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# MSEIRS Model for Pediatrics with Lower Respiratory Tract Infection

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## I. INTRODUCTION

The model uses the techniques of epidemiological models, the idea is to abstract away the particular details of an infection and express individuals as progressing through a set of states at different rates. 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 that can be used to assess the attributes that are responsible for the prevalence of the diseases in pediatrics patients that are having Lung Respiratory Tract Infections (LRTIs). In this epidemiological model, individuals transition from a Passive Immunity Infant to Susceptible state to Latent period to an Infectious one to a Recovered state at a certain rate, and become Susceptible again at a different rate. This model is called the MSEIRS model, because individuals move between them M (Passive Immunity infant), E (Latent period) S (Susceptible) and I (Infectious states) R (Recovered).

The Passive Immunity for Infant - Susceptible – Latent – Infected – Recovered - Susceptible (MSEIRS)

model was introduced by Kermack and McKendrick, in 1927 (Leah Edelstein-Keshet 2005). In the model, the population is divided into three distinct groups of: the Passive Immunity for Infant (M), Latent period (E), Susceptibles (S), Infecteds (I) and Recovereds (R) where M, E, S, I and R represent the number of children in each of the groups respectively and the total population  $N = M + E + S + I + R$ . The Susceptibles are those who are not infected and not immune, the Infecteds are those who are infected and can transmit the disease, and the Recovered are those who are immune to re-infection. The characteristic feature of LRTI is that immunity after infection is temporary, such that the recovered children can become susceptible again if all the risk factors are still present.

## II. MATHEMATICAL MODEL FORMULATION

*Passive Immunity* is an immunity obtained from external source: immunity from disease acquired by the transfer of antibodies from one person to another, e.g. through injections or between a mother and a fetus through the placenta looking at the case of infection spread on the population of children, there is an arrival of new susceptible population. In this type of situation, births and deaths rate must be included in the model. The differential equations represent the model which indicates the rate of change of number of individuals in each compartment with respect to time. Below is the Schematic diagram for the single age class M - Passive Immunity Infant, S- Susceptible, E – Latent period, I – Infectious, R – Recovered (MSEIRS) model for LRTI transmission (Weber et al., (2001).

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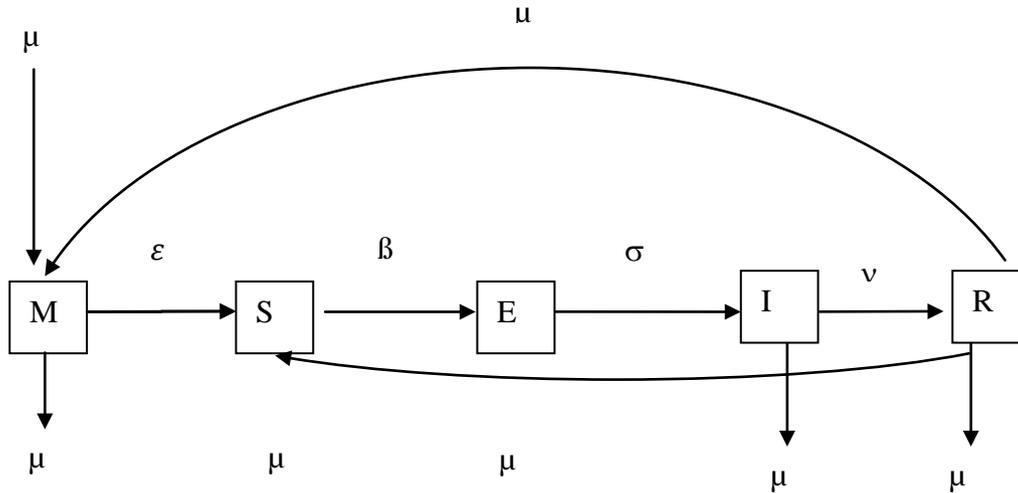


Fig. 1: Schematic diagram for the single age class MSEIRS (M = Passive Immunity Infants) Susceptible Latent Immune Recovered Susceptible Model for LRTI transmission

The above assumptions lead to the following differential equations for LRTI.

An additional feature of LRTI is employed. By this Newborn babies whose mothers are immune are taken into consideration .As a result, these children are protected by the antibodies present in their mothers. Thus, group M of children who are completely protected by these antibodies are considered. The ratio of these newborn babies M is equal to the ratio of the general population that is immunized after recovering from infection. Protection reduces and these children M become susceptible at a rate. Under the above assumptions, the following are the results.

$$\frac{dM}{dt} = \mu - (+\mu)M, \quad M(0) \tag{2.1}$$

$$\frac{dS}{dt} = M - \beta SI - \mu S + \gamma R, \quad S(0) \tag{2.2}$$

$$\frac{dE}{dt} = \beta SI - (\sigma + \mu)E, \quad E(0) \tag{2.3}$$

$$\frac{dI}{dt} = \sigma E - (v + \mu)I, \quad I(0) \tag{2.4}$$

$$\frac{dR}{dt} = vI - (\mu + \gamma)R, \quad R(0) \tag{2.5}$$

Table 1: The description of parameters used in the model

Parameter	Description	Unit
S	Susceptible population	Number/unit time
M	Birth rate of the children i.e. the mortality rate	Number / unit time
I	Infected population	Number/unit time
R	Infected population that Recovered	Number/unit time
M	Passively immune infants	Number/unit time
μ	Birth rate of the children i.e. the mortality rate	Number/unit time
γ	rate of loss of immunity	Number/unit time
v	Rate of loss of infections	Number/unit time
β	Transmission parameter (constant rate)	Number/unit time
R <sub>0</sub>	Basic reproduction number	Number/unit time
Σ	Contact number	Number/unit time
E	Rate of loss of protection by maternal antibodies	Number/unit time

The unit time is (per year)

### III. MODEL ANALYSIS

#### a) Two Classes of Epidemiology Models

To introduce the terminologies, notation, and standard results for epidemiology models, two different types of models are formulated and analyzed. They are Epidemic models and Endemic models. Epidemic model is used to describe rapid outbreaks that occur in

less than a year due to the availability of some risk factors, while endemic models are used for studying diseases of longer periods, during which there is a renewal of susceptibles by births or recovery from temporary immunity. The two classic SIR models provide an intuitive basis for understanding more complex epidemiology modeling results.

i. Model Equilibrium Solutions

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

Thus we have,

$$\frac{dM}{dt} = \mu - (\varepsilon + \mu)M = 0 \tag{3.1}$$

$$\frac{dS}{dt} = M - \beta SI - \mu S + \gamma R = 0 \tag{3.2}$$

$$\frac{dE}{dt} = \beta SI - (\sigma + \mu)E = 0 \tag{3.3}$$

$$\frac{dI}{dt} = \sigma E - (\nu + \mu)I = 0 \tag{3.4}$$

$$\frac{dR}{dt} = \nu I - (\mu + \gamma)R = 0 \tag{3.5}$$

From equations 3.1, 3.2, 3.3, 3.4 and 3.5 simultaneously, we obtained the Virus – free equilibrium  
From equation 3.3

$$\sigma E - \mu I - \nu I = 0$$

Since the infection Free State is known to be diseases free, then,  
I = 0, Thus  
This Become

$$\sigma E = 0, \text{ i. e } E = 0$$

From Equation 3.4

$$\nu I - \mu R - \gamma R = 0$$

Since, I=0, the Equation Becomes

$$-(\mu + \gamma)R = 0$$

Thus, R = 0

b) Local stability for Virus-free Equilibrium

We linearize the system of equations given, using the Jacobian matrix approach to obtain:  
Evaluating the Jacobian matrix at the virus – free equilibrium E give

$$J(M, S, E, I, R) \begin{bmatrix} -\varepsilon - \mu & 0 & 0 & 0 & 0 \\ \varepsilon & -\beta I - \mu & 0 & -\beta S & \gamma \\ 0 & \beta I & -\mu - \sigma & \beta S & 0 \\ 0 & 0 & 0 & -\mu - \nu & 0 \\ 0 & 0 & 0 & \nu & -\mu - \gamma \end{bmatrix} = 0$$

We defined the characteristic polynomial equation for the J (E) solve for the eigen valves, to get: After a while, the eigen values  $\lambda_i, i=1, 2, 3, 4, 5$  are given as

$$\lambda_1 = -\mu$$

$$\lambda_2 = -\mu - \gamma$$

$$\lambda_3 = -\xi - \mu$$

$$\lambda_4 = \frac{1}{2(\xi + \mu)} [-2\mu^2 \mu \nu - 2\mu \varepsilon - \mu \sigma - \nu \varepsilon - \sigma \varepsilon + A]$$

$$\lambda_5 = \frac{-1}{2(\xi + \mu)} [2\mu^2 \mu \nu - 2\mu \varepsilon - \mu \sigma - \nu \varepsilon - \sigma \varepsilon + A]$$

Summary, I = 0, E = 0 and R = 0

From Equation 1.0

$$\mu - \varepsilon M - \mu M = 0$$

$$\mu = \varepsilon M + \mu M = 0$$

$$\mu = (\varepsilon + \mu)M$$

This Gives

$$M = \frac{\mu}{\varepsilon + \mu}$$

From Equation 3.1

$$\varepsilon M - \beta SI - \mu S + \gamma R = 0$$

Since I = 0 and R = 0, this reduces to

$$\varepsilon M - \mu S = 0$$

$$\varepsilon M = \mu S$$

$$MS = \frac{\varepsilon}{\mu}$$

But

$$M = \frac{\mu}{\varepsilon + \mu}$$

Thus,

$$\frac{\varepsilon}{\mu} * \frac{\mu}{\varepsilon + \mu} = \frac{\varepsilon}{\varepsilon + \mu}$$

Finally for, virus free equilibrium, the solution set is as follows:

$$\left[ M = \frac{\mu}{\varepsilon + \mu}, S = \frac{\varepsilon}{\varepsilon + \mu}, E = 0, I = 0, R = 0 \right]$$

Where

$$A = \sqrt{\mu^2 v^2 - 2\mu^2 v\sigma + \mu^2 \sigma^2 + 2\mu v^2 \varepsilon - 2\mu v^2 \varepsilon - 4\mu v \varepsilon \sigma + 4\mu \varepsilon \beta \sigma + 2\mu \varepsilon \sigma^2 + v^2 \varepsilon^2 - 2v \varepsilon^2 \sigma + 4\varepsilon^2 \beta \sigma + \varepsilon^2 \sigma^2}$$

From the results above,  $\lambda_1, < 0, \lambda_2 < 0, \lambda_3 < 0,$  and  $\lambda_5 < 0$  provided

$$\mu^2 v^2 - 2\mu^2 v\sigma + \mu^2 \sigma^2 + 2\mu v^2 \varepsilon - 4\mu v \varepsilon \sigma + 4\mu \varepsilon \beta \sigma + 2\mu \varepsilon \sigma^2 + v^2 \varepsilon^2 - 2v \varepsilon^2 \sigma + 4\varepsilon^2 \beta \sigma + \varepsilon^2 \sigma^2 \geq 0$$

That is,

$$\mu^2 v^2 - \mu^2 \sigma^2 + 2\mu v^2 \varepsilon + 4\mu \varepsilon \beta \sigma + 2\mu \varepsilon \sigma^2 + v^2 \varepsilon^2 + 4\varepsilon^2 \beta \sigma + \varepsilon^2 \sigma^2 \geq 2\mu^2 v\sigma + 4\mu v \varepsilon \sigma + 2v \varepsilon^2 \sigma$$

So also,  $\lambda_4$  must be less than zero, i.e.  $\lambda_4 < 0$

Hence,

$$\lambda_4 = \frac{1}{2(\varepsilon + \mu)} (-2\mu^2 - \mu v - 2\mu \varepsilon - \mu \sigma - v \varepsilon - \sigma \varepsilon + A) < 0,$$

Which implies that,

$$\frac{A}{2(\varepsilon + \mu)} < \frac{1}{2(\varepsilon + \mu)} (2\mu^2 - \mu v - 2\mu \varepsilon - \mu \sigma - v \varepsilon - \sigma \varepsilon)$$

That is

$$A < 2\mu^2 - \mu v - 2\mu \varepsilon - \mu \sigma - v \varepsilon - \sigma \varepsilon$$

Finally, the result is

$$Ro = \frac{A}{2\mu^2 + \mu v + 2\mu \varepsilon + \mu \sigma + v \varepsilon + \sigma \varepsilon} < 1$$

A has been defined earlier above.

Where  $R_0$  is the basic reproduction number,

It is imperative to note that the *Basic Reproductive Number*, denoted as  $R_0$ , is an important threshold in modelling of infectious diseases since it tells us if a population is at risk from a disease or not. Thus, whenever  $R_0 < 1$  the new cases (i.e. incidence) of the disease will be on the decrease and the disease will eventually be eliminated.

Based on foregoing, the Basic Reproduction number ( $R_0$ ) for our model is less than unity i.e.

$$Ro = \frac{A}{2\mu^2 + \mu v + 2\mu \varepsilon + \mu \sigma + v \varepsilon + \sigma \varepsilon} < 1$$

Then,  $I(t)$  decreases monotonically to zero as  $t \rightarrow \infty$ . Therefore, the virus-free equilibrium is locally stable.

Local stability for endemic equilibrium we have:

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

This gives

$$\frac{dM}{dt} = \mu - \varepsilon M - \mu M = 0 \tag{3.6}$$

$$\frac{dS}{dt} = \varepsilon M - \beta SI - \mu S + \gamma R = 0 \tag{3.7}$$

$$\frac{dE}{dt} = \beta SI - \sigma E - \mu E = 0 \tag{3.8}$$

$$\frac{dI}{dt} = \sigma E - \sigma E - \mu I - vI \tag{3.9}$$

$$\frac{dR}{dt} vI - \mu R - \gamma R = 0 \tag{3.10}$$

In this scenario, the state is assumed to be virus endemic,  $I > 0$

From equation 3.6

$$\mu - \varepsilon M - \mu M = 0$$

$$\mu = (\varepsilon + \mu)M = 0$$

$$M = \frac{\mu}{\varepsilon + \mu} \tag{3.11}$$

From equation 3.9

$$\sigma E - \mu I vI = 0$$

i.e.

$$\sigma E = (\mu + v)I$$

Hence,

$$E = \frac{\mu + v}{\sigma} I \tag{3.12}$$

From equation 3.10

$$vI - \mu R - \gamma R = 0$$

$$vI = (\mu + \gamma)R$$

$$R = \left( \frac{v}{\mu + \gamma} \right) I \tag{3.13}$$

From Equation 3.8

$$\beta SI - \sigma E - \mu E = 0$$

$$\beta SI = (\sigma + \mu)E$$

$$S = \left( \frac{\sigma + \mu}{\beta I} \right) E$$

But

$$E = \left( \frac{\mu + v}{\sigma} \right) I$$

Thus,

$$S = \left( \frac{\sigma + \mu}{\beta I} \right) E$$

$$S = \left(\frac{\sigma + \mu}{\beta I}\right) \left(\frac{\mu + \nu}{\sigma}\right) I = \frac{(\delta + \mu)(\mu + \nu)}{\beta \sigma} \tag{3.14}$$

Substitute for M, E, R and S in Equation 3.7

$$\epsilon M - \beta SI - \mu S + \gamma R = 0$$

That is:

$$\epsilon * \frac{\mu}{\epsilon + \mu} - \beta * \frac{(\delta + \mu)(\mu + \nu)}{\beta \sigma} * I - \mu * \frac{(\delta + \mu)(\mu + \nu)}{\beta \sigma} + \gamma * \left(\frac{\nu}{\mu + \gamma}\right) I = 0$$

Solving for I yields:

$$I = \frac{-\mu(\mu + \gamma)(\mu^3 + \mu^2\nu + \mu^2\epsilon + \mu^2\sigma + \mu\nu\epsilon + \mu\nu\sigma + \mu\epsilon\sigma + \nu\epsilon\sigma - \epsilon\beta\sigma)}{\beta(\mu^3 + \mu^2\nu + \mu^2\epsilon + \mu^2\gamma + \mu^2\sigma + \mu\nu\epsilon + \mu\nu\gamma + \mu\nu\sigma + \mu\epsilon\gamma + \mu\epsilon\sigma + \mu\gamma\sigma + \nu\epsilon\sigma + \epsilon\gamma\sigma)}$$

Consequently,

$$R = \left(\frac{\nu}{\mu + \gamma}\right) I$$

The above yields,

$$\frac{-\nu(\mu + \gamma)(\mu^3 + \mu^2\nu + \mu^2\epsilon + \mu^2\sigma + \mu\nu\sigma + \mu\epsilon\sigma + \nu\epsilon\sigma - \epsilon\beta\sigma)}{\beta(\mu^3 + \mu^2\nu + \mu^2\epsilon + \mu^2\gamma + \mu^2\sigma + \mu\nu\epsilon + \mu\nu\gamma + \mu\nu\sigma + \mu\epsilon\sigma + \mu\gamma\sigma + \nu\epsilon\sigma + \epsilon\gamma\sigma)}$$

Also,

$$E = \left(\frac{\mu + \nu}{\sigma}\right) I = \frac{-\nu(\mu + \gamma)(\mu^3 + \mu^2\nu + \mu^2\epsilon + \mu^2\sigma + \mu\nu\sigma + \mu\epsilon\sigma + \nu\epsilon\sigma - \epsilon\beta\sigma)}{\beta\sigma(\mu^3 + \mu^2\nu + \mu^2\epsilon + \mu^2\gamma + \mu^2\sigma + \mu\nu\epsilon + \mu\nu\gamma + \mu\nu\sigma + \mu\epsilon\sigma + \mu\gamma\sigma + \nu\epsilon\sigma + \epsilon\gamma\sigma)}$$

For mathematical acceptability,  $(M_E, S_E, E_E, I_E, R_E) > 0$

Thus,

$$I_E = \frac{-\nu(\mu + \gamma)(\mu^3 + \mu^2\nu + \mu^2\epsilon + \mu^2\sigma + \mu\nu\sigma + \mu\epsilon\sigma + \nu\epsilon\sigma - \epsilon\beta\sigma)}{\beta(\mu^3 + \mu^2\nu + \mu^2\epsilon + \mu^2\gamma + \mu^2\sigma + \mu\nu\epsilon + \mu\nu\gamma + \mu\nu\sigma + \mu\epsilon\sigma + \mu\gamma\sigma + \nu\epsilon\sigma + \epsilon\gamma\sigma)} > 0$$

Let,

$$A = -(\mu + \nu)(\mu^3 + \mu^2\nu + \mu^2\epsilon + \mu^2\sigma + \mu\nu\epsilon + \mu\nu\sigma + \mu\epsilon\sigma + \nu\epsilon\sigma)$$

$$B = \epsilon\beta\sigma \text{ and}$$

$$C = (\mu^3 + \mu^2\nu + \mu^2\epsilon + \mu^2\gamma + \mu^2\sigma + \mu\nu\epsilon + \mu\nu\gamma + \mu\nu\sigma + \mu\epsilon\sigma + \mu\gamma\sigma + \nu\epsilon\sigma + \epsilon\gamma\sigma)$$

Hence,

$$\begin{aligned} I_E &= \frac{(\mu + \gamma)(A - B)}{B * C} > 0 \\ &= -(\mu + \gamma)(A - B) > 0 \\ &= -(\mu + \gamma)A + B(\mu + \gamma) > 0 \\ &= B(\mu + \gamma) > (\mu + \gamma)A \end{aligned}$$

Dividing through by A, we then have,

$$R_0 = \frac{B}{A} > 1$$

More elaborately, we have:

$$R_0 = \frac{\epsilon\beta\sigma}{\mu^3 + \mu^2\nu + \mu^2\epsilon + \mu^2\sigma + \mu\nu\epsilon + \mu\nu\sigma + \mu\epsilon\sigma + \nu\epsilon\sigma} > 1$$



If  $\beta = 64.5$ ,  $\nu = 36$ ,  $\varepsilon = 13$ ,  $\delta = 91$ ,  $\mu = 0.041$  are all parameter for a period of one year, then we have the following expression:

$$R_0 = \frac{13 * 64.5 * 91}{(0.041)^3 + (0.041)^2 * 36 + (0.041)^2 * 13 + (0.041)^2 * 91 + (0.041)(36)(13) + (0.041)(36)(91) + (0.041)(91)(13) + (36)(13)(91)} > 1$$

$$R_0 = 2.944076535 > 1$$

If  $R_0 > 1$  then  $I(t)$  increases and reaches its maximum and reduces as  $R_0 \rightarrow \infty$ . When the number of children infected increases in this state, it is called the epidemic state. In the long run, the whole population become susceptible if  $R_0 > 1$

system of first – order ordinary differential equation (ODE). So, in computing  $y(t_n)$ , it needs only the solution at the immediately preceding time point,  $y(t_{n-1})$ . In general, ode45 is the best function to apply as a first try for most problems involving systems of first order ODES. Runge kutta of order four is also used in plotting the graphs; it's a powerful and popular method because of its accuracy and stability. Also, its simplicity and stability make it one of the most widely used numerical algorithms for stiff and non-stiff equations, while it converges faster than that of order two or three.

#### IV. NUMERICAL SOLUTION AND SIMULATION

The SEIRS model was solved numerically using Runge – Kutta method. We adopted Matlab ode45 program, which is based on an explicit Runge Kutta (4, 5) formula. It is a one-step solver used in solving a

Table 4.1: Simulating the MSEIRS model using the following parameter values

Parameters	$V$	$b_0$	$b_1$	$\delta$	$\phi$	$\mu$	$\gamma$	$\zeta$	$\beta$	$R_0$
MSEIRS (Virus free State)	36	50	0.14	91	0.15	0.041	1.8	13	64.5	0.9515728172
MSEIRS (Epidemic State)	36	20	0.20	91	0.15	0.041	1.8	13	27	2.944076535

These are the parameters used in plotting the graphs: although some of it changes are due to the fact that they are the major factors that are determining the

situations of the environment that is, if it is of the virus – free and endemic state.

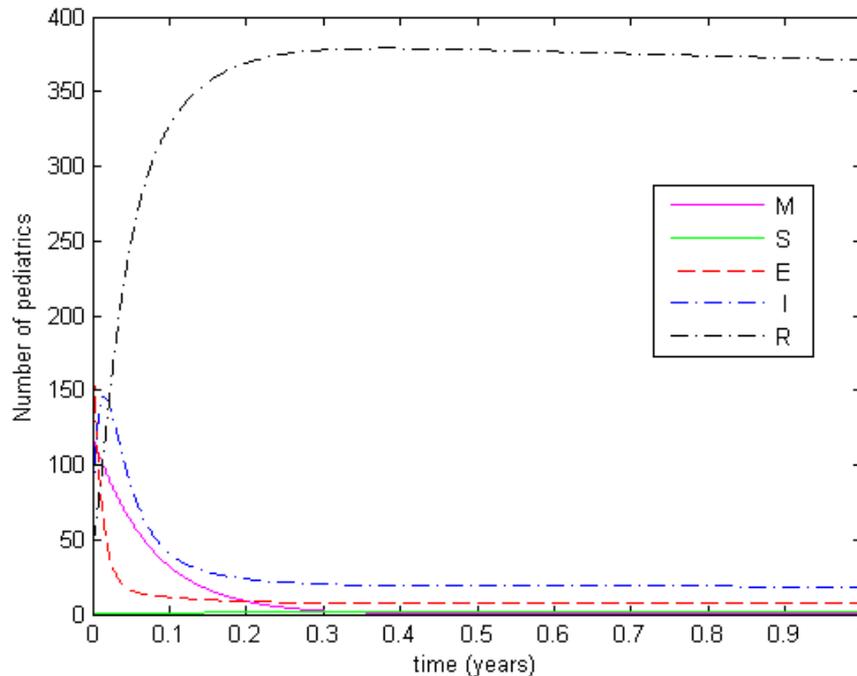


Figure 4.1(a): Graphical representation of MSEIRS model between the space of one year with  $M_0 = 120$ ,  $S_0 = 100$ ,  $E_0 = 82$ ,  $I_0 = 67$ ,  $R_0 = 46$

Figure 4.1 shows the graphical representation of MSEIRS model between the space of one year. In these model, we assume newborn infants of immune mothers that are protected by maternal antibodies. We then introduce a group of M of children that are born

completely protected. According to the graph above, we assume the fraction of newborns that are protected is equal to the fraction of the general population that have temporary immunity after recovering from infection. The protected children become susceptible.

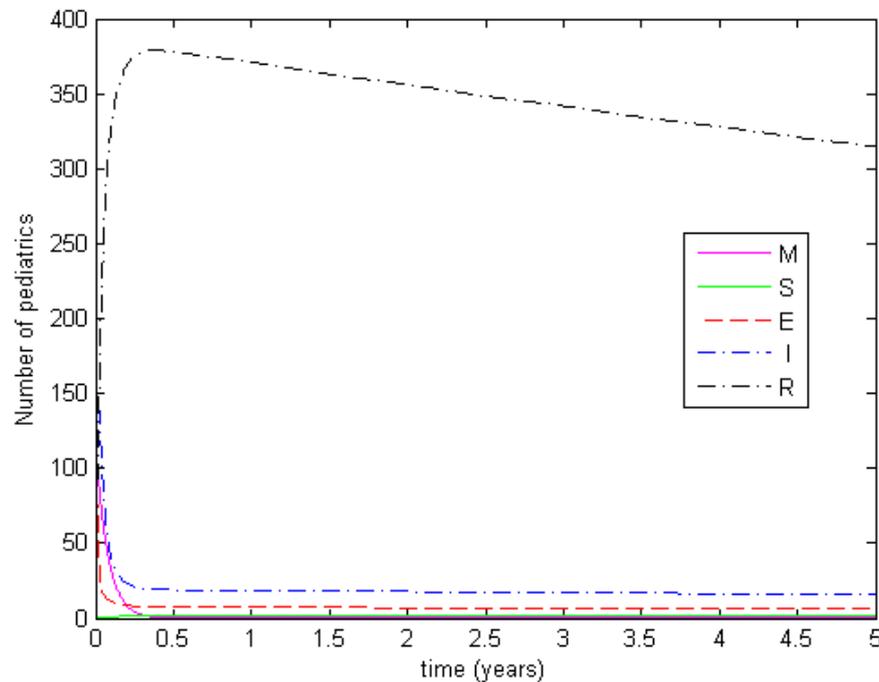


Figure 4.2: Graphical representation of MSEIRS model between the space of five years

Figure 4.2 shows the graphical representation of MSEIRS model between the space of five years. In these model, we assume newborn infants of immune mothers that are protected by maternal antibodies. We then introduce a group of M of children that are born completely protected. According to the graph above, we assume the fraction of newborns that are protected is equal to the fraction of the general population that have temporary immunity after recovering from infection. The protected children become susceptible.

## V. CONCLUSION

To conclude, while this model would benefit against real world data, in its present form it has been shown to be useful in three areas: providing a systems-level view, exposing weaknesses and dependencies and evaluating new technologies. With more data this sort of model could provide valuable insight and prediction for the entire LRTI disease.

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