SEIRS MODEL FOR PEDIATRICS WITH LOWER RESPIRATORY TRACT INFECTION

Abstract

The ability of the immune system to detect and eliminate most pathogens is essential for the survival of lower respiratory tract infection. (Olubadeji, 2016). Lower respiratory tract infection (LRTI) constituted the second leading cause of death in all age bracket in Nigeria (Loddenkemper, 2013) Chronic lower respiratory diseases rank as the third leading cause of death in the United States. Intense research has been on how to reduce the spread of infection, which involves the mathematical modelling of the spread of infection based on mathematical epidemiological approach, This is necessary because a threshold cannot be discerned from the data generated from the Hospitals, rather it requires a mathematical model to analyze and simulate the LRTI dynamics on the enviroment. It also enables the calculation of the basic reproductive number (R_0) which is an important threshold for determining whether the environments are at risk or not. In this paper, we adopt the susceptible- Exposed-infected-recovered-susceptible (SEIRS) model to depict the spread of infections in our environment. We qualitatively analyze the model and establish that the virus-free state is locally asymptotically stable provided the basic reproduction number is less than unity. We solved the model numerically and simulate the solution for different scenarios on the network. The findings from our simulations are discussed.

Keywords: Mathematical Model, Basic Reproductive Number, Lower respiratory tract infection, Equilibrium Solution.

1. Introduction

Child mortality and morbidity is a factor that can be associated with the well-being of a population. It is also taken as one of the development indicators of health and socioeconomic status in any country (Alderman and Behrman, 2004). In order to reduce child mortality and morbidity which is one of the important Millennium goals, there is need to develop an effective and efficient model both (mathematics and computer science) that can be used to assess the attributes that are responsible for the prevalence of the diseases in pediatrics patients that are having LRTIs. This research is focused on children because a child's death is emotionally and physically damaging for the mourning parents. Lower respiratory tract infection continues to be the second leading killer of children under five years of age worldwide. It is the leading cause of morbidity and mortality in both developing and developed countries (Rudan et al,2008). WHO, 2010 recognized respiratory diseases as the second important cause of death for children under five years in 2010. WHO, 2004 states that respiratory infections like pneumonia is one of the main three causes for newborn infant deaths.

Mathematical models have been used in the control of infectious diseases like that in Muhammed and Orukpe, 2014 where it was applied to malaria control. In this paper, a modified mathematical version of Andreas *et al., 2001* for epidemics caused by respiratory syncytial virus is formulated. It is assumed herein that birth rate and death rate are unequal and that there exist latent individuals who have been infected but not yet infectious. All parameters are defined in Table (1).

Under the above assumptions, the governing equations for epidemics caused by respiratory syncytial virus is given as:

$$\frac{dS}{dt} = \delta - \beta(t)SI + \gamma R - \mu S, \tag{1}$$

$$\frac{dE}{dt} = \beta(t)SI - \mu E - E\sigma, \tag{2}$$

$$\frac{dI}{dt} = E\sigma - \mu I - \vartheta I, \tag{3}$$

$$\frac{dR}{dt} = \vartheta I - \mu R - \gamma R, \tag{4}$$

$$N(t) = S(t) + E(t) + I(t) + R(t),$$

where $\beta(t) = b_0\,(1 + b_1 \cos(2\pi t + \varphi))$ is the transmission parameter. For mathematical sensibility, it is assumed that $b_j > 0$ for $j = 0, 1$ and $\delta, \mu, \sigma, \vartheta, \gamma > 0$. These equations are equipped with the following initial conditions: $S(0) = S_0$, $E(0) = E_0$, $I(0) = I_0$ and $E(0) = E_0$. The population size is assumed to vary with birth and death rates unequal so that

$$\frac{dN}{dt} = -N\mu + \delta, \tag{5}$$

Table 1: The description of parameters and values used in the model

δ	0.051	birth rate
μ	0.041	death rate
ϑ	66	rate of loss of infectiousness
σ	91	contact rate
γ	60	rate of loss of immunity
ϕ	0.61	phase angle
b_0	0.15	average transmission parameter
b_1	1.8	amplitude parameter

1 Disease-free equilibrium

The population is free from infection if $I = E = 0$ and there is an infection-free equilibrium if the models (1)-(4) are set at zero and the solution is given by

$$\psi_0 = (S, E, I, R) = (\delta/\mu\,, 0, 0)$$

which corresponds to the result derived by Gumel [2]. The equilibrium stability is established by finding the Jacobian of Eqns. (1)-(4) at ψ_0. The eigenvalues derived based on signs predict the local stability of ψ_0. The Jacobian is given by:

$$J_0 = \begin{bmatrix} -\mu & 0 & -\frac{\beta\delta}{\mu} & \gamma \\ 0 & -(\mu+\sigma) & \frac{\beta\delta}{\mu} & 0 \\ 0 & \sigma & -(\mu+\vartheta) & 0 \\ 0 & 0 & \vartheta & -(\mu+\gamma) \end{bmatrix}, \tag{6}$$

where $\lambda_1 = -\mu$, $\lambda_2 = -(\mu + \gamma)$ and the roots of the quadratic

$$f(\lambda) = \lambda^2 + (2\,\mu + \sigma + \vartheta)\,\lambda + (\mu+\sigma)\,(\mu+\vartheta) - \frac{\beta\,\delta\,\sigma}{\mu}$$

Now, $\lambda_1 < 0$ and $\lambda_2 < 0$ since all parameters have been assumed to be positive. In the light of Moghadas and Gumel [3], defining

$$\Re_0 = \frac{\beta\,\delta\,\sigma}{\mu\,(\mu+\sigma)\,(\mu+\vartheta)},$$

shows that $<_0 < 1$, thus $f(\lambda)$ has a negative real part. In fact,

$$\lambda_j = \frac{-(2\,\mu^2 + \mu\,\vartheta + \mu\,\sigma) \pm \sqrt{\mu^2\vartheta^2 - 2\,\mu^2\vartheta\,\sigma + \mu^2\sigma^2 + 4\,\mu\,\beta\,\delta\,\sigma}}{2\mu}, \quad j = 3, 4.$$

Lemma 1. *The disease-free equilibrium ψ_0 is locally asymptotically stable if $\mathbb{R}_0 < 1$ and unstable if $\mathbb{R}_0 > 1$.*

2 Endemic equilibrium

When infection is present (*i.e.* I 6≠ 0 and E 6≠ 0) the Eqns. (1)-(4) have a unique equilibrium $\psi^* = (S^*, E^*, I^*, R^*)$ individually expressed as

$$S = \frac{(\mu+\vartheta)(\mu+\sigma)}{\beta\sigma}, \tag{7}$$

$$E = \frac{\mu(\mu+\sigma)(\mu+\gamma)(\mu+\vartheta)^2(\Re_0-1)}{[(\mu+\vartheta)(\mu+\gamma)+\sigma(\gamma+\mu+\vartheta)]}, \tag{8}$$

$$I = \frac{(\mu+\gamma)\mu(\mu+\sigma)(\mu+\nu)(\Re_0-1)}{[(\mu+\vartheta)(\mu+\gamma)+\sigma(\gamma+\mu+\vartheta)]}, \tag{9}$$

$$R = \frac{\vartheta(\mu+\sigma)(\mu+\nu)(\Re_0-1)}{\beta[(\mu+\vartheta)(\mu+\gamma)+\sigma(\gamma+\mu+\vartheta)]}. \tag{10}$$

It is clearly seen from Eqns. (7)-(10) that if $\mathbb{R}_0 < 1$, then the model possesses negative endemic equilibrium. This simply means that I^* and E^* have a negative value which is not realistic in the real world. Thus, for reality, it is demanded that $\mathbb{R}_0 > 1$ since this yields $(S^*, E^*, I^*, R^*) > 0$.

The local stability of ψ^*, it is recalled from the total population that

$$\frac{dN}{dt} = \delta - \mu N, N(0) = N_0,$$

which implies that

$$N(t) = \frac{\delta}{\mu} + \mathrm{e}^{-t\mu}\left(N_0 - \frac{\delta}{\mu}\right).$$

Therefore,

$$N(t) \to \frac{\delta}{\mu} \text{ as } t \to \infty.$$

It can definitely be assumed that when the population size has its final value, (*i.e.*, $N = \delta/\mu = S(t) + E(t) + I(t) + R(t)$), $R(t) = \delta/\mu - S(t) - E(t) - I(t)$.

Lemma 2. If $\mathbb{R}_0 > 1$ then the unique endemic equilibrium ψ^* is locally asymptotically stable.

3 **Graphical Illustrations**

From Figs. 1a and 1b, it can be clearly seen that the birth rate enhances the number of paediatrics that are latent and infected. This is true in the real life since an addition in population brings about an increment in the infected individuals and those infected but are not yet infectious. Depicted on Figs. 2a and 2b is the effect of death rate on the population of paediatrics. It can be seen that the death rate decreases the latent individuals as well as the infected population. This is also true in the real world since death rate reduces the whole population in the long run. Figs. 3a and 3b illustrate the effects of rate of loss of immunity on the latent and infected individuals. It can be evidently seen that the rate of loss of immunity increases the latent and infectious profiles. As depicted in Figs. 4a and 4b, an increase in the rate of loss of infectiousness causes an increase in the latent profiles but a decrease in the infected profiles. As described in Figs. 5a and 5b an enhancement in the average transmission parameter and amplitude parameter lead to a decrease in latent individuals' profiles and an increase in the infectious population.

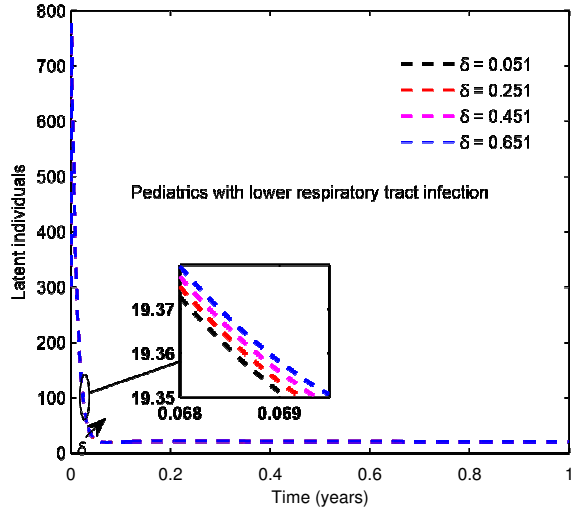

Figure 1a: Effect of δ on $E(t)$.

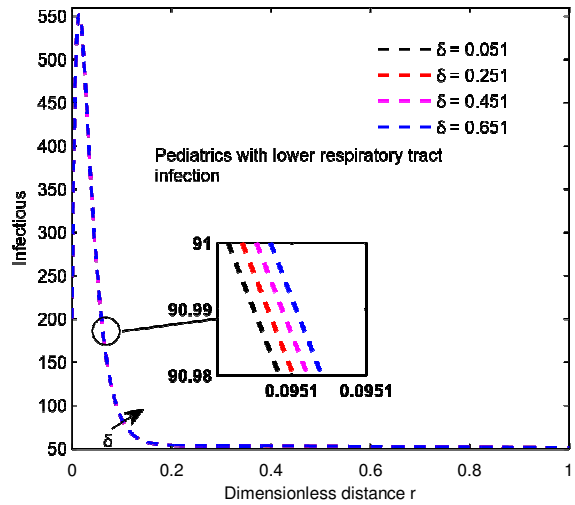

Figure 1b: Effect of δ on $I(t)$.

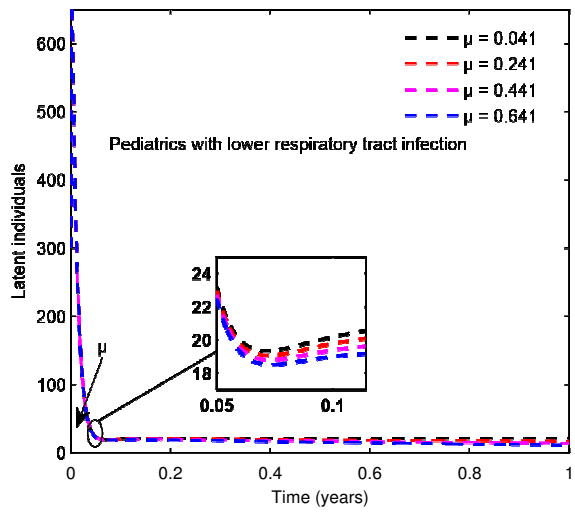

Figure 2a: Effect of μ on $E(t)$.

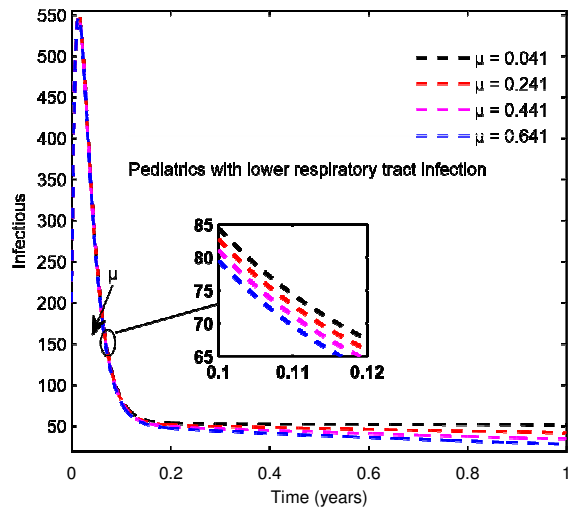

Figure 2b: Effect of μ on $I(t)$.

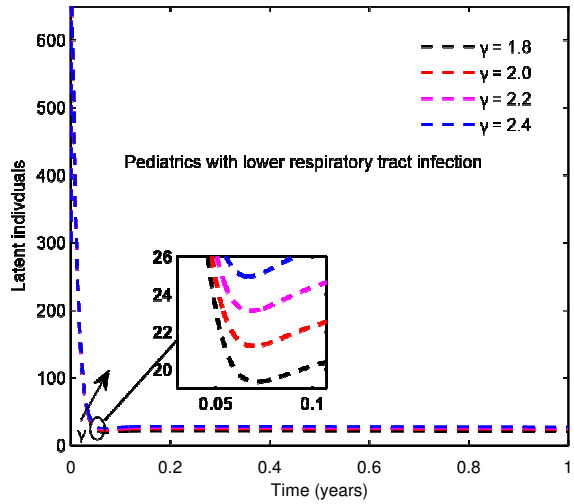

Figure 3a: Effect of γ on $E(t)$.

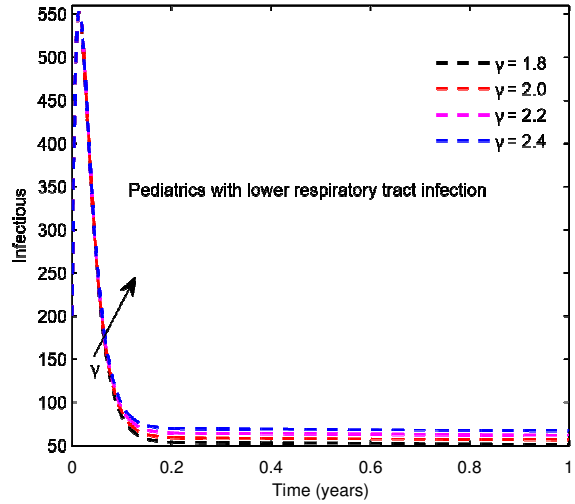

Figure 3b: Effect of γ on $I(t)$.

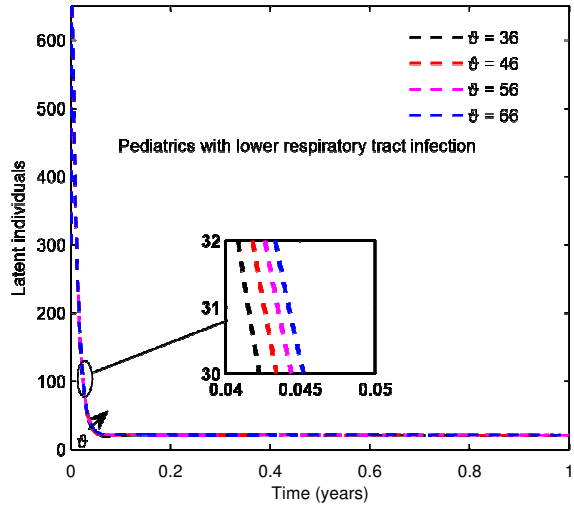

Figure 4a: Effect of ϑ on $E(t)$.

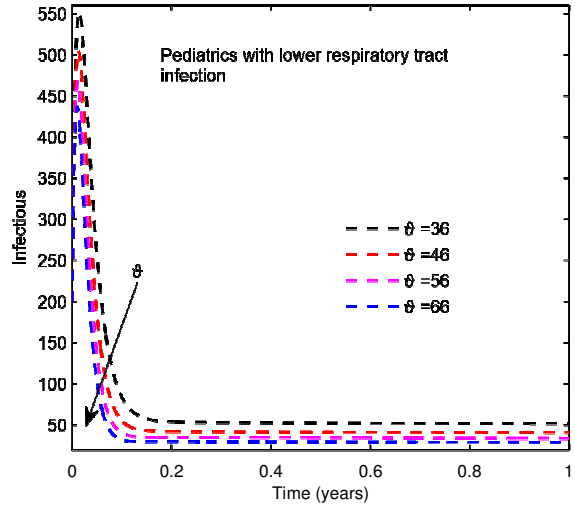

Figure 4b: Effect of ϑ on $I(t)$.

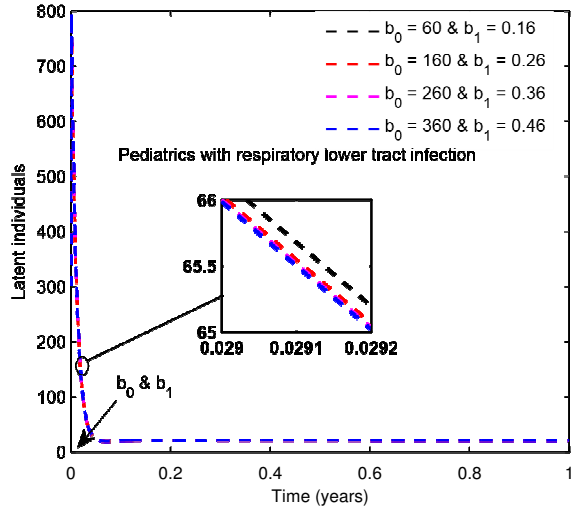

Figure 5a: Effect of b_0 and b_1 on $E(t)$.

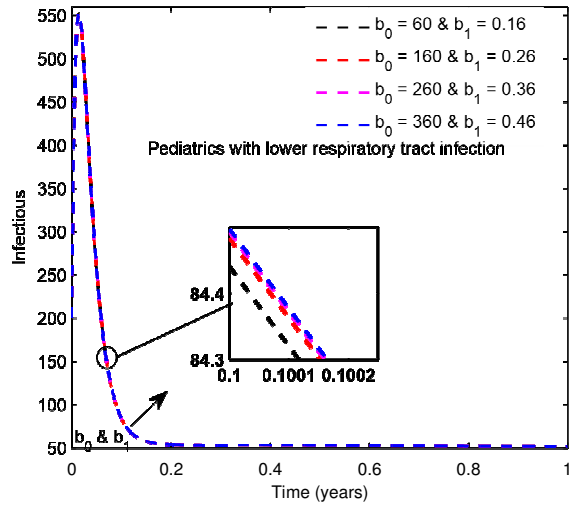

Figure 5b: Effect of b_0 and b_1 on $I(t)$.

4 Conclusions

In this work, mathematical model has been applied to lower respiratory tract infection. Based on the research conducted on the paediatrics with lower tract infection, the following conclusions were drawn

1. An enhancement in the birth rate increases both latent and infected population.
2. A hike in the death rate decreases both latent and infected population.
3. Increasing the rate of loss of immunity increases the latent and infectious profiles.
4. An increase in the rate of loss of infectiousness causes an increases the latent population but diminishes the infected profiles.
5. An enhancement in the average transmission parameter and amplitude parameter lead to a decrease in latent individuals profiles and an increase in the infectious.

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