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Mathematical Model of Pertussis and Pneumonia Co-Infection in Infants with Maternally Derived Immunity

(Model Matematik bagi Jangkitan Bersama Batuk Kokol dan Pneumonia pada Bayi dengan Imuniti daripada Ibu)

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ABSTRACT

The transmission dynamics of a pertussis-pneumonia co-infection model is analyzed. The model takes into account temporary immunity of infected infants and includes a maternally derived immunity compartment. The basic reproduction number of the co-infected model is obtained using the next generation matrix, and stability analysis is carried out. The model exhibits four equilibria, namely, the pertussis-free equilibrium, the pneumonia-free equilibrium, the co-infection-free equilibrium and co-infection endemic equilibrium. Subsequently, the local stability of the co-infection-free equilibrium is analyzed and is shown to be locally asymptotically stable. Similarly, by constructing a suitable Lyapunov function, the co-infection endemic equilibrium is shown to be globally asymptotically stable. Numerical simulations are carried out to illustrate the validity of these results.

Keywords: Childhood respiratory diseases; common quadratic Lyapunov function; global stability; maternally derived immunity; pertussis-pneumonia co-infection

ABSTRAK

Analisis dinamik penularan model jangkitan bersama batuk kokol-radang paru-paru dijalankan. Model ini mengambil kira imuniti sementara bayi yang dijangkiti dan merangkumi ruang imuniti yang berasal daripada ibu. Nombor pembiakan asas model yang dijangkiti bersama diperoleh menggunakan matriks generasi berikutnya dan analisis kestabilan dilakukan. Model ini menunjukkan empat keseimbangan, iaitu, keseimbangan bebas batuk kokol, keseimbangan bebas radang paru-paru, keseimbangan bebas jangkitan dan keseimbangan endemik jangkitan bersama. Selepas itu, kestabilan tempatan keseimbangan bebas jangkitan dianalisis dan terbukti ia stabil secara asimptot. Begitu juga dengan membina fungsi Lyapunov yang sesuai, keseimbangan endemik jangkitan bersama terbukti stabil secara asimptot. Simulasi berangka dijalankan untuk menggambarkan kesahihan hasil ini.

Kata kunci: Fungsi kuadratik umum Lyapunov; imuniti terbitan ibu; jangkitan bersama batuk kokol-radang paru-paru; kestabilan global; penyakit respiratori kanak-kanak

INTRODUCTION

Respiratory diseases which affect the lungs as well as other parts of the respiratory system is a major cause of ill health for children (Gouveia & Fletcher 2000). Childhood respiratory disease may be caused by infection, exposure to second-hand smoke, radon, asbestos, and other forms of air pollution. Asthma, chronic obstructive pulmonary disease, pneumonia, pulmonary fibrosis, pertussis, and lung cancer are amongst the various types of respiratory diseases (NCI 2018). Different infectious agents may infect the host immune system as co-infection involves a variety of pathogens affecting the co-infected host. There are various types of pathogens that tend to infect humans. These include fungi, viruses, bacteria, protozoa, and helminths which tend to co-occur within individuals. Improved understanding of co-infection dynamics is required because co-infecting pathogens can interact directly with one another, or indirectly via the

infectious agents being harbored by host or host immune system (Clay et al. 2020; Cox 2001; Glidden et al. 2021; Griffiths et al. 2011). The interaction within the hosts could change the transmission, clinical progression and control of multiple infectious diseases (Birger et al. 2015; Griffiths et al. 2011; Pinky & Dobrovolny 2016).

Clinical research findings show the coexistence of pertussis and pneumonia in patients (Cheon et al. 2015; Jiang et al. 2021; Zouari et al. 2012). It was noted also that pertussis, or known as whooping cough causes a small fraction of severe pneumonia cases among hospitalized children (1-59 months of age) from the low and middle-income African and Asian countries (Barger-Kamate et al. 2016; Chang et al. 2019). Among these pneumonia cases in which pertussis was detected in infants less than 6 months old, 3.7% lead to in-hospital death (Barger-Kamate et al. 2016; Muloiwa et al. 2018). Thus, contending with the engagement of the above studies, there is a gap in the literature that needs to investigate the dynamical behaviour of these co-infected respiratory diseases in infants.

The primary objective of this study was to investigate the co-dynamics of pertussis and pneumonia co-infection in infants with maternally derived immunity using a deterministic approach of mathematical model. The importance and necessity of formulating and analyzing the mathematical models of co-infectious diseases cannot be over emphasized. Recent studies on the mathematical models of co-infectious diseases have presented interesting dynamics on the epidemiology of the respective co-infectious diseases (see for instance, Aggarwal 2020; Okosun et al. 2019; Omame et al. 2020; Shah et al. 2020). Similarly, some studies have related the co-dynamic effect of pneumonia (a severe respiratory disease in infants) with other disease, such as, co-infection of pneumonia and influenza (Cheng et al. 2017; Mbabazi et al. 2018), and co-infection of pneumonia and meningitis (Tilahun 2019). However, it is glaring and becoming apparent when engaging the existing literature that there is an absolute gap in the studies concerning the join effect of co-infection with pneumonia in infants using mathematical modeling. This provides a literature gap and a contribution to the field of knowledge. Therefore, the novelty of this study stalk on the introduction of the maternally derived immunity into the SIR model as MSIR, and a response to the reality checks and objective of the study. This model is an amplified version of the MSEIR model developed by Hethcote (2000). According to Hethcote (2000), uncovered the model for a population without age structure but exponentially changing population size and that with continuous age structure. Conversely, the dynamics on MSIR model by Bichara et al. (2014) and Yakubu et al. (2020) on the global dynamics of multi-strains SIS, SIR, and MSIR model and dynamical analysis on the transmission of pertussis with maternally derived immunity was considered for this particular study which incorporate some important epidemiological features with regards pertussis and pneumonia.

In addition, this paper presents a mathematical model for the co-dynamics of pertussis and pneumonia co-infection. To the best of our knowledge, this mathematical investigation on the co-infection of pertussis and pneumonia has not been given the scholarly attention it deserves. The narratives for the model steers on the transmission dynamics of these co-infectious diseases in infants. At birth, the infants are assumed to have natural immunity obtained from the transfer of maternal antibodies through the placenta (Barger-Kamate et al. 2016; Muloiwa et al. 2018), and thus the maternally derived immunity compartment (M) is infused into the model, thereby, making the model an MSIR model. Unlike the model of Bichara et al. (2014) and Yakubu et al. (2020), the co-infectious model extends to having more infected and recovered compartments because it involves co-infection of two diseases. Subsequently, the global stability of the model is analysed and numerical simulations are presented to observe the dynamic behaviour of the model.

DESCRIPTION OF THE MODEL

The model is based on the models of Bichara et al. (2014), Hethcote (2000), and Yakubu et al. (2020). The pertussis-pneumonia co-infection model consists of eight mutually exclusively compartments. The total population at time (t) denoted as $N(t)$ is given by

$$N(t) = M(t) + S(t) + I_{wc}(t) + I_{p}(t) + I_{wc-p}(t) + R_{wc}(t) + R_{p}(t) + R_{wc-p}(t) \quad (1)$$

The eight compartments are the maternally derived immunity $M(t)$, susceptible $S(t)$, pertussis-only infected $I_{wc}(t)$, pneumonia-only infected $I_{p}(t)$, pertussis-pneumonia co-infected $I_{wc\text{-}p}(t)$, pertussis-only recovered $R_{wc}(t)$, pneumonia-only recovered $R_{p}(t)$ pertussis-pneumonia recovered $R_{wc\text{-}p}(t)$ populations. The subscripts (wc), (p) and $(wc\text{-}p)$ represents pertussis (whooping cough), pneumonia and pertussis-pneumonia coinfection, respectively. Figure 1 presents the schematic flow chart

on the transmission of the co-infectious disease model. It illustrates the behavior of individuals going in and out of the eight compartments as the transmission of the diseases has been biologically described (Kilgore et al. 2016; Tilahun 2019).

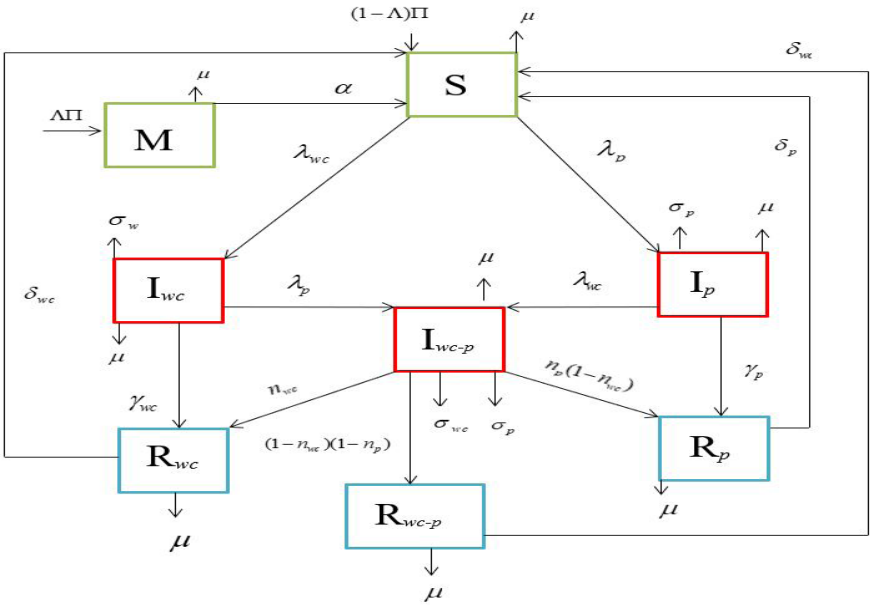


FIGURE 1. The flowchart of the co-infection model

In formulating the co-infection model, it is assumed that $\Lambda\Pi$ is the total proportion of immunized individuals, where Λ is the maternally immunized infants against infection and Π per capita birth rate, and the non-immunized individuals is $(1-\Lambda)\Pi$. The susceptible population increases from individuals that suffer waning of vaccine efficiency α, and sub-classes of pertussis-only recovered, pneumonia-only recovered and pertussis-pneumonia recovered by losing their temporary immunity at the rate δ_{wc}, δ_p and δ_{wc-p}, respectively. Individuals in the susceptible population can contract pertussis-only with the force of infection $\lambda_{wc} = \beta_{wc} c_{wc} (I_{wc} + I_{wc-p})$, where β_{wc} is the pertussis transmission rate and c_{wc} denotes the contact rate to join the I_{wc} compartment. Similarly, pneumonia-only can be contracted with the force of infection $\lambda_p = \beta_p c_p (I_p + I_{wc-p})$ where β_p denotes the transmission rate of pneumonia and c_p is the contact rate, thereby joining the I_p compartment. Pertussis-only infected infants have the tendency of getting infected with pneumonia with the force of infection λ_p and join the co-infected compartment (I_{wc-p}), and also the co-infected compartment increases when infants with pneumonia get infected by pertussis with the force of infection λ_{wc}. The population suffers a natural death at the rate μ and also disease-induced death caused by pertussis-only and pneumonia-only at the rate $\sigma_{wc} I_{wc}$ and $\sigma_p I_p$, respectively. In a similar way, the co-infected infants suffer a disease-induce death caused by severity of pertussis infection at the rate $\sigma_{wc} I_{wc-p}$ or severity in pneumonia infection at the rate $\sigma_p I_{wc-p}$. Pertussis-only infected infants recover from the disease at the rate γ_{wc} and join the pertussis-only recovered compartment. In a similar way, the pneumonia-only infected infants recover and join the pneumonia-only compartment at the rate γ_p. The co-infected infants recover at the rate γ. They either recover only from pertussis and join the pertussis-only recovered compartment with probability n_{wc}, or recover from pneumonia and join the pneumonia-only compartment with probability $n_p(1-n_{wc})$, or recover from both diseases and join the co-infection recovered compartment with probability $(1-n_{wc})(1-n_p)$, thereby having $n_{wc} + n_p(1-n_{wc}) + (1-n_{wc})(1-n_p) = 1$. From the above description and the flow chart shown in Figure 1, the following system of non-linear differential equations for the pertussis-pneumonia co-infection model is derived:

$$\begin{aligned}
\frac{dM}{dt} &= \Lambda\Pi - (\alpha+\mu)M \\
\frac{dS}{dt} &= (1-\Lambda)\Pi + \alpha M + \delta_{wc}R_{wc} + \delta_p R_p + \delta_{wc-p}R_{wc-p} - (\lambda_{wc}+\lambda_p+\mu)S \\
\frac{dI_{wc}}{dt} &= \lambda_{wc}S - (\lambda_p+\gamma_{wc}+\sigma_{wc}+\mu)I_{wc} \\
\frac{dI_p}{dt} &= \lambda_p S - (\lambda_{wc}+\gamma_p+\sigma_p+\mu)I_p \\
\frac{dI_{wc-p}}{dt} &= \lambda_{wc}I_p + \lambda_p I_{wc} - (\gamma+\sigma_{wc}+\sigma_p+\mu)I_{wc-p} \\
\frac{dR_{wc}}{dt} &= \gamma_{wc}I_{wc} + \gamma n_{wc}I_{wc-p} - (\delta_{wc}+\mu)R_{wc} \\
\frac{dR_p}{dt} &= \gamma_p I_p + \gamma n_p(1-n_{wc})I_{wc-p} - (\delta_p+\mu)R_p \\
\frac{dR_{wc-p}}{dt} &= \gamma(1-n_{wc})(1-n_p)I_{wc-p} - (\delta_{wc-p}+\mu)R_{wc-p}
\end{aligned} \tag{2}$$

The system (2) satisfies the following conditions:

$$M(t),\ S(t),\ I_{wc}(t),\ I_p(t),\ I_{wc-p}(t),\ R_{wc}(t),\ R_p(t),\ R_{wc-p}(t) \geq 0$$

Considering the basic co-infection model, it can be seen that

$$\begin{aligned}
\frac{dN}{dt} &= \frac{dM}{dt} + \frac{dS}{dt} + \frac{dI_{wc}}{dt} + \frac{dI_p}{dt} + \frac{dI_{wc-p}}{dt} + \frac{dR_{wc}}{dt} + \frac{dR_p}{dt} + \frac{dR_{wc-p}}{dt} \\
&\leq \Pi - \mu(M + S + I_{wc} + I_p + I_{wc-p} + R_{wc} + R_p + R_{wc-p}) \\
&\quad - \sigma_{wc}(I_{wc} + I_{wc-p}) - \sigma_p(I_p + I_{wc-p}).
\end{aligned} \tag{3}$$

Suppose that there is no death due to disease, that is $(\sigma_{wc} = \sigma_p = 0)$, then (3) becomes

$$\begin{aligned}
\frac{dN}{dt} &\leq \Pi - \mu N - \sigma_{wc}(I_{wc} + I_{wc-p}) - \sigma_p(I_p + I_{wc-p}) \\
&\leq \Pi - \mu N,
\end{aligned}$$

so that $N \leq \frac{\Pi}{\mu}$. The feasible region for the system (2) is defined as

$$\Omega_{wc-p} = \left\{ \left(M, S, I_{wc}, I_p, I_{wc-p}, R_{wc}, R_p, R_{wc-p}\right) \in \mathbb{R}_+^8 : 0 \leq M + S + I_{wc} + I_p + I_{wc-p} + R_{wc} + R_p + R_{wc-p} \leq \frac{\Pi}{\mu} \right\}.$$

EQUILIBRIUM SOLUTIONS

The equilibrium solution of the model (2) is obtained by equating the system to zero, i.e.,

$$\frac{dM}{dt} = \frac{dS}{dt} = \frac{dI_{wc}}{dt} = \frac{dI_p}{dt} = \frac{dI_{wc-p}}{dt} = \frac{dR_{wc}}{dt} = \frac{dR_p}{dt} = \frac{dR_{wc-p}}{dt} = 0$$

Solving for the compartments, the following equilibria are obtained:

1. In the absence of pneumonia disease:

$$E_{wc}^e = (M^e, S_{wc}^e, I_{wc}^e, 0, 0, R_{wc}^e, 0, 0)$$

This gives rise to the endemic equilibrium point of pertussis disease given by

$$\begin{aligned}
M^e &= \frac{\Lambda\Pi}{\alpha+\mu},\ S_{wc}^e = \frac{\gamma_{wc}+\sigma_{wc}+\mu}{\beta_{wc}c_{wc}}, \\
I_{wc}^e &= \frac{(\delta_{wc}+\mu)\left[\beta_{wc}c_{wc}\left(\alpha\Lambda\Pi + (\alpha+\mu)\phi\right) - \mu(\alpha+\mu)(\gamma_{wc}+\sigma_{wc}+\mu)\right]}{\beta_{wc}c_{wc}(\alpha+\mu)\left[\gamma_{wc}\mu + (\delta_{wc}+\mu)(\sigma_{wc}+\mu)\right]}, \\
R_{wc}^e &= \frac{\gamma_{wc}\left[\beta_{wc}c_{wc}\left(\alpha\Lambda\Pi + (\alpha+\mu)\phi\right) - \mu(\alpha+\mu)(\gamma_{wc}+\sigma_{wc}+\mu)\right]}{\beta_{wc}c_{wc}(\alpha+\mu)\left[\gamma_{wc}\mu + (\delta_{wc}+\mu)(\sigma_{wc}+\mu)\right]}.
\end{aligned}$$

2. In the absence of pertussis, that is the pertussis-free disease state:

$$E_p^e = (M_p^e, S_p^e, 0, I_p^e, 0, 0, R_p^e, 0)$$

This gives rise to the endemic equilibrium point of pneumonia disease given by:

$$\begin{aligned}
M^e &= \frac{\Lambda\Pi}{\alpha+\mu},\quad S_p^e = \frac{\gamma_p+\sigma_p+\mu}{\beta_p c_p}, \\
I_p^e &= \frac{(\delta_p+\mu)\left[\beta_p c_p\left(\alpha\Lambda\Pi + (\alpha+\mu)\phi\right) - \mu(\alpha+\mu)(\gamma_p+\sigma_p+\mu)\right]}{\beta_p c_p(\alpha+\mu)\left[\gamma_p\mu + (\delta_p+\mu)(\sigma_p+\mu)\right]}, \\
R_p^e &= \frac{\gamma_p}{\delta_p+\mu} I_p^e.
\end{aligned}$$

3. At a complete co-infection-free state,

$$E_{wc-p}^f = (M^f, S^f, 0, 0, 0, 0, 0, 0)$$

This gives rise to the pertussis-pneumonia free equilibrium point given by

$$\begin{aligned}
M^f &= \frac{\Lambda\Pi}{\alpha+\mu},\ S^f = \frac{\alpha\Lambda\Pi + (\alpha+\mu)(1-\Lambda)\Pi}{\mu(\alpha+\mu)}, \\
I_{wc}^f &= I_p^f = I_{wc-p}^f = R_{wc}^f = R_p^f = R_{wc-p}^f = 0
\end{aligned}$$

4. In the presence of both pertussis and pneumonia co-infection state,

$$E_{wc-p}^* = (M^*, S^*, I_{wc}^*, I_p^*, I_{wc-p}^*, R_{wc}^*, R_p^*, R_{wc-p}^*)$$

This gives rise to the pertussis-pneumonia endemic equilibrium state given by.

$$\begin{aligned}
M^* &= \frac{\Lambda\Pi}{\alpha+\mu},\ S^* = \frac{(g_{wc}+\lambda_p^{**})(g_p+\lambda_{wc}^{**})g_{wc-p}I_{wc-p}^{**}}{\lambda_{wc}^{**}\lambda_p^{**}(g_{wc}+g_p+\lambda_{wc}^{**}+\lambda_p^{**})}, \\
I_{wc}^* &= \frac{[\alpha\Lambda\Pi + (\alpha+\mu)\phi]\lambda_{wc}^{**}}{(\alpha+\mu)(\lambda_{wc}^{**}+\lambda_p^{**}+\mu)(\lambda_p^{**}+g_{wc})},
\end{aligned}$$

$$I_p^* = \frac{[\alpha\Lambda\Pi + (\alpha+\mu)\phi]\lambda_p^{**}}{(\alpha+\mu)(\lambda_{wc}^{**}+\lambda_p^{**}+\mu)(\lambda_{wc}^{**}+g_p)},$$

$$I_{wc-p}^* = \frac{\lambda_{wc}^{**}\lambda_p^{**}(\lambda_{wc}^{**}+\lambda_p^{**}+g_{wc}+g_p)g_0}{(\alpha+\mu)(\lambda_{wc}^{**}+\lambda_p^{**}+\mu)(\lambda_{wc}^{**}+g_p)(\lambda_p^{**}+g_{wc})g_{wc-p}},$$

$$R_{wc}^* = \frac{\gamma_{wc}I_{wc}^e + \gamma n_{wc}I_{wc-p}^e}{\delta_{wc}+\mu},\ R_p^* = \frac{\gamma_p I_p^e + \gamma n_p(1-n_{wc})I_{wc-p}^e}{\delta_p+\mu},$$

$$R_{wc-p}^* = \frac{\gamma(1-n_{wc})(1-n_p)I_{wc-p}^e}{\delta_{wc-p}+\mu}.$$

where $g_0 = \alpha\Lambda\Pi + (\alpha+\mu)\phi$, $g_{wc} = \gamma_{wc} + \sigma_{wc} + \mu$, $g_p = \gamma_p + \sigma_p + \mu$, $g_{wc-p} = \gamma + \sigma_{wc} + \sigma_p + \mu$ and $\phi = (1-\Lambda)\Pi$. The superscripts *f, e* and * represent disease-free, endemic and co-infection endemic equilibrium states.

BASIC REPRODUCTION NUMBER

The basic reproduction number, denoted as R_0 is a threshold parameter that plays a vital role in studying the dynamical behaviour of a model. It is the average number of secondary infections produced by an index case of completely uninfected population (Diekmann & Heesterbeek 2000; Diekmann et al. 1990). Note that when $R_0 < 1$, then, it implies that probability of new cases of co-infectious disease to persist in the population is insufficient for an outbreak to occur, and when, $R_0 > 1$, then the disease will become endemic and cause a drastic decline in the uninfected population of infants (Burrell et al. 2016). The next generation method (Diekmann et al. 1990; Siddik et al. 2020) is used to obtain the basic reproduction defined by $R_0 = \rho(FV^{-1})$, where ρ is the spectral radius and

$$F = \begin{pmatrix} \beta_{wc}c_{wc}S^f & 0 & \beta_{wc}c_{wc}S^f \\ 0 & \beta_p c_p S^f & \beta_p c_p S^f \\ 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} (\gamma_{wc}+\sigma_{wc}+\mu) & 0 & 0 \\ 0 & (\gamma_p+\sigma_p+\mu) & 0 \\ 0 & 0 & (\gamma+\sigma_{wc}+\sigma_p+\mu) \end{pmatrix}.$$

Thus,

$$FV^{-1} = \begin{pmatrix} \frac{\beta_{wc}c_{wc}S^f}{\gamma_{wc}+\sigma_{wc}+\mu} & 0 & 0 \\ 0 & \frac{\beta_p c_p S^f}{\gamma_p+\sigma_p+\mu} & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

The dominant eigenvalue of FV^{-1} is given by

$$R_0 = \rho(FV^{-1}) = \max\{R_{0wc}, R_{0p}\}$$
$$= \max\left\{\frac{\beta_{wc}c_{wc}(\alpha\Lambda\Pi + (\alpha+\mu)\phi)}{\mu(\alpha+\mu)(\gamma_{wc}+\sigma_{wc}+\mu)}, \frac{\beta_p c_p(\alpha\Lambda\Pi + (\alpha+\mu)\phi)}{\mu(\alpha+\mu)(\gamma_p+\sigma_p+\mu)}\right\}. \quad (4)$$

STABILITY ANALYSIS

In this section, the global stability for pertussis-only and pneumonia-only diseases is studied for the equilibrium points obtained, while local and global stability analysis of the co-infectious disease in model (2) is examined to observe the behavior of the model.

GLOBAL STABILITY OF $E_{wc}^f(M^f, S_{wc}^f, I_{wc}^f, 0, 0, R_{wc}^f, 0, 0)$ AT PERTUSSIS-FREE EQUILIBRIUM

Theorem 1 The pertussis-free equilibrium is globally asymptotically stable if $R_{0wc} \le 1$.

Proof The globally asymptotically stability of the pertussis-free equilibrium is proved using the Lyapunov function method (Vargas-De-León 2009).

Let the Lyapunov function V_{wc} be defined such that $V_{wc} = \frac{I_{wc}}{\gamma_{wc}+\sigma_{wc}+\mu}$. Taking the time derivative of V_{wc} gives

$$\frac{dV_{wc}}{dt} = \frac{1}{(\gamma_{wc}+\sigma_{wc}+\mu)}\frac{dI_{wc}}{dt} \quad (5)$$

Substituting the value of $\frac{dI_{wc}}{dt}$ as in (2) into (5), noting that there is absence of pneumonia and thus no co-infection, this gives

$$\frac{dV_{wc}}{dt} = \frac{(\beta_{wc}c_{wc}SI_{wc} - (\gamma_{wc}+\sigma_{wc}+\mu)I_{wc})}{(\gamma_{wc}+\sigma_{wc}+\mu)}$$
$$\Rightarrow \frac{dV_{wc}}{dt} = \frac{\beta_{wc}c_{wc}SI_{wc}}{(\gamma_{wc}+\sigma_{wc}+\mu)} - I_{wc}$$
$$\Rightarrow \frac{dV_{wc}}{dt} \le \left(\frac{\beta_{wc}c_{wc}(\alpha\Lambda\Pi + (\alpha+\mu)\phi)}{\mu(\alpha+\mu)(\gamma_{wc}+\sigma_{wc}+\mu)} - 1\right)I_{wc}$$
$$\Rightarrow \frac{dV_{wc}}{dt} \le (R_{0wc} - 1)I_{wc}$$

The above analysis shows that $\frac{dV_{wc}}{dt} \le 0$ if $R_{0wc} \le 1$ and also, $\frac{dV_{wc}}{dt} = 0$ if $I_{wc} = 0$ or $R_{0wc} = 1$. Thus, the pertussis-free equilibrium is globally asymptotically stable if $R_{0wc} \le 1$.

GLOBAL STABILITY OF $E_p^f(M^f, S_p^f, 0, I_p^f, 0, 0, R_p^f, 0)$ PNEUMONIA-FREE EQUILIBRIUM

Theorem 2 The pneumonia-free equilibrium is globally asymptotically stable if $R_{0p} \leq 1$

Proof The Lyapunov function method is used to prove the globally asymptotically stability of the pneumonia-free equilibrium (Vargas-De-León 2009).

Let the Lyapunov function V_p be defined such that $V_p = \dfrac{I_p}{\gamma_p + \sigma_p + \mu}$. The time derivative is given by

$$\frac{dV_p}{dt} = \frac{1}{(\gamma_p + \sigma_p + \mu)} \frac{dI_p}{dt} \tag{6}$$

Substitute the value $\dfrac{dI_p}{dt}$ as in (2) into (6), noting that that there is absence of pertussis and thus no co-infection, this gives

$$\begin{aligned}
\frac{dV_p}{dt} &= \frac{\beta_p c_p S I_p - (\gamma_p + \sigma_p + \mu) I_p}{(\gamma_p + \sigma_p + \mu)} \\
\Rightarrow \frac{dV_p}{dt} &= \frac{\beta_p c_p S I_p}{(\gamma_p + \sigma_p + \mu)} - I_p \\
\Rightarrow \frac{dV_p}{dt} &\leq \left(\frac{\beta_p c_p (\alpha\Lambda\Pi + (\alpha + \mu)\phi)}{\mu(\alpha + \mu)(\gamma_p + \sigma_p + \mu)} - 1 \right) I_p \\
\Rightarrow \frac{dV_p}{dt} &\leq (R_{0p} - 1) I_p
\end{aligned}$$

It is observed that $\dfrac{dV_p}{dt} \leq 0$ if $R_{0p} \leq 0$. Similarly, $\dfrac{dV_p}{dt} = 0$ if $I_p = 0$ or $R_{0p} = 1$. Therefore, by the LaSalle's Principle (LaSalle 1976), the pneumonia-free equilibrium is globally asymptotically stable if $R_{0p} \leq 1$. The local stability of the pertussis-pneumonia-free co-infection model is investigated in the next theorem, thereafter the global stability of the endemic state is analysed.

Theorem 3 The co-infection-free equilibrium point is locally asymptotically stable if $R_0 < 1$, otherwise unstable.

Proof The Jacobian of the model (2) at co-infection-free equilibrium (E_{wc-p}^f) is calculated and yields

$$J(E_{wc-p}^f) = \begin{pmatrix}
-(\alpha+\mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
\alpha & -\mu & -\frac{\beta_{wc} c_{wc} g_0}{\mu(\alpha+\mu)} & -\frac{\beta_p c_p g_0}{\mu(\alpha+\mu)} & -\frac{(\beta_{wc} c_{wc} + \beta_p c_p) g_0}{\mu(\alpha+\mu)} & \delta_{wc} & \delta_p & \delta_{wc-p} \\
0 & 0 & \frac{\beta_{wc} c_{wc} g_0}{\mu(\alpha+\mu)} - g_{wc} & 0 & \frac{\beta_{wc} c_{wc} g_0}{\mu(\alpha+\mu)} & 0 & 0 & 0 \\
0 & 0 & 0 & \frac{\beta_p c_p g_0}{\mu(\alpha+\mu)} - g_p & \frac{\beta_p c_p g_0}{\mu(\alpha+\mu)} & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & -g_{wc-p} & 0 & 0 & 0 \\
0 & 0 & \gamma_{wc} & 0 & \gamma n_{wc} & -(\delta_{wc} + \mu) & 0 & 0 \\
0 & 0 & 0 & \gamma_p & \gamma n_p (1 - n_{wc}) & 0 & -(\delta_p + \mu) & 0 \\
0 & 0 & 0 & 0 & \gamma (1 - n_p)(1 - n_{wc}) & 0 & 0 & -(\delta_{wc-p} + \mu)
\end{pmatrix}$$

The eigenvalues of the Jacobian matrix $\left(J(E_{wc-p}^f)\right)$ is given by

$$\lambda_1 = -\mu < 0, \ \lambda_2 = -(\alpha + \mu) < 0, \ \lambda_3 = -g_{wc-p} < 0, \ \lambda_4 = -(\mu + \delta_p) < 0, \ \lambda_5 = -(\mu + \delta_{wc}) < 0,$$

$$\lambda_6 = -(\mu + \delta_{wc-p}) < 0, \ \lambda_7 = \frac{\beta_{wc} c_{wc} (\alpha\Lambda\Pi + (\alpha + \mu)\phi)}{\mu(\alpha + \mu)} - g_{wc}, \ \lambda_8 = \frac{\beta_p c_p (\alpha\Lambda\Pi + (\alpha + \mu)\phi)}{\mu(\alpha + \mu)} - g_p,$$

The first six eigenvalues show that the co-infection-free equilibrium is stable, however λ_7 and λ_8 must be negative for this equilibrium state to be stable, that is, $\dfrac{\beta_{wc} c_{wc} (\alpha\Lambda\Pi + (\alpha + \mu)\phi)}{\mu(\alpha + \mu)} - g_{wc} < 0$ and $\dfrac{\beta_p c_p (\alpha\Lambda\Pi + (\alpha + \mu)\phi)}{\mu(\alpha + \mu)} - g_p < 0$, hence re-writing this gives $\dfrac{\beta_{wc} c_{wc} (\alpha\Lambda\Pi + (\alpha + \mu)\phi)}{\mu(\alpha + \mu)} < g_{wc}$, which implies that $\dfrac{\beta_{wc} c_{wc} (\alpha\Lambda\Pi + (\alpha + \mu)\phi)}{\mu(\alpha + \mu)} < 1$. Note that $R_{0wc} = \dfrac{\beta_{wc} c_{wc} (\alpha\Lambda\Pi + (\alpha + \mu)\phi)}{\mu(\alpha + \mu)}$, therefore it means that $R_{0wc} < 1$ and in a similar way, it is seen that $R_{0p} < 1$. Thus, CFE is locally asymptotically stable if and only if $R_0 = \max\{R_{0wc}, R_{0p}\}$.

GLOBAL STABILITY OF $E^* = (M^*, S^*, I_{wc}^*, I_p^*, I_{wc-p}^*, R_{wc}^*, R_p^*, R_{wc-p}^*)$ CO-INFECTION ENDEMIC EQUILIBRIUM

Theorem 4 The co-infection endemic equilibrium of the basic model (2) is globally asymptotically stable.

Proof Using the common quadratic Lyapunov function method as in Shah et al. (2020) and Vargas-De-León (2009). Let the Lyapunov function be given by

$$\begin{aligned}
G(M, S, I_{wc}, I_p, I_{wc-p}, R_{wc}, R_p, R_{wc-p}) = \\
\frac{1}{2}[(M - M^e) + (S - S^e) + (I_{wc} - I_{wc}^e) \\
+ (I_p - I_p^e) + (I_{wc-p} - I_{wc-p}^e) + (R_{wc} - R_{wc}^e) \\
+ (R_p - R_p^e) + (R_{wc-p} - R_{wc-p}^e)]^2
\end{aligned}$$

The time derivative of the Lyapunov function (G) is given by

$$\begin{aligned}
\frac{dG}{dt} = [(M - M^e) + (S - S^e) + (I_{wc} - I_{wc}^e) + (I_p - I_p^e) + \\
(I_{wc-p} - I_{wc-p}^e) + (R_{wc} - R_{wc}^e) \\
+ (R_p - R_p^e) + (R_{wc-p} - R_{wc-p}^e)][\frac{dM}{dt} + \frac{dS}{dt} + \frac{dI_{wc}}{dt} + \frac{dI_p}{dt} + \\
\frac{dI_{wc-p}}{dt} + \frac{dR_{wc}}{dt} + \frac{dR_p}{dt} + \frac{dR_{wc-p}}{dt}]
\end{aligned} \tag{7}$$

Substituting (3) into (7) yields

$$\begin{aligned}\frac{dG}{dt}=&[(M-M^e)+(S-S^e)+(I_{wc}-I_{wc}^e)+(I_p-I_p^e)+\\&(I_{wc-p}-I_{wc-p}^e)+(R_{wc}-R_{wc}^e)\\&+(R_p-R_p^e)+(R_{wc-p}-R_{wc-p}^e)][\Pi-\mu(M+S+I_{wc}+I_p+\\&I_{wc-p}+R_{wc}+R_p+R_{wc-p})]\end{aligned}\tag{8}$$

given that $\Pi=\mu(M^e+S^e+I_{wc}^e+I_p^e+I_{wc-p}^e+R_{wc}^e+R_p^e+R_{wc-p}^e)$, then (8) becomes

$$\begin{aligned}\frac{dG}{dt}=&[(M-M^e)+(S-S^e)+(I_{wc}-I_{wc}^e)+(I_p-I_p^e)+\\&(I_{wc-p}-I_{wc-p}^e)+(R_{wc}-R_{wc}^e)+(R_p-R_p^e)\\&+(R_{wc-p}-R_{wc-p}^e)][\Pi-\mu\Big((M-M^e)+(S-S^e)+\\&(I_{wc}-I_{wc}^e)+(I_p-I_p^e)+(I_{wc-p}-I_{wc-p}^e)\\&+(R_{wc}-R_{wc}^e)+(R_p-R_p^e)+(R_{wc-p}-R_{wc-p}^e)\Big)]\\=&-\mu\Big[(M-M^e)+(S-S^e)+(I_{wc}-I_{wc}^e)+(I_p-I_p^e)\\&+(I_{wc-p}-I_{wc-p}^e)+(R_{wc}-R_{wc}^e)\\&+(R_p-R_p^e)+(R_{wc-p}-R_{wc-p}^e)\Big]^2\\\le&\,0\end{aligned}$$

Here, $\frac{dG}{dt}\le 0$. Note also that $\frac{dG}{dt}=0$ if and if $M=M^e$, $S=S^e$, $I_{wc}=I_{wc}^e$, $I_p=I_p^e$, $I_{wc-p}=I_{wc-p}^e$, $R_{wc}=R_{wc}^e$, $R_p=R_p^e$, $R_{wc-p}=R_{wc-p}^e$. It is seen that by the common quadratic Lyapunov method, the co-infection endemic equilibrium is globally asymptotically stable.

SENSITIVITY ANALYSIS

A sensitivity analysis is carried out on some basic parameters of the model. This analysis helps to ascertain the robustness of parameters that have great influence on the basic reproduction number R_0. Following LaSalle (1976) and Tilahun (2019), the normalized forward sensitivity index on the parameters that appear in R_0 are estimated. The sensitivity index is defined by $\chi_k^{R_0}=\frac{\partial R_0}{\partial k}\times\frac{k}{R_0}$ where, k is any parameter in R_0. Considering that $R_0=\max\{R_{0w},R_{0p}\}$, the sensitivity analysis of the parameters involved are obtained separately. For example, the sensitivity index of R_0 with respect to the pertussis transmission rate is given by

$$\chi_{\beta_{wc}}^{R_0}=\frac{\partial R_0}{\partial\beta_{wc}}\times\frac{\beta_{wc}}{R_0}=\frac{c_{wc}(\alpha+\mu(1-\Lambda)\Pi)}{(\alpha+\mu)(\gamma_{wc}+\sigma_{wc}+\mu)}\times\frac{(\alpha+\mu)(\gamma_{wc}+\sigma_{wc}+\mu)}{c_{wc}(\alpha+\mu(1-\Lambda)\Pi)}>0$$

Other indices $\chi_\alpha^{R_{0wc}}$, $\chi_{c_{wc}}^{R_{0wc}}$, $\chi_{\gamma_{wc}}^{R_{0wc}}$, $\chi_\mu^{R_{0wc}}$, $\chi_{\sigma_{wc}}^{R_{0wc}}$ and $\chi_{\beta_p}^{R_{0p}}$, $\chi_{c_p}^{R_{0p}}$, $\chi_\mu^{R_{0p}}$, $\chi_{\gamma_p}^{R_{0p}}$, $\chi_{\sigma_p}^{R_{0p}}$ are obtained in the same manner and evaluated using parameter values in Table 1.

TABLE 1. Parameter value

Parameter	Value	Source
Λ	0.35	Kilgore et al. (2016)
Π	0.095	Kilgore et al. (2016)
α	0.25	Nthiiri et al. (2015)
β_{wc}	1.513	Nthiiri et al. (2015)
β_p	1.013	Tilahun (2019)
c_{wc}	0.5-1	Estimated
c_p	0.5-1	Pesco et al. (2015)
δ_{wc}	0.06	Kilgore et al. (2016)
δ_p	0.05	Pesco et al. (2015)
δ_{wc-p}	0.07	Estimated
μ	0.00313	Estimated
σ_{wc}	0.0309	Pesco et al. (2015)
σ_p	0.0206	Tilahun (2019)
γ	0.18	Cheon (2015)
γ_{wc}	0.0205	Pesco et al. (2015)
γ_p	0.0195	Pesco et al. (2015)
n_{wc}	0.5-1	Estimated
$n_p(1-n_{wc})$	0.5-1	Estimated
$(1-n_{wc})(1-n_p)$	0.5-1	Estimated

Table 2 shows the summary of the sensitivity analysis. This sensitivity index is a local estimate of the best way to reduce R_0. An increase in a more sensitive parameter while keeping other parameters constant will increase the value of the basic reproduction number, thereby causing an increase in the endemicity of the disease since they are positive indices. Note that in examining sensitivity analysis, it is not biologically appropriate to suggest that human mortality $(\mu, \sigma_{wc}, \sigma_p)$ be increased in order to control the spread of disease.

TABLE 2. Sensitivity indices

Parameters	Sensitivity indices
R_{0wc}	
β_{wc}	+ve
c_{wc}	+ve
α	+ve
γ_{wc}	-ve
σ_{wc}	-ve
R_{0p}	
β_p	+ve
c_p	+ve
γ_p	-ve
σ_p	-ve
μ	-ve

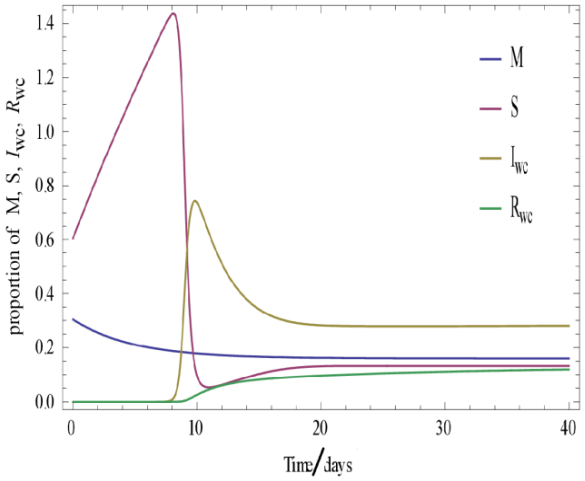
NUMERICAL SIMULATIONS

Numerical simulation has been performed for the pertussis-pneumonia co-infection model using the Mathematica software package. The numerical simulation supports the analytic results of the pertussis-pneumonia co-infection model obtained in previous sections. The behaviour of infant population in the different compartments is observed. The parameter values in Table 1 are used for the simulation. Figure 2 illustrates a time series graph of the co-infection model which is partitioned with respect to the single disease only and co-infection of the disease, in order to have a clearer insight on the behaviour of each compartment in relation to the co-infection dynamics. The phase portrait of the pertussis-pneumonia co-infection model is presented in Figure 3 which shows the stability behaviour the infected infant population.

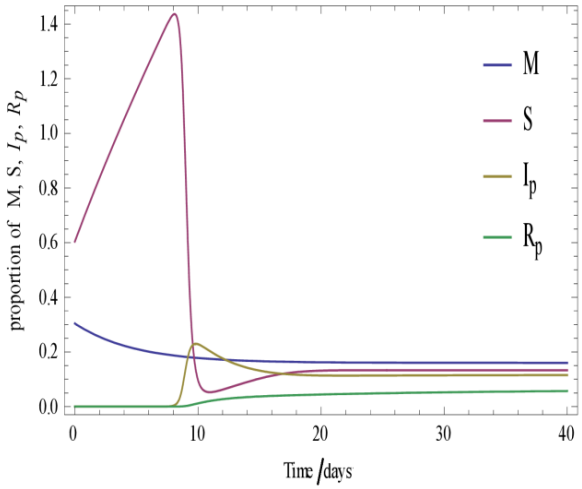
Figure 2 shows the time series solution of the co-infection model, where it is seen how the healthy infant population which have temporary immunity decreases due to contact with the disease. The graph also shows a relatively high increase in the population of infants with co-infection of both diseases as observed in Figure 2(d). This signifies the effect of infants with pertussis been exposed to the pneumonia infected populace. The stability of the co-infection endemic equilibrium is seen in the phase portrait as shown in Figure 3.

The trajectories of the equilibrium state are illustrated in Figure 3 which presents the graph of (a) susceptible and pertussis-only infected, (b) susceptible and pneumonia-only infected while (c) susceptible and pertussis-pneumonia co-infected infant population. The vaccine efficiency parameter is varied to observe its implication on the stability of the system, noting

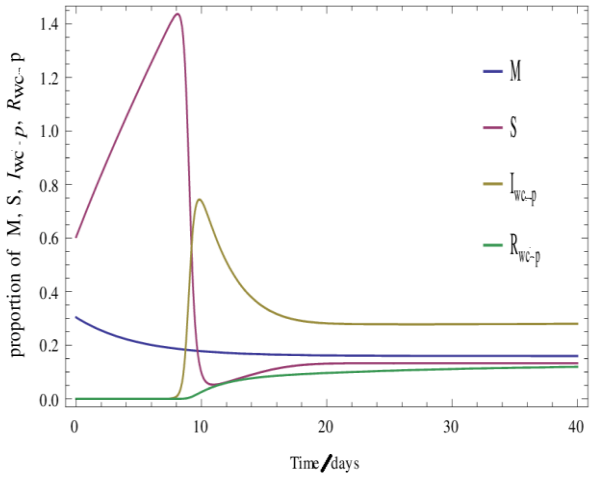
that it is a significant parameter of the model (2). The increased variation in the vaccine efficiency parameter relates that more infants with strengthened immune system will remain in a susceptible state for a longer period before becoming infected due to waning of the vaccine. However, it is observed from Figure 3(a)-3(c) that susceptible infants show a high risk of becoming co-infected with pertussis and pneumonia as compared to pertussis-only and pneumonia-only infected infants, respectively. The figure shows the conditions for stability has been established even with variation in the vaccine efficiency parameter and it is globally stable.



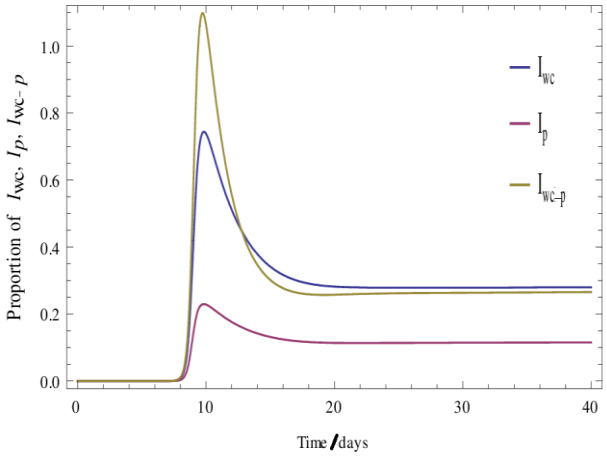
(a) Pertussis



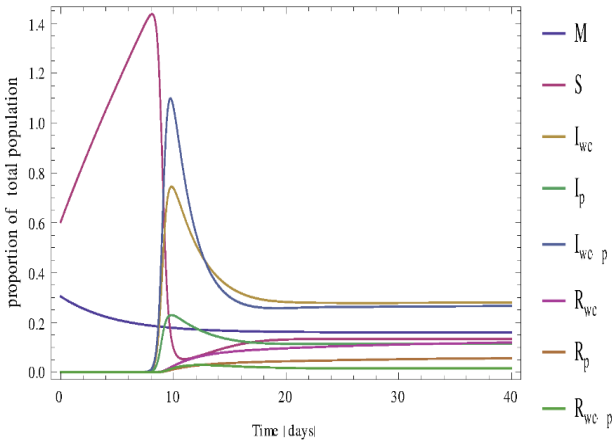
(b) Pneumonia



(c) Co-infection

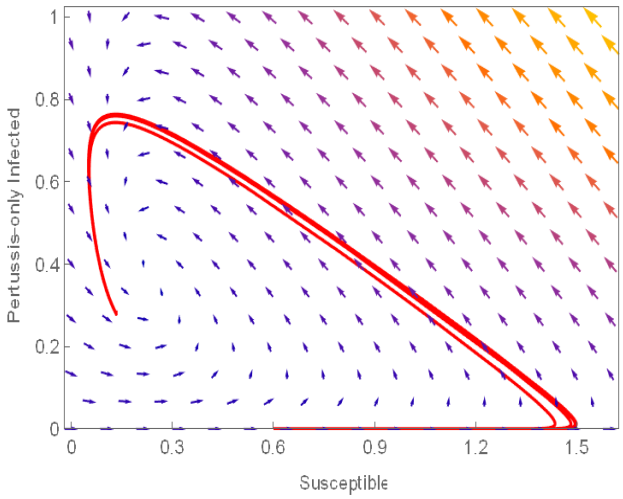


(d) All infected

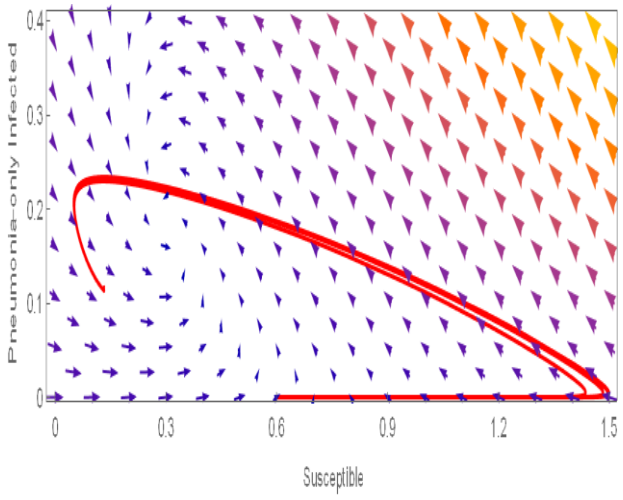


(e) Total population

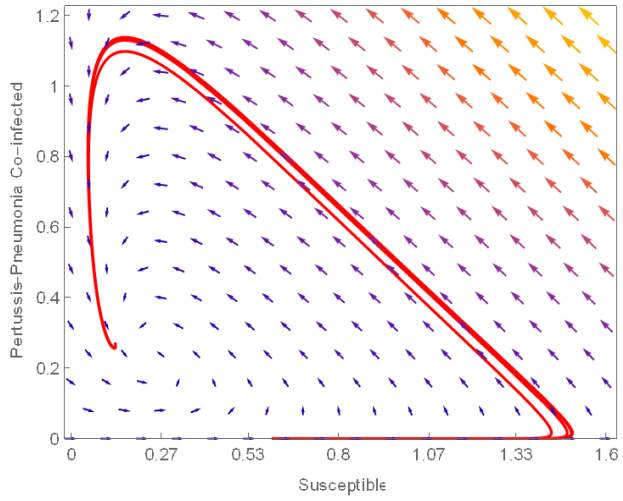
FIGURE 2. Time series plot for the pertussis-pneumonia co-infection model



(a)



(b)



(c)

FIGURE 3. The phase portrait of pertussis-pneumonia co-infectious disease with variation in the vaccine efficiency parameter (0.205, 0.405, 0.605 and 0.805) at (a) S and I_{wc}, (b) S and I_p, and (c) S and $I_{wc\text{-}p}$ 2D-planes

RESULTS AND DISCUSSION

In this paper, a model for pertussis-pneumonia co-infection was developed and the transmission dynamics of this model in infant was analysed. The model was divided into eight compartments; maternally derived immunity, susceptible, pertussis-only infected, pneumonia-only infected, pertussis-pneumonia co-infected, pertussis-only recovered, pneumonia-only recovered and pertussis-pneumonia co-infection recovered. The stability of the basic model was investigated considering the co-infection-free and endemic equilibrium states. In addition, the basic reproduction number was calculated using the next generation matrix and the model has a dominant eigenvalue of FV^{-1} given by $R_0 = \rho(FV^{-1}) = \max\left\{R_{0wc}, R_{0p}\right\}$. R_0 is a very important threshold parameter that is used in observing the dynamic behaviour in disease modelling because it is a determining factor to whether or not the disease will persist in the population. Theoretically, the global stability for co-infection endemic equilibrium was investigated using the common quadratic Lyapunov function method which showed that the system was globally asymptotically stable. Sensitivity analysis of basic parameters in the basic reproduction of the co-infection model are analysed in their respective order. Numerical simulations were carried out to have a graphical illustration on the behaviour of the infant populace with respect time as seen in Figure 2. Though the disease infected infant (single and co-infected) population attains a stable equilibrium point, the population does not go extinct which could result in the reinfection of the disease if not given adequate clinical attention. Similarly, from the basic model (2), considering the Theorem 4 with parameter values as seen in Table 1, the system exhibits global stability as illustrated in Figure 3. The variation in the vaccine parameter did not alter the stability behaviour of the system, however, it is seen that infant with strengthened immune system remain susceptible for a longer period, however, they show a relatively high risk of becoming co-infected with pertussis and pneumonia.

CONCLUSION

A system of eight ordinary differential equations which incorporates the maternally derived immunity compartment is developed and analyzed. The model exhibits four equilibria; namely, the pertussis-free equilibrium, the pneumonia-free equilibrium, the co-infection-free equilibrium and co-infection endemic equilibrium. Using the Lyapunov function technique, the endemic equilibria were shown to be globally asymptotically stable. The sensitivity analysis was carried out and showed the sensitivity of each parameter as seen in Table 2. An increase in the dominant parameter while keeping other parameters constant will increase the value of the basic reproduction number, thereby, increasing the tendency of endemicity of the disease. Numerical simulations were also carried out which shows the global dynamics of the system to be well established.

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