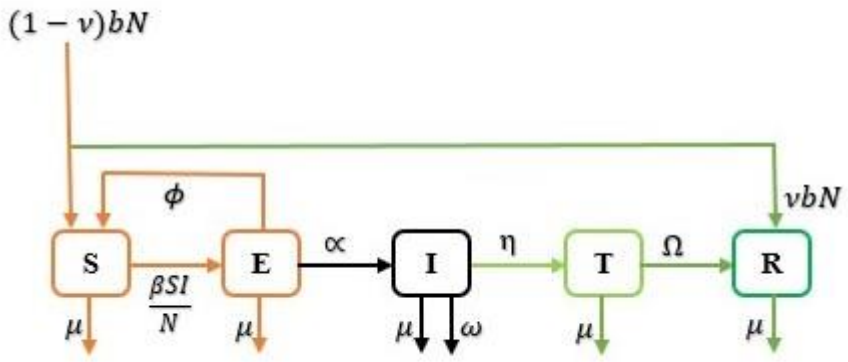

Fig. 1. Schematic Diagram of Pertussis Model

Table I below shows the models parameter and description.

Table I

S/N	Parameter	Description
1	b	Birth rate
2	β	Susceptible and infected interaction rate
3	α	Transmission rate
4	ω	Pertussis induced death rate
5	v	Vaccinated Individual
6	η	Treatment rate
7	Ω	Recovery/cure rate
8	μ	Natural death rate
9	ϕ	Exposed individual with perfect natural immune

III. STEADY STATES

The rate of change of the total population in equation (7) is studied from the stability equilibrium points to obtain two steady states which is

$$\frac{dN}{dt}=\frac{dS}{dt}+\frac{dE}{dt}+\frac{dI}{dt}+\frac{dT}{dt}+\frac{dR}{dt} \tag{8}$$

from Equation (8), adding the equation of the model at t = 0 gives, has two steady states, the first steady state is

$$\frac{dN}{dt}=(1-v)bN-\mu S-\mu E-(\omega+\mu)I-\mu T-\mu R \tag{9}$$

substituting equation (1) into (9)

$$\frac{dN}{dt}=(1-v)bN-\mu(N)-I_S\omega \tag{10}$$

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Initially when no disease spread,it follows that

$$\frac{dN}{dt} = (1-\nu)bN - \mu(N) \tag{11}$$

And since,

$$0 \le (1-\nu)bN - \mu(N)$$
$$\frac{(1-\nu)bN}{\mu} \le N(t) \tag{12}$$

$$\frac{dN}{dt} = -\mu S - E\mu - (\omega+\mu)I - T\mu + \nu bN - \mu R$$

$$\frac{dN}{dt} = \nu bN - \mu(N)$$
$$N(t) = \frac{\nu bN}{\mu}$$

The first steady state that is the disease-free equilibrium (DFE), which is positive and feasible.

$$\mathrm{N}_0 = (S_0, \mathrm{E}_0, I_0, T_0, R_0) = \left(\frac{bN(1-V)}{\mu}, 0, 0, 0, 0, \frac{\nu bN}{\mu}\right) \tag{13}$$

The second steady state is the Endemic Equilibrium Point (EEP), Denoted as,

$$N^* = (S^*, \mathrm{E}^*, I^*, T^*, R^*) \tag{14}$$

Where,

$$S^* = \frac{(1-\nu)b + \phi E^*}{\beta I^* + \mu}, E^* = \frac{\beta S^* I^*}{(\alpha+\phi+\mu)}, I^* = \frac{\alpha E^*}{(\eta+\omega+\mu)}, T^* = \frac{\eta I^*}{(\Omega+\mu)}, R^* = \frac{\nu b + \Omega T^*}{\mu}$$

Other endemic points will be shown in scenarios.

IV. BASIC REPRODUCTION NUMBER

The Basic reproduction number indicates the number of secondary infections an infected individual can produce in a susceptible or vulnerable population. To obtain the basic reproduction number, we take the derivatives of the affected classes in equations (3) and (4)

$$\frac{dE}{dt} = \frac{\beta SI}{N} - (\alpha+\phi+\mu)E$$
$$\frac{dI}{dt} = \alpha E - (\eta+\omega+\mu)I \tag{15}$$

Let $a = (E, I)$ then

$$\frac{da}{dt} = \mathrm{F}_i(a) - \nu_i(a) \tag{16}$$

where $\mathrm{F}_i(a)$ denotes the rate of secondary infection and $v_i(a)$ represents disease transmission rate. given by

$$\mathrm{F}_i(a)=\begin{bmatrix}\frac{\beta SI}{N}\\0\end{bmatrix}, v_i(a)=\begin{bmatrix}(\alpha+\phi+\mu)E\\-\alpha E+(\eta+\omega+\mu)I\end{bmatrix}$$

Taking the Jacobian at disease free equilibrium we employ

$$\mathrm{F}_i=\begin{bmatrix}\frac{\partial \mathrm{F}_1}{\partial E} & \frac{\partial \mathrm{F}_1}{\partial I}\\ \frac{\partial \mathrm{F}_2}{\partial E} & \frac{\partial \mathrm{F}_2}{\partial I}\end{bmatrix}, v_i=\begin{bmatrix}\frac{\partial v_1}{\partial E} & \frac{\partial v_1}{\partial I}\\ \frac{\partial v_2}{\partial E} & \frac{\partial v_2}{\partial I}\end{bmatrix}$$

Now let

$$\mathrm{DF}_i(a)=F=\begin{bmatrix}0 & \beta S\\0 & 0\end{bmatrix} \text{ and } \mathrm{D}v_i(a)=V=\begin{bmatrix}\alpha+\phi+\mu & 0\\-\alpha & \eta+\omega+\mu\end{bmatrix}$$

Using the next generation matrix, FV^{-1} we obtain

$$V^{-1}=\begin{bmatrix}\alpha+\phi+\mu & 0\\-\alpha & \eta+\omega+\mu\end{bmatrix}^{-1}=\frac{1}{(\alpha+\phi+\mu)(\eta+\omega+\mu)}\begin{bmatrix}\eta+\omega+\mu & 0\\\alpha & \alpha+\phi+\mu\end{bmatrix}$$

$$V^{-1}=\begin{bmatrix}\frac{1}{(\alpha+\phi+\mu)} & 0\\ \frac{\alpha}{(\alpha+\phi+\mu)(\eta+\omega+\mu)} & \frac{1}{(\eta+\omega+\mu)}\end{bmatrix} \quad (17)$$

Now, FV^{-1} becomes,

$$FV^{-1}=\begin{bmatrix}0 & \beta S\\0 & 0\end{bmatrix}\times\begin{bmatrix}\frac{1}{(\alpha+\phi+\mu)} & 0\\ \frac{\alpha}{(\alpha+\phi+\mu)(\eta+\omega+\mu)} & \frac{1}{(\eta+\omega+\mu)}\end{bmatrix} \quad (18)$$

Since the Reproduction number is the largest and first eigen value, then

$$FV^{-1}=\begin{bmatrix}\frac{\beta S\alpha}{(\alpha+\phi+\mu)(\eta+\omega+\mu)} & \frac{\beta S}{(\eta+\omega+\mu)}\\0 & 0\end{bmatrix}$$

Alternatively, the eigenvalues λ of FV^{-1} can be computed from the characteristic equation.

$$|FV^{-1}-\lambda|=0 \quad (19)$$

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$$FV^{-1} = \begin{bmatrix} \dfrac{\beta S\alpha}{(\alpha+\phi+\mu)(\eta+\omega+\mu)} - \lambda & \dfrac{\beta S}{(\eta+\omega+\mu)} \\ 0 & 0-\lambda \end{bmatrix}$$

Therefore,

$$R_0 = \frac{\beta S\alpha}{(\alpha+\phi+\mu)(\eta+\omega+\mu)}$$

$$R_0 = \frac{\beta b(1-\nu)\alpha}{\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)} \quad \text{Where, } S = \frac{b(1-\nu)}{\mu} \tag{20}$$

V. LOCAL STABILITY ANALYSIS OF DFE AND EEP

Theorem 1: The diseases free equilibrium is locally asymptotic stable if $R_0 < 1$ and unstable if $R_0 > 1$,

Proof:

The Jacobian matrix J_{dfe} of the system evaluated at the disease-free equilibrium is

$$J_{dfe} = \begin{bmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial T} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial T} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial T} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial T} & \frac{\partial f_4}{\partial R} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial E} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial T} & \frac{\partial f_5}{\partial R} \end{bmatrix} = \begin{bmatrix} -\mu & \phi & -\dfrac{\beta(1-\nu)}{\mu} & 0 & 0 \\ 0 & -(\alpha+\phi+\mu) & \dfrac{\beta(1-\nu)}{\mu} & 0 & 0 \\ 0 & \alpha & -(\eta+\omega+\mu) & 0 & 0 \\ 0 & 0 & \eta & -(\Omega+\mu) & 0 \\ 0 & 0 & 0 & \Omega & -\mu \end{bmatrix} \tag{21}$$

Where,

$$f_1 = \frac{dS}{dt}, f_2 = \frac{dE}{dt}, f_3 = \frac{dI}{dt}, f_4 = \frac{dT}{dt}, f_5 = \frac{dR}{dt} \text{ and } x_1 = -(\alpha+\phi+\mu), x_2 = -(\eta+\omega+\mu), x_3 = -(\Omega+\mu)$$

Finding the eigenvalues at the J_{dfe}, Then characteristic equation $|J_{dfe} - \lambda I| = 0$ is expanded and simplified as follows:

$$J_{dfe} = \begin{bmatrix} -\mu-\lambda_1 & \phi & -\dfrac{\beta(1-\nu)b}{\mu} & 0 & 0 \\ 0 & -x_1-\lambda_2 & \dfrac{\beta(1-\nu)b}{\mu} & 0 & 0 \\ 0 & \alpha & -x_2-\lambda_3 & 0 & 0 \\ 0 & 0 & \eta & -x_3-\lambda_4 & 0 \\ 0 & 0 & 0 & \Omega & -\mu-\lambda_5 \end{bmatrix} \tag{22}$$

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From the Jacobian matrix of (22), we obtained a characteristic polynomial:

$$(-\mu-\lambda_1)(-x_1-\lambda_2)(-x_2-\lambda_3)(-x_3-\lambda_4)(-\mu-\lambda_5)=0$$

Thus, from equation (22) it is obvious that the eigenvalues

$$\lambda_1=-\mu,\lambda_2=-x_1,\lambda_3=-x_2,\lambda_4=-x_3,\lambda_5=-\mu, \tag{23}$$

$\lambda_1,\lambda_2,\lambda_3,\lambda_4,\lambda_5$ and λ_6 are all negative quantities.

Since we know that the first, fourth and fifth columns gives the first three (3) eigenvalues which are $\lambda_4=-(\Omega+\mu)$ and $\lambda_1=\lambda_5=-\mu$ (repeated roots).

The rest is obtained by taking the determinant of the (2 × 2) sub matrix formed by excluding the first, fourth and fifth rows and columns of system (22) we get.

$$|J_{dfe}-\lambda I|=\begin{bmatrix}-(\alpha+\phi+\mu)-\lambda & \dfrac{\beta(1-\nu)b}{\mu}\\ \alpha & -(\eta+\omega+\mu)-\lambda\end{bmatrix}=0 \tag{24}$$

$$=-(-(\alpha+\phi+\mu)-\lambda)(-(\eta+\omega+\mu)-\lambda)+\frac{\alpha\beta(1-\nu)b}{\mu}=0$$

$$=(\mu+\lambda)(\Omega+\mu+\lambda)(\mu+\lambda)[-\lambda^2-(\alpha+\phi+\eta+\omega+2\mu)\lambda-(\alpha+\phi+\mu)(\eta+\omega+\mu)+\frac{\alpha\beta(1-\nu)b}{\mu}]=0$$

$$R_0(\alpha+\phi+\mu)(\eta+\omega+\mu)=\frac{\beta\alpha(1-\nu)b}{\mu}$$

$$-\lambda^2-(\alpha+\phi+\eta+\omega+2\mu)\lambda-(\alpha+\phi+\mu)(\eta+\omega+\mu)-R_0(\alpha+\phi+\mu)(\eta+\omega+\mu)=0$$

$$\frac{1}{(\alpha+\phi+\mu)(\eta+\omega+\mu)}\lambda^2+\frac{(\alpha+\phi+\eta+\omega+2\mu)}{(\alpha+\phi+\mu)(\eta+\omega+\mu)}\lambda+1-R_0=0$$

It is clear from equation (23) that λ_2 and λ_3 are negative, Since $1-R_0>0$ .it infers that the disease free equilibrium is asymptotic stable if $R_0<0$ and unstable if $R_0>0$.

VI. SENSITIVITY ANALYSIS

In this research work, Sensitivity analysis is explored by changing the parameter values of the reproduction number R_0 either one at a time or simultaneously, to observe its impact on the the model's predictions. Such tangible parameters with huge impact on the model can help in making informed decisions for researchers and policy makers for disease control strategy.

Therefore, the sensitivity index of R_0 differentiable with respect to parameter β can be is defined as

$$\Upsilon_{\beta}^{R_0} = \frac{\beta}{R_0}\frac{\partial R_0}{\partial \beta}$$

$$R_0 = \frac{\beta b(1-\nu)\alpha}{\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)}$$

$$\frac{\partial R_0}{\partial \beta} = \frac{\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)b(1-\nu)\alpha}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2} = \frac{b(1-\nu)\alpha}{\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)}$$

$$\Upsilon_{\beta}^{R_0} = \frac{\beta}{R_0}.\frac{b(1-\nu)\alpha}{\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)}$$

Sensitivity index for b (birth rate) is derived as: $\Upsilon_{b}^{R_0} = \frac{b}{R_0}\frac{\partial R_0}{\partial b}$

$$\frac{\partial R_0}{\partial b} = \frac{\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)\beta(1-\nu)\alpha}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2} = \frac{\beta(1-\nu)\alpha}{\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)}$$

$$\Upsilon_{b}^{R_0} = \frac{b}{R_0}.\frac{\beta(1-\nu)\alpha}{\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)}$$

Sensitivity index for ν (vaccination rate) is derived as: $\Upsilon_{\nu}^{R_0} = \frac{\nu}{R_0}\frac{\partial R_0}{\partial \nu}$

$$\frac{\partial R_0}{\partial \nu} = -\frac{\beta b\alpha\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2} = -\frac{\beta b\alpha}{\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)}$$

$$\Upsilon_{\nu}^{R_0} = -\frac{\nu}{R_0}.\frac{\beta b\alpha}{\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)}$$

Sensitivity index for $\boldsymbol{\mu}$ (natural death rate) is derived as: $\Upsilon_{\mu}^{R_0} = \frac{\mu}{R_0}\frac{\partial R_0}{\partial \mu}$

$$\frac{\partial R_0}{\partial \mu} = -\frac{\beta b(1-\nu)\alpha(\alpha\eta+\alpha\omega+2\mu\alpha+\phi\eta+\phi\omega+2\mu\phi+2\mu\eta+2\mu\omega+3\mu^2)}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2}$$

$$\Upsilon_{\mu}^{R_0} = -\frac{\mu}{R_0}.\frac{\beta b(1-\nu)\alpha(\alpha\eta+\alpha\omega+2\mu\alpha+\phi\eta+\phi\omega+2\mu\phi+2\mu\eta+2\mu\omega+3\mu^2)}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2}$$

Sensitivity index for α (transmission rate) is derived as: $\Upsilon_{\alpha}^{R_0} = \frac{\alpha}{R_0}\frac{\partial R_0}{\partial \alpha}$

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$$\frac{\partial R_0}{\partial \alpha} = \frac{\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)\beta b(1-\nu) - \beta b(1-\nu)\alpha(\mu\eta+\mu\omega+\mu^2)}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2}$$

$$\Upsilon_{\alpha}^{R_0} = \frac{\alpha}{R_0}.\frac{\mu(\alpha+\phi+\mu)(\eta+\omega+\mu)\beta b(1-\nu) - \beta b(1-\nu)\alpha(\mu\eta+\mu\omega+\mu^2)}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2}$$

Sensitivity index for ϕ (strong immunity of the exposed individuals) is derived as: $\Upsilon_{\phi}^{R_0} = \frac{\phi}{R_0}\frac{\partial R_0}{\partial \phi}$

$$\frac{\partial R_0}{\partial \phi} = -\frac{\beta b(1-\nu)\alpha(\mu\eta+\mu+\mu^2)}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2}$$

$$\Upsilon_{\phi}^{R_0} = -\frac{\phi}{R_0}.\frac{\beta b(1-\nu)\alpha(\mu\eta+\mu+\mu^2)}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2}$$

Sensitivity index for η (treatment rate) is derived as: $\Upsilon_{\eta}^{R_0} = \frac{\eta}{R_0}\frac{\partial R_0}{\partial \eta}$

$$\frac{\partial R_0}{\partial \eta} = -\frac{\beta b(1-\nu)\alpha(\mu\alpha+\mu\phi+\mu^2)}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2}$$

$$\Upsilon_{\eta}^{R_0} = -\frac{\eta}{R_0}.\frac{\beta b(1-\nu)\alpha(\mu\alpha+\mu\phi+\mu^2)}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2}$$

Sensitivity index for ω (pertussis induced death rate) is derived as: $\Upsilon_{\omega}^{R_0} = \frac{\omega}{R_0}\frac{\partial R_0}{\partial \omega}$

$$\frac{\partial R_0}{\partial \omega} = -\frac{\beta b(1-\nu)\alpha(\mu\alpha+\mu\phi+\mu^2)}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2}$$

$$\Upsilon_{\omega}^{R_0} = -\frac{\omega}{R_0}.\frac{\beta b(1-\nu)\alpha(\mu\alpha+\mu\phi+\mu^2)}{(\mu(\alpha+\phi+\mu)(\eta+\omega+\mu))^2}$$

Inputting numeric values of Table 2 into the forward sensitivity index derived above, gives

$$\Upsilon_{\beta}^{R_0} = \frac{\beta}{R_0}\frac{\partial R_0}{\partial \beta} = 1, \qquad \Upsilon_{b}^{R_0} = \frac{b}{R_0}\frac{\partial R_0}{\partial b} = 1, \qquad \Upsilon_{\nu}^{R_0} = \frac{\nu}{R_0}\frac{\partial R_0}{\partial \nu} = -1.5,$$

$$\Upsilon_{\mu}^{R_0} = \frac{\mu}{R_0}\frac{\partial R_0}{\partial \mu} = -0.00612721, \qquad \Upsilon_{\alpha}^{R_0} = \frac{\alpha}{R_0}\frac{\partial R_0}{\partial \alpha} = -0.763157, \qquad \Upsilon_{\eta}^{R_0} = \frac{\eta}{R_0}\frac{\partial R_0}{\partial \eta} = -0.914634,$$

$$\Upsilon_{\phi}^{R_0} = \frac{\phi}{R_0}\frac{\partial R_0}{\partial \phi} = -0.486714, \qquad \Upsilon_{\omega}^{R_0} = \frac{\omega}{R_0}\frac{\partial R_0}{\partial \omega} = -0.0762195$$

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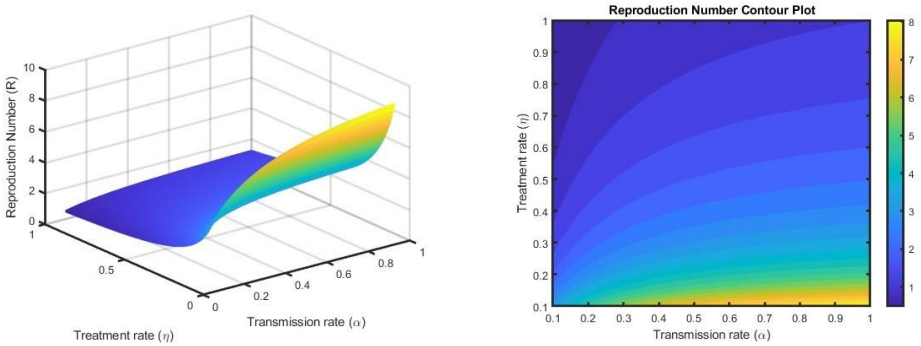
VII. NUMERICAL SIMULATIONS

In this section, Numerical solution is performed and Simulation conducted using MATLAB 2021a software. The initial conditions used in simulating are S (0) = 9960, E (0) = 40, I (0) = 1, T (0) = 0, R (0) = 0 and N (0) = 10001 while the parameter values used is shown in table II below. Now we will experiment in scenarios to elaborate the dynamics and sensitivity of the pertussis system. Some of the parameters used in this work are taking from existing researchers and others fitted.

Table II

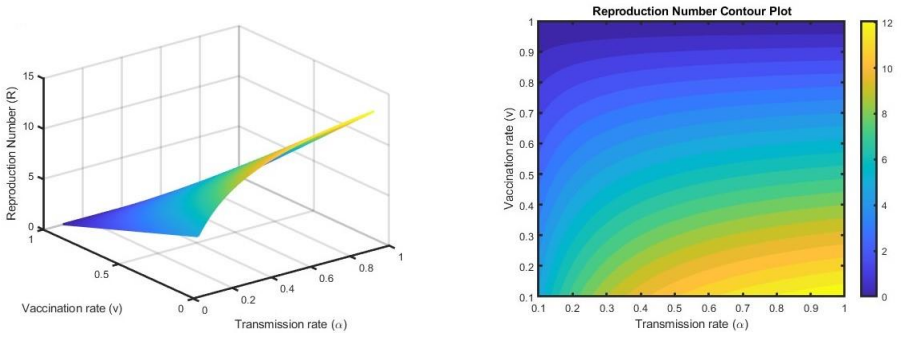
S/N	Parameter	Description	value	Cite
1	b	Birth rate	0.019	[18]
2	β	Susceptible and infected interaction rate	0.55	[18]
3	α	Transmission rate	0.8	[21]
4	ω	Pertussis induced death rate	0.055	Fitted
5	v	Vaccinated Individual	0.6	Fitted
6	η	Treatment rate	0.6	Fitted
7	Ω	Recovery/cure rate	0.5	[18]
8	μ	Natural death rate	0.006	[18]
9	ϕ	Exposed individual with perfect natural immune	0.2	Fitted

The reproduction number is a major parameter representing disease spread potentials, Fig. 2. Shows a 3D plot created for α and η which helps us calculate the reproduction number at each grid point. The visuals show how the reproduction number varies with respect to changes in transmission as a result of change in treatment, vaccination and exposed individuals with strong immunity rates. Same applies to Fig. 3 and Fig. 4 respectively.



(a) **(b)**

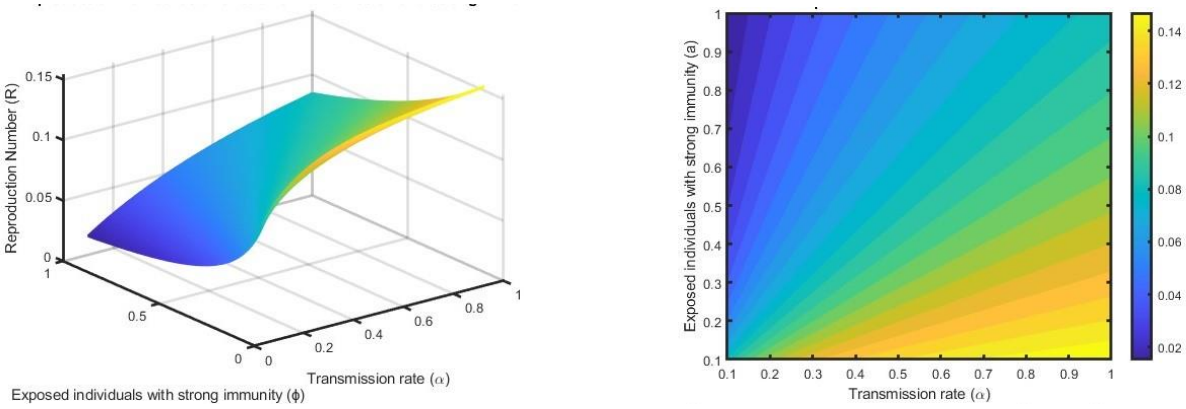
Fig. 2. The subplot (a) illustrates the impact of α (The disease transmission rate) and η (treatment) on R_0 (reproduction number), where (b) shows the respective contour plot rate.



(c) **(d)**

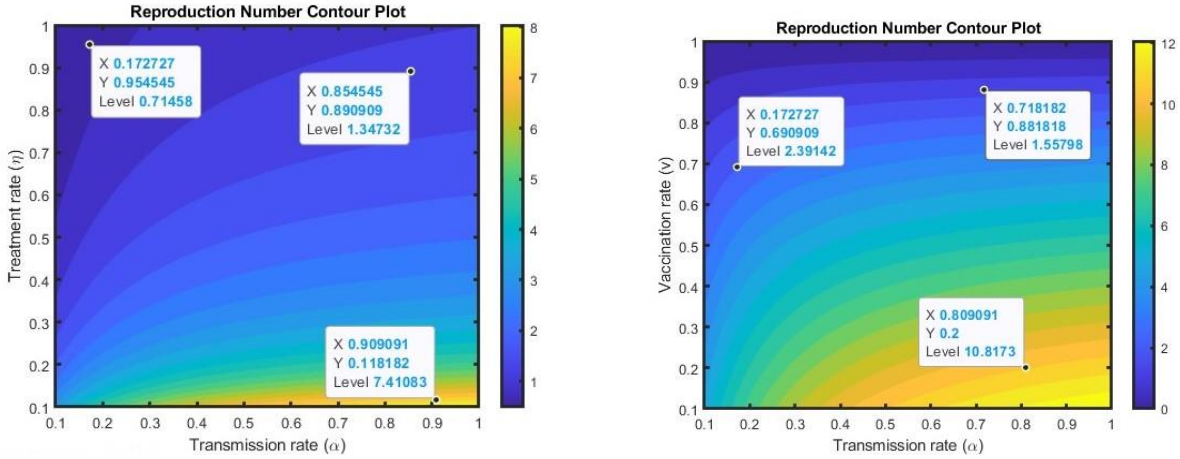
Fig. 3. The subplot (c) illustrates the impact of α (the disease transmission rate) and ν (vaccination) R_0

(reproduction number), where (d) shows the respective contour plot rate.

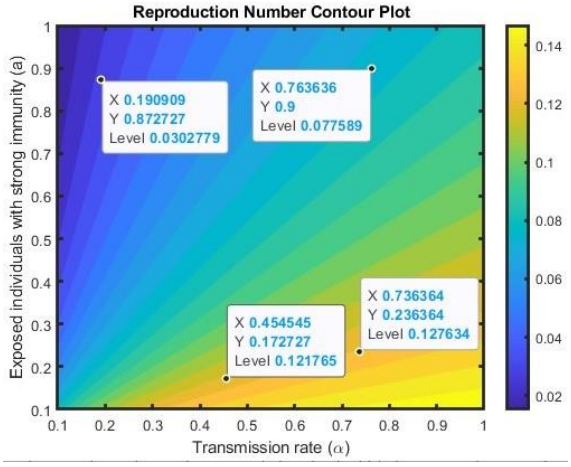


(e) **(f)**

Fig. 4. The subplot (e) illustrates the impact of α (the disease transmission rate) and ϕ (Exposed individual with perfect natural immune) R_0 (reproduction number), where (f) shows the respective contour plot rate.



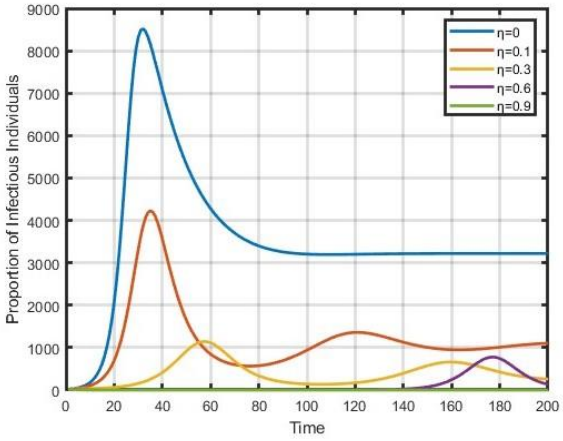
(i) **(ii)**



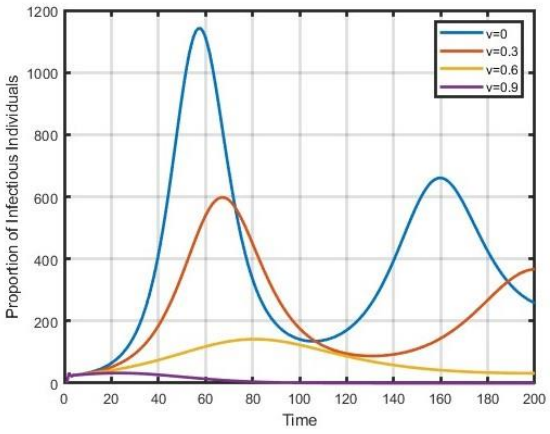
(iii)

Fig. 5. The subplot (i)-(iii) illustrates the respective contour plot impact value of the transmission rate, treatment rate, vaccination rate and exposed individual with strong immunity on the reproduction number.

α (The disease transmission rate) and ϕ (Exposed individual with perfect natural immune) R_0 (reproduction number), where (f) shows the respective contour plot rate.

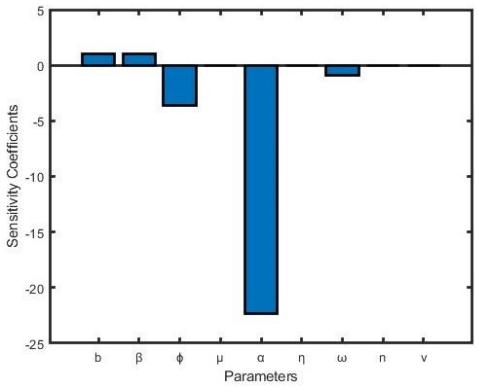


(g)

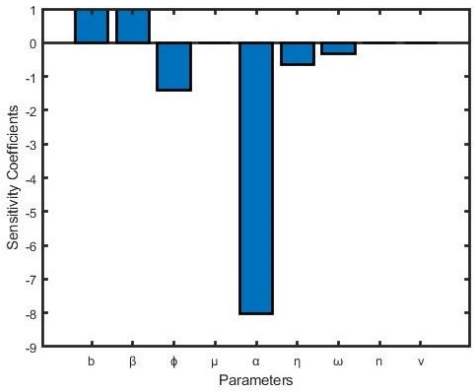


(h)

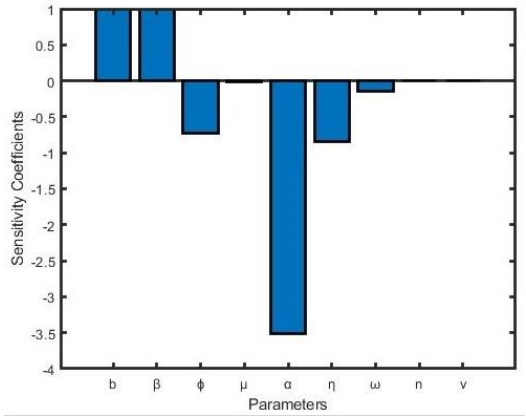
Fig. 6. (g) illustrates the sensitivity impact of increase in $\eta = 0, 0.1, 0.3, 0.6, 0.9$ (different levels of treatment rate) on the proportion of infected or infectious individuals, where (h) sensitivity impact of increase in $v = 0, 0.1, 0.3, 0.6, 0.9$ (different levels of vaccination rate).



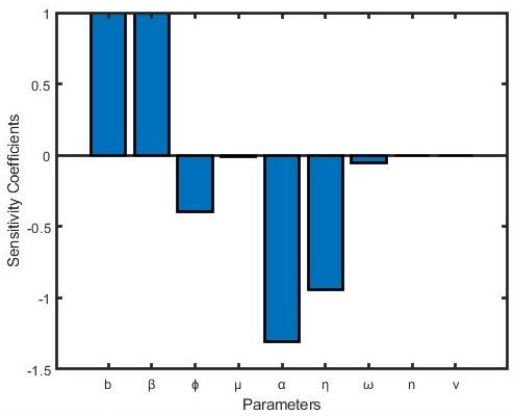
(i) $\eta = 0$.



(j) $\eta = 0.1$



(k) $\eta = 0.3$.



(l) $\eta = 0.9$

Fig. 7. The bar chats subplot (i), (j), (k) and (l) illustrates the Sensitivity coefficients of parameter η (different levels of treatment rate) on other parameters in R_0 (reproduction number).

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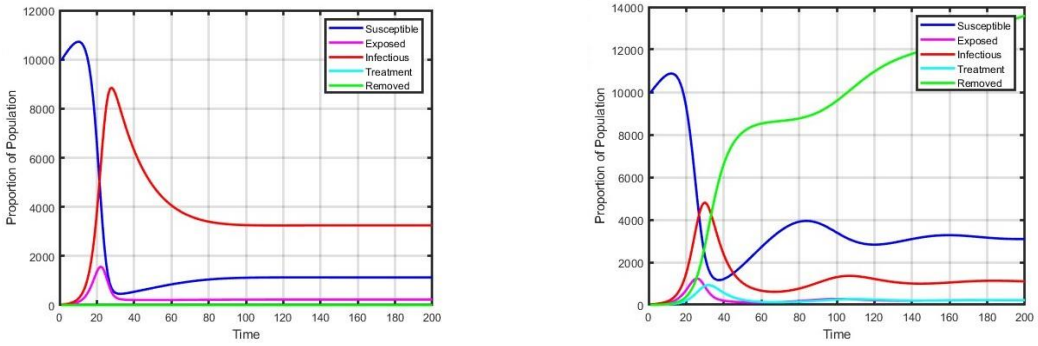
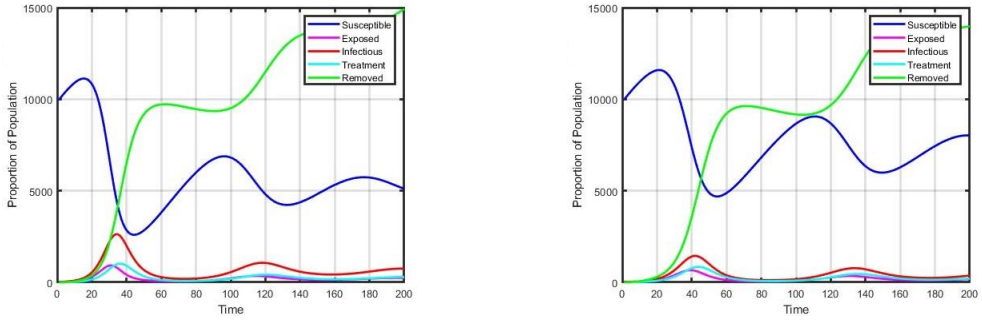
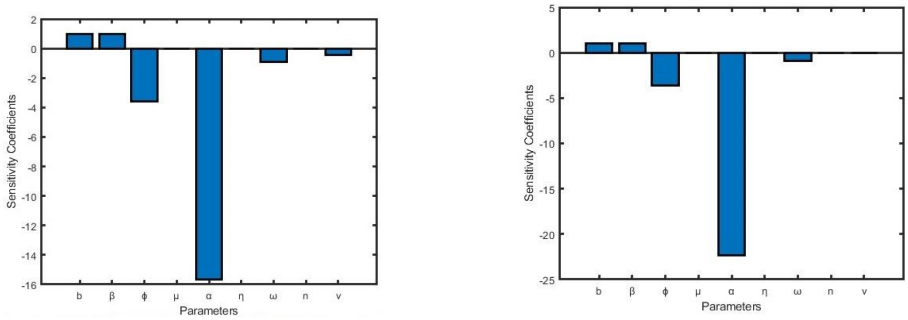


(m) $\eta = 0$ **(n) $\eta = 0.1$**

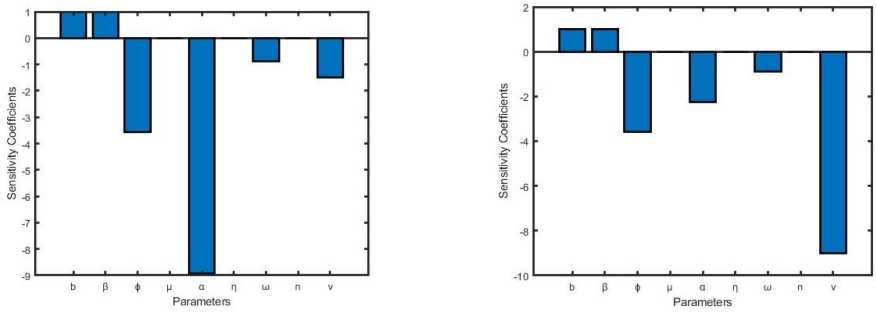


(o) $\eta = 0.2$ **(p) $\eta = 0.3$**

Fig. 8. The subplots (m), (n), (o) and (p) illustrates the impact of parameter η (different levels of treatment rate) on the total population respectively.



(q). $\nu = 0$ **(r). $\nu = 0.3$**



(s). $\nu = 0.6$ **(t). $\nu = 0.9$**

Fig. 9. The bar chats subplot (q), (r), (s) and (t) illustrates the Sensitivity coefficients of parameter ν (different levels of vaccination rate) on other parameters in R_0 (reproduction number).

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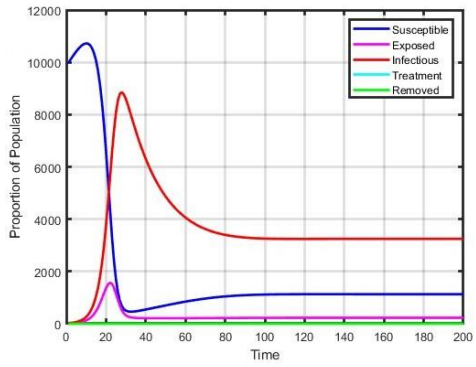
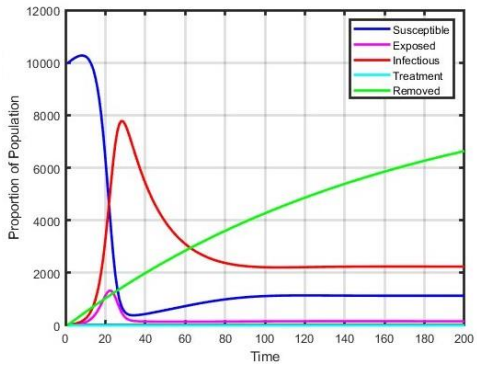
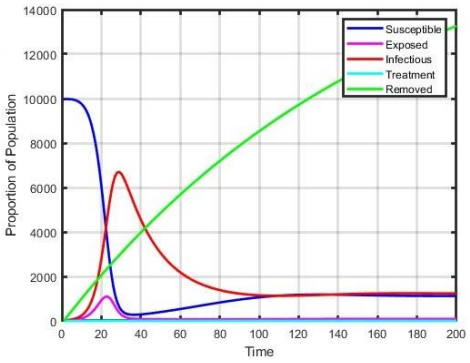


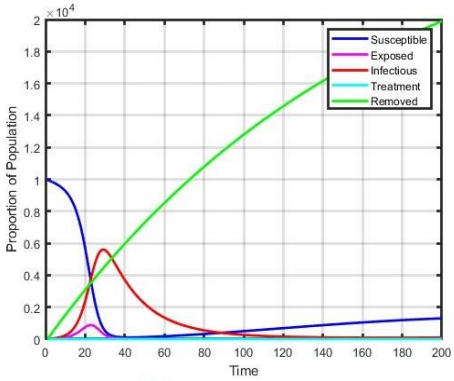
(u). $\nu = 0$



(v). $\nu = 0.3$

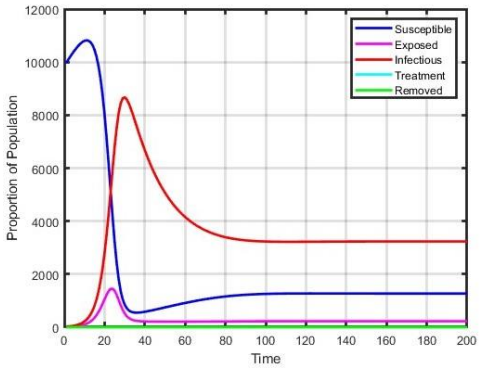


(w). $\nu = 0.6$

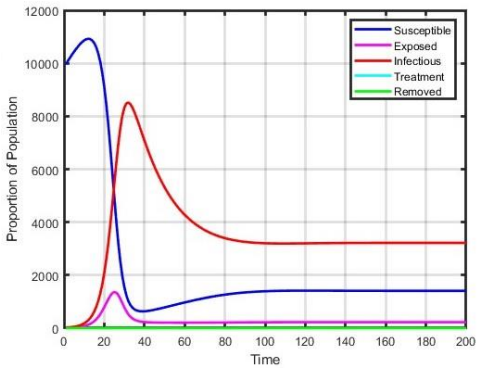


(x). $\nu = 0.9$

Fig. 10. The subplots (u), (v), (w) and (x) illustrates the impact of parameter ν (different levels of vaccination rate) on the total population respectively.



(y). $\phi = 0.1$



(z). $\phi = 0.2$

Fig. 11. The subplots (y) and (z) illustrates the impact of parameter ϕ (different levels of perfect immunity rate) on the total population respectively.

VIII. CONCLUSION

In this study, a mathematical model for transmission dynamics of pertussis (whooping cough) is developed. The basic reproduction number R_0 was derived and the forward sensitivity analysis on R_0 was carried out to determine the impact of each parameter in disease threshold. It was observed that without disease intervention strategy $R_0 = 28$ that is $v = \eta = \alpha = 0$. Furthermore, results shows that increase in intervention parameter, reduced R_0 to the range (15-17) for pertussis [23] as suggested by European Center for Disease prevention and control (2014).

For instance, Table III bellow illustrates in summary impact of vaccination during pregnancy (maternally derived immunity), vaccination and treatment.

Table III

Parameter / **Value**	**v**	η	α
0	$R_0 = 28$	$R_0 = 28$	$R_0 = 28$
0.3	$R_1 = 20$	$R_1 = 4$	$R_1 = 20$
0.6	$R_2 = 11$	$R_2 = 2$	$R_2 = 16$
0.9	$R_3 = 3$	$R_3 = 1.6$	$R_3 = 13$
1.2	$R_4 = 0$	$R_4 = 1.2$	$R_4 = 11$

We will summarize in scenarios,

Scenario 1, from Table III, results shows that vaccination (parameter) of individuals is the most effective disease control strategy on the basic reproduction number. From Table III when ($v = 1.2$, $R_4 = 0$), when treatment ($\eta = 1.2$, $R_4 = 1.2$) and when maternally derived immunity ($\alpha = 1.2$, $R_4 = 11$) respectively. Which infers that the adolescents and young adults serve as asymptomatic source of disease spread (reservoir).

Scenario 2, The parameter in R_0 was manually computed using Table II, arranging the magnitude of each parameter sensitivity in descending order yields. $v = -1.5$, $\beta = b = 1$, $\eta = -0.914634$, $\alpha = -0.763157$, $\phi = -0.486714$, $\omega = -0.0762195$, $\boldsymbol{\mu} = -0.00612721$, considering the magnitude and direction of each parameter in the sensitivity index, Results shows that increasing the parameter values of. $v = -1.5$, $\eta = -0.914634$, $\alpha = -0.763157$, $\phi = -0.486714$, $\omega = -0.0762195$, $\boldsymbol{\mu} = -0.00612721$, and reducing the parameter values of $\beta = b = 1$ will significantly reduce the basic reproduction number of pertussis disease spread. Therefore, we can pinpoint $v = -1.5$, $\eta = -0.914634$, $\phi = -0.486714$ as the most tangible and influential parameters of R_0.

Furthermore, Fig. 6. illustrates the sensitivity impact of treatments and vaccination on the proportion of infected or infectious individuals by increasing the parameter $\eta = 0, 0.1, 0.3, 0.6, 0.9$ and $v = 0, 0.1, 0.3, 0.6, 0.9$. Results shows that vaccination is a better intervention strategy for pertussis as Treatment shows disease reemergence possibility.

Scenario 3, with the Exposed individual with perfect natural immune = 0.2, Fig. 7. And Fig. 8 Shows the bar chat sensitivity coefficients of R_0 by gradually increasing the treatment and vaccination parameter respectively. The corresponding results of each parameter are displayed in bars, increase in treatment and vaccination parameters significantly reduced the disease transmission rate. It was observed here that treatment of disease was more impactful than vaccination rate.

Scenario 4, Finally, the impact of Vaccination and treatment compartment was examined in the developed SEITR Model. Fig. 8 and Fig. 10 shows the behavior of the system with initial condition of S (0) = 9960, E (0) = 40, I (0) = 1, T (0) = 0, R (0) = 0 and N (0) = 10001. It was observed that Increase in treatment and vaccination proportion drastically reduced all other compartment with increase in the recovery rate. The fig. 10. for vaccination comparatively shows a smoother graph than fig. 8. Which shows that vaccination is a very reliable control strategy than treatment. On the other hand, in fig. 11 which is the impact of maternally derived immunity was not too significant in pertussis disease control, however, the exposed class shows noticeably reduction in its compartment.

Based on graphical experiments performed on the system, arranging the impact of hierarchical intervention strategy is vaccination, treatment and maternal derived immunity via (vaccinating pregnant women). In summary, the combination of the three intervention strategies will lower whooping cough reproduction number, increase recovery rate and completely eradicate infection if well implemented by policy makers.

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