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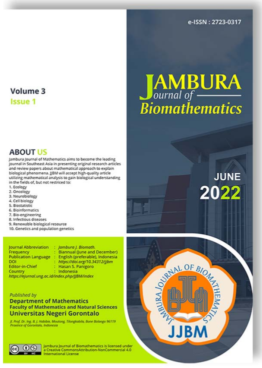

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Optimal Control and Model Analysis of The Spread of Pneumonia in Toddlers in East Java-Indonesia Using The Pontryagin's Minimum Principle

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ABSTRACT. *Pneumonia is a type of acute respiratory infection (ARI) that attacks the lungs and is caused by various microorganisms, such as bacteria, viruses, parasites, fungi, exposure to chemicals, or physical damage to the lungs. Pneumonia is included in the list of 10 diseases with the highest number of cases according to the Indonesian Ministry of Health reported in April 2023. Pneumonia is the biggest cause of death in toddlers aged 12-59 months, reaching 12.5%. Therefore, to reduce the spread of pneumonia, this research will discuss providing optimal control using the mathematical model of SEIR (Susceptible-Exposed-Infected-Recovered). The model used is a pneumonia spreading model with implementing control in the form of first stage treatment and second stage treatment. The results of the stability analysis show that at the disease-free equilibrium point and the endemic equilibrium point, the system is stable respectively. Based on controllability analysis, it is obtained that the system is controlled so that the system can be controlled. In addition, based on the results of the analysis of the optimal control problem with Pontryagin's Minimum Principle simulated with Runge Kutta order 4, it shows that the first stage of treatment control* (u_1) *and the second stage of treatment* (u_2) *are very effective in reducing the number of individuals infected with mild pneumonia and severe pneumonia respectively.*

1. Introduction

According to the World Health Organization, pneumonia is a form of acute respiratory infection (ARI) that attacks the lungs. This disease is caused by various microorganisms, such as viruses, fungi and bacteria, which cause fluid to build up in the lungs and can inhibit the breathing process. Pneumonia tends to occur frequently in children under five and is the main cause of death in the children. Based on a report from the Indonesian Republic Ministry of Health in April 2023, pneumonia is included in the list of 10 diseases with the highest number of cases. Pneumonia is the biggest cause of death in toddlers aged 12-59 months, reaching 12.5%. In 2022, East Java will be the province with the second highest number of pneumonia cases discovered in toddlers after West Java, reaching 92,128 cases, which is a significant increase from the previous year [1].

Pneumonia is an infection or acute inflammation of the lung tissue caused by various microorganisms, such as bacteria, viruses, parasites, fungi, exposure to chemicals, or physical damage to the lungs [2–6]. Infected individuals experience a latent stage where the bacteria or virus remains in the body without showing any symptoms. Once past the latent stage, infected individuals can become infectious and begin to exhibit flu-like symptoms such as headache, fever, cough, shortness of breath, and other common flu symptoms [7–9]. The duration of infection varies between three weeks to a month, depending on the health condition of the infected individual. After recovery, a person acquires temporary immunity against the disease for a certain time periods [10]. According to the Integrated Management of Sick Toddlers, pneumonia in toddlers is classified into three types, including non-pneumonia, pneumonia and severe pneumonia. In non-pneumonia cases, there are no signs of pneumonia in the body. Meanwhile, in cases of pneumonia, the symptoms shown are rapid breathing without any inward pulling of the chest wall. In cases of severe pneumonia, the symptoms shown get worse accompanied by indrawing of the chest wall [11, 12].

Pneumonia can be prevented through vaccination which involves administering antigenic materials to stimulate the formation of active immunity against certain diseases, which aims to prevent or reduce the impact of infection by organisms on a person's body [7, 13, 14]. In addition, treatment for pneumonia varies depending on the type of infection. In most cases, treatment involves administering antibiotics such as amoxicillin. For mild viral pneumonia, special treatment is usually not given because it tends to heal by itself. The cornerstone of pneumonia treatment involves maintaining oxygenation levels, ensuring adequate hydration, as well as adequate rest [15, 16].

The application of mathematical models in efforts to control pneumonia has been widely applied in previous research. For example, in 2021, Side, et al has built a mathematical model that describes the spreading up of pneumonia in toddlers by using 4

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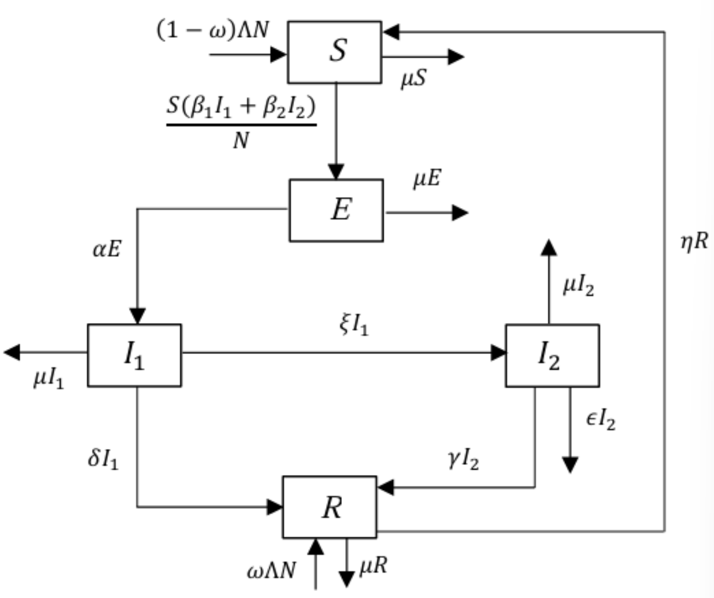


Figure 1. The compartment diagram of a mathematical model of the spread of pneumonia in toddlers in east Java.

Table 1. Initial values of pneumonia distribution model variables

Variable	Description	Initial Value	Calculation Method
s_0	Initial number of susceptible individuals	0.7423	$1-(e_0+i_{1,0}+i_{2,0}+r_0)$
e_0	Initial number of exposed individuals	0.0482	Avg. estimated number / Avg. toddler pop.
$i_{1,0}$	Initial number of mildly infected individuals	0.0274	Avg. mild cases / Avg. toddler pop.
$i_{2,0}$	Initial number of severely infected individuals	0.0005	Avg. severe cases / Avg. toddler pop.
r_0	Initial number of recovered individuals	0.1816	Avg. recovered toddlers / Avg. toddler pop.

subpopulations, namely S (Susceptible) for susceptible individuals, E (Exposed) for individuals who are detected but not yet infected, I (Infected) for individuals who are infected and can transmit the disease, as well as R (Recovered) for individuals who recover through vaccination. In this study, an analysis of the mathematical model was also carried out and determined the minimum proportion of vaccinations [17]. Then, research on the spreading up of pneumonia was also developed by Imran, et al. [17]. This research discusses the SEIPR mathematical model, namely S (Susceptible) is a healthy individual but is susceptible to pneumonia, E (Exposed) is an individual exposed to pneumonia, I (Infected) for an infected individual, P (Treatment) is an infected individual who given treatment, and R (Recovered) for individuals who recover from pneumonia. The development of this mathematical model was carried out by taking into account the aspects such as immunization and treatment, as a step to reduce the spread of pneumonia [18]. Apart from that, Aldila, et al. [19] also modified the pneumonia spread mathematical model by including 5 subpopulations, such as the susceptible population (Susceptible), the exposed population (Exposed), the population infected with pneumonia in the first stage (Infected-1), the population infected with pneumonia in the second stage (Infected-2), and the population recovered (Recovered). The mathematical model resulted from this research was then validated towards data from the Jakarta City Health Department. Apart from that, this research also discusses sensitivity analysis and optimal control in the form of treatment which is analyzed using Pontryagin's Maximum Principle [19].

In this paper, we refer to the mathematical model written by Aldila et al. [19] and apply it to cases of pneumonia in toddlers that have occurred in East Java province by referring to data from the East Java Provincial Health Office. The development of this mathematical model was carried out by adapting the case to toddlers that occurred in East Java, i.e. by adding assumptions, parameters, and optimal controls to control the spread of pneumonia among toddlers in East Java Province. The controls used include first stage treatment and second stage treatment for toddlers. A stability analysis was carried out around the equilibrium point. The optimal control further is determined by using Pontryagin's Minimum Principle. Following by numerical simulation of the optimal control problem was carried out using the 4th order Runge Kutta method using Matlab software.

2. Mathematical Model

We then formulated a mathematical model for the problem of pneumonia spread among toddlers by referring to the articles [19–26]. The mathematical model is divided into five subpopulations, namely the subpopulation susceptible to infection with pneumonia $(S(t))$, the subpopulation exposed and infected with pneumonia but unable to transmit it $(E(t))$, the subpopulation infected with the first stage of pneumonia $(I_1(t))$, subpopulation infected with second stage pneumonia $(I_2(t))$, and subpopulation who recovered from pneumonia $(R(t))$. The total population $(N(t))$ denoted as $N(t)$ and expressed as $N(t) =$

Table 2. Initial values of pneumonia spreading up model parameters

Parameter	Description	Value	Unit	Data Source
Λ	Recruitment rate	0.0004953039	1/day	East Java Province Health Profile 2019-2022
μ	Natural death rate	0.0000383715	1/day	Central Bureau of Statistics
ϵ	Death rate due to pneumonia	0.0000010076	1/day	East Java Province Health Profile 2019-2022
α	Transition rate from e to i_1	0.3333333333	1/day	Centers for Disease Control and Prevention
δ	Recovery rate for i_1	0.047619	1/day	National Heart, Blood, and Lung Institute
γ	Recovery rate for i_2	0.005555	1/day	National Heart, Blood, and Lung Institute
ω	Proportion of individuals fully vaccinated	0.8179	-	East Java Province Health Profile 2019-2022
β_1	Rate of mild pneumonia infection	0.0000000131	1/day	East Java Province Health Profile 2019-2022
β_2	Rate of severe pneumonia infection	0.0000000015	1/day	East Java Province Health Profile 2019-2022
ξ	Rate of disease progression from i_1 to i_2	0.5	1/day	Integrated Management of Sick Toddlers
η	Rate of transmission from i_1 to i_2	0.0241	1/day	[11]

$S(t) + E(t) + I_1(t) + I_2(t) + R(t)$. Further, the control provided in this model is in the form of treatment given to individual I_1. However, In our research, the development model of Adila's mathematical model lies in Recovered individuals $(R(t))$ which not only come from Infected-1 $(I_1(t))$ and Infected-2 $(I_2(t))$ individuals, but also from individuals who have received the full dose of vaccine. Apart from that, the mathematical model was also carried out by adding assumptions and controls including first stage treatment control given to the Infected-1 $(I_1(t))$ subpopulation and second stage treatment control given to the Infected-2 $(I_2(t))$ subpopulation. We therefore assume and restrict that the development of mathematical model for the spread of pneumonia are as follows.

1. The mathematical model is formed from 5 subpopulations that interact with each other, namely the susceptible subpopulation or Susceptible (S), the exposed subpopulation or Exposed (E), the subpopulation infected with mild pneumonia or Infected-1 (I_1), the subpopulation infected with severe pneumonia or Infected-2 (I_2), and the subpopulation recovered or Recovered (R).
2. The population is closed.
3. Pneumonia is caused by bacteria.
4. The death rate consists of natural deaths for all subpopulations and denoted by the parameter μ and deaths due to pneumonia for subpopulation I_2 and denoted by the parameter ϵ.
5. Individuals who have not received the vaccine at all or have not received the complete dose of the vaccine will be included as susceptible individuals and denoted by notation of S.
6. Individuals who have received the complete vaccine can be included in the group of recovered individuals and denoted by R.
7. The interaction between individual S and individual I_1 or I_2, and if the vaccine dose for individual S has not been fulfilled, this will cause individuals in compartment S to move to compartment E.
8. When individual E experiences a weakened immune system, the bacteria that cause pneumonia become active, causing individual E to become infected with mild pneumonia denoted by I_1.
9. Individuals who have recovered, denoted by R, can revert to being susceptible, denoted by S.
10. The first stage of treatment for individual I_1 involves antibiotics. Meanwhile, the second stage of treatment for individual I_2 involves hospitalization with oxygen, fever-reducing medication for patients with high temperatures, and airway clearance.
11. Individual I_1 can change into individual I_2 with a disease progression rate of ξ.
12. The natural death rate denoted by μ, while the recovery rate is indicated by the symbol of η. In this case, it is assumed that the recovery rate at R is the same as the death rate at S.

So that, the mathematical model of the spread up of pneumonia in toddlers in East-Java can be seen in Figure 1.

Based on the compartment diagram above, the equation can be formulated as follows.

$$\begin{aligned}
\frac{dS}{dt} &= (1-\omega)\Lambda N - \frac{S(\beta_1 I_1 + \beta_2 I_2)}{N} + \eta R - \mu S, \\
\frac{dE}{dt} &= \frac{S(\beta_1 I_1 + \beta_2 I_2)}{N} - \mu E - \alpha E, \\
\frac{dI_1}{dt} &= \alpha E - \delta I_1 - \xi I_1 - \mu I_1, \\
\frac{dI_2}{dt} &= \xi I_1 - \mu I_2 - \epsilon I_2 - \gamma I_2, \\
\frac{dR}{dt} &= \omega \Lambda N + \delta I_1 + \gamma I_2 - \mu R - \eta R.
\end{aligned} \tag{1}$$

Based on the eq. (1), the mathematical model will further be transformed into the form of non-dimensional. Each population can be expressed as $s = \frac{S}{N}, e = \frac{E}{N}, i_1 = \frac{I_1}{N}, i_2 = \frac{I_2}{N}, r = \frac{R}{N}$. The model mathematics model can be written as follows.

$$\begin{aligned}
\frac{ds}{dt} &= (1-\omega)\Lambda - s(\beta_1 i_1 + \beta_2 i_2) + \eta r - \mu s, \\
\frac{de}{dt} &= s(\beta_1 i_1 + \beta_2 i_2) - \mu e - \alpha e,
\end{aligned}$$

$$\frac{di_1}{dt} = \alpha e - \delta i_1 - \xi i_1 - \mu i_1, \quad (2)$$
$$\frac{di_2}{dt} = \xi i_1 - \mu i_2 - \epsilon i_2 - \gamma i_2,$$
$$\frac{dr}{dt} = \omega\Lambda + \delta i_1 + \gamma i_2 - \mu r - \eta r.$$

Furthermore, the data to complete the numerical calculations in eq. (2) was obtained from data collected from the East Java health service from 2019 to 2022 [1, 2, 27, 28]. This data will be processed to obtain parameter values and initial values for the model. The following are the initial values and parameter values for the mathematical model for the spread of pneumonia in toddlers.

3. Analysis Solutions

In this section, we are looking for analytical solutions from equilibrium point, linearization, stability, and basic reproduction number.

3.1. Equilibrium Point

The mathematical model of the spreading up of pneumonia disease in toddlers satisfies the equilibrium state when $\frac{ds}{dt} = \frac{de}{dt} = \frac{di_1}{dt} = \frac{di_2}{dt} = \frac{dr}{dt} = 0$. The disease-free equilibrium point occurs when the epidemiological model reaches equilibrium state without the spread of infectious disease. However, the endemic equilibrium point is when the model is in an equilibrium state and the spread of disease is occurring [29]. In disease-free conditions, there are no susceptible subpopulations, subpopulations infected with mild pneumonia, and subpopulations infected with severe pneumonia, so that the values $e = 0, i_1 = 0$, and $i_2 = 0$. Further, by substituting the values $e = 0, i_1 = 0$, and $i_2 = 0$ to the eq. (2), we obtain:

$$E_0 = (s^0, e^0, i_1^0, i_2^0, r^0),$$
$$E_0 = \left(\frac{(\mu+\eta)(1-\omega)\Lambda + \eta\omega\Lambda}{\mu(\mu+\eta)}, 0, 0, 0, \frac{\omega\Lambda}{\mu+\eta}\right).$$

In endemic conditions, $e \neq 0, i_1 \neq 0, i_2 \neq 0$, so the endemic equilibrium point can be written as follows,

$$E_1 = (s^*, e^*, i_1^*, i_2^*, r^*)$$

where:

$$s^* = \frac{(1-\omega)\Lambda + \eta r}{\beta_1 i_1 + \beta_2 i_2 + \mu},$$
$$e^* = \frac{s(\beta_1 i_1 + \beta_2 i_2)}{\mu + \alpha},$$
$$i_1^* = \frac{\alpha e}{\delta + \xi + \mu},$$
$$i_2^* = \frac{\xi i_1}{\mu + \epsilon + \gamma},$$
$$r^* = \frac{\omega\Lambda + \delta i_1 + \gamma i_2}{\mu + \eta}.$$

3.2. Linearization

The mathematical model of the spreading up of pneumonia disease in toddlers is a non-linear mathematical model. It will therefore be analyzed the stability of the system. A linearization process is required first. The linearization process in this pneumonia spreading up in toddlers model uses a Taylor series expansion around the disease-free equilibrium point and by assuming $s - s^0 = v, e - e^0 = w, i_1 - i_1^0 = x, i_2 - i_2^0 = y, r - r^0 = z$, the eq. (2) can be written as follows:

$$\begin{pmatrix} \frac{dv}{dt} \\ \frac{dw}{dt} \\ \frac{dx}{dt} \\ \frac{dy}{dt} \\ \frac{dz}{dt} \end{pmatrix} = J(s^0, e^0, i_1^0, i_2^0, r^0) \begin{pmatrix} v \\ w \\ x \\ y \\ z \end{pmatrix}$$

where J is the Jacobian matrix, i.e.

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial s} & \frac{\partial f_1}{\partial e} & \frac{\partial f_1}{\partial i_1} & \frac{\partial f_1}{\partial i_2} & \frac{\partial f_1}{\partial r} \\ \frac{\partial f_2}{\partial s} & \frac{\partial f_2}{\partial e} & \frac{\partial f_2}{\partial i_1} & \frac{\partial f_2}{\partial i_2} & \frac{\partial f_2}{\partial r} \\ \frac{\partial f_3}{\partial s} & \frac{\partial f_3}{\partial e} & \frac{\partial f_3}{\partial i_1} & \frac{\partial f_3}{\partial i_2} & \frac{\partial f_3}{\partial r} \\ \frac{\partial f_4}{\partial s} & \frac{\partial f_4}{\partial e} & \frac{\partial f_4}{\partial i_1} & \frac{\partial f_4}{\partial i_2} & \frac{\partial f_4}{\partial r} \\ \frac{\partial f_5}{\partial s} & \frac{\partial f_5}{\partial e} & \frac{\partial f_5}{\partial i_1} & \frac{\partial f_5}{\partial i_2} & \frac{\partial f_5}{\partial r} \end{pmatrix}$$

Further, the Jacobian matrix can be expressed by:

$$J = \begin{pmatrix} j_1 & 0 & -\beta_1 s & -\beta_2 s & \eta \\ j_2 & j_3 & \beta_1 s & \beta_2 s & 0 \\ 0 & \alpha & j_4 & 0 & 0 \\ 0 & 0 & \xi & j_5 & 0 \\ 0 & 0 & \delta & \gamma & j_6 \end{pmatrix},$$
$$j_1 = -(\beta_1 i_1 + \beta_2 i_2 + \mu),$$
$$j_2 = \beta_1 i_1 + \beta_2 i_2,$$
$$j_3 = -(\mu + \alpha),$$
$$j_4 = -(\delta + \xi + \mu),$$
$$j_5 = -(\mu + \epsilon + \gamma),$$
$$j_6 = -(\mu + \eta).$$

3.3. Stability

Following the linearization process, the Jacobi matrix is obtained is then used for stability analysis. Pneumonia disease-free stability is determined by substituting the disease-free equilibrium point (E_0) in the Jacobi matrix, then calculating the characteristic equation using $|J(E_0) - \lambda I| = 0$ to obtain eigenvalues. We further obtain the eigenvalues as follows:

$$\lambda_1 = -0.000038;\ \lambda_2 = -0.024138;\ \lambda_3 = -0.547657;$$
$$\lambda_4 = -0.005595;\ \lambda_5 = -0.333372.$$

Based on the eigenvalues, it can be seen that all eigenvalues are negative. This indicates that the system is asymptotically stable at the disease-free equilibrium point. Further, the endemic stability is calculated by substituting the endemic equilibrium point (E_1) in the Jacobi matrix to obtain the eigenvalues by using $|J(E_1) - \lambda I| = 0$. We further obtain the eigenvalue as follows:

$$\lambda_1 = -0.547660;\ \lambda_2 = -0.333369;\ \lambda_3 = -0.000038;$$

$$\lambda_4 = -0.005595;\ \lambda_5 = -0.024138.$$

Based on these eigenvalues, it can be seen that all eigenvalues are negative. This shows that the system is asymptotically stable as well at the endemic equilibrium point.

3.4. Basic Reproduction Number

In the eq. (2), the population that causes infection is the population e, i_1, and i_2 so the system of differential equation can be written as follows:

$$\begin{aligned}\frac{de}{dt} &= s\beta_1 i_1 + s\beta_2 i_2 - (\mu e + \alpha e),\\ \frac{di_1}{dt} &= \alpha e - (\delta i_1 + \xi i_1 + \mu i_1),\\ \frac{di_2}{dt} &= \xi i_1 - (\mu i_2 + \epsilon i_2 + \gamma i_2).\end{aligned}$$

Further, we introduce $\mathcal{F}$ matrix, which is the matrix of new infections in the population, and it expresses as follows,

$$\mathcal{F} = \begin{pmatrix} s\beta_1 i_1 + s\beta_2 i_2 \\ 0 \\ 0 \end{pmatrix}.$$

The matrix that describes the movement of individuals from one infected class to another class in a time period denoted by $\mathcal{V}$ as follows,

$$\mathcal{V} = \begin{pmatrix} \mu e + \alpha e \\ -\alpha e + \delta i_1 + \xi i_1 + \mu i_1 \\ -\xi i_1 + \gamma i_2 + \epsilon i_2 + \mu i_2 \end{pmatrix}.$$

The both the matrices of $\mathcal{F}$ and $\mathcal{V}$ are respectively derived towards e, i_1, and i_2, then the matrices of F and V can be written respectively as follows:

$$F = \begin{pmatrix} 0 & s\beta_1 & s\beta_2 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} \mu+\alpha & 0 & 0 \\ -\alpha & \delta+\xi+\mu & 0 \\ 0 & -\xi & \gamma+\epsilon+\mu \end{pmatrix}.$$

Then, the value of the disease-free equilibrium point

$$\begin{aligned}E_0 &= (s^0, 0, 0, 0, r^0),\\ &= \left(\frac{(\mu+\eta)(1-\omega)\Lambda + \eta\omega\Lambda}{\mu(\mu+\eta)}, 0, 0, 0, \frac{\omega\Lambda}{\mu+\eta}\right).\end{aligned}$$

is substituted into the F matrix obtained

$$F = \begin{pmatrix} 0 & \frac{\beta_1(\mu+\eta)(1-\omega)\Lambda + \beta_1\eta\omega\Lambda}{\mu(\mu+\eta)} & \frac{\beta_2(\mu+\eta)(1-\omega)\Lambda + \beta_2\eta\omega\Lambda}{\mu(\mu+\eta)} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

Further, by employing multiplication between the matrices F and V^{-1}, it can be obtained

$$FV^{-1} = \begin{pmatrix} \frac{1}{(\mu+\alpha)} & 0 & 0 \\ \frac{\alpha}{(\mu+\alpha)(\delta+\xi+\mu)} & \frac{1}{(\delta+\xi+\mu)} & 0 \\ k_1 & k_2 & \frac{1}{\gamma+\epsilon+\mu} \end{pmatrix},$$

$$\begin{aligned}k_1 &= \frac{\alpha\xi}{(\mu+\alpha)(\delta+\xi+\mu)(\gamma+\epsilon+\mu)},\\ k_2 &= \frac{\xi}{(\delta+\xi+\mu)(\gamma+\epsilon+\mu)}.\end{aligned}$$

By using the next generation matrix method, the spectral radius of the matrix FV^{-1} is obtained or in other words the basic reproduction number (R_0), which is follows.

$$R_0 = \rho(FV^{-1})$$

Or it written as

$$\begin{aligned}R_0 = &\frac{\alpha\beta_1\left((\mu+\eta)(1-\omega)\Lambda + \alpha\beta_1\eta\omega\Lambda\right)(\gamma+\epsilon+\mu)}{(\mu+\eta)(\mu+\alpha)(\delta+\xi+\mu)(\gamma+\epsilon+\mu)}\\ &+ \frac{\alpha\xi\beta_2\left((\mu+\eta)(1-\omega)\Lambda + \alpha\xi\beta_2\eta\omega\Lambda\right)\mu}{(\mu+\eta)(\mu+\alpha)(\delta+\xi+\mu)(\gamma+\epsilon+\mu)}\end{aligned} \quad (3)$$

By substituting the parameter values in Table 2, the basic reproduction number shows a value of $R_0 < 1$. This suggests that the spread of pneumonia is limited because each infected individual only transmits the disease to a few other individuals and will likely lead to a decrease in infection cases.

3.5. Optimal Control

3.5. Mathematical Model of The Spreading Up of Pneumonia in Toddlers when Controls are Included

Below is the compartment diagram of the mathematical model of the spreading up of pneumonia disease in toddlers when the first-stage treatment control (u_1) given to the population infected with mild pneumonia (I_1) and the second-stage treatment control (u_2) given to the population infected with severe pneumonia (I_2).

Based on the compartment diagram in Figure 2, a mathematical model of the spreading up of pneumonia disease in toddlers with controls u_1 and u_2 is expressed as follows:

$$\begin{aligned}\frac{dS}{dt} &= (1-\omega)\Lambda N - \frac{S(\beta_1 I_1 + \beta_2 I_2)}{N} + \eta R - \mu S,\\ \frac{dE}{dt} &= \frac{S(\beta_1 I_1 + \beta_2 I_2)}{N} - (\mu+\alpha)E,\\ \frac{dI_1}{dt} &= \alpha E - (\delta+u_1)I_1 - \xi I_1 - \mu I_1,\\ \frac{dI_2}{dt} &= \xi I_1 - (\gamma+u_2)I_2 - \epsilon I_2 - \mu I_2,\\ \frac{dR}{dt} &= \omega\Lambda N + (\delta+u_1)I_1 + (\gamma+u_2)I_2 - (\mu+\eta)R.\end{aligned} \quad (4)$$

This eq. (4) further is transformed into the form of nondimensional by introducing the variables of $s = \frac{S}{N}, e = \frac{E}{N}, i_1 = \frac{I_1}{N}, i_2 = \frac{I_2}{N}, r = \frac{R}{N}$ so the eq. (4) can be expressed as follows,

$$\begin{aligned}\frac{ds}{dt} &= (1-\omega)\Lambda - s(\beta_1 i_1 + \beta_2 i_2) + \eta r - \mu s,\\ \frac{de}{dt} &= s(\beta_1 i_1 + \beta_2 i_2) - (\mu+\alpha)e,\\ \frac{di_1}{dt} &= \alpha e - (\delta+u_1)i_1 - \xi i_1 - \mu i_1,\\ \frac{di_2}{dt} &= \xi i_1 - (\gamma+u_2)i_2 - \epsilon i_2 - \mu i_2,\\ \frac{dr}{dt} &= \omega\Lambda + (\delta+u_1)i_1 + (\gamma+u_2)i_2 - (\mu+\eta)r,\end{aligned} \quad (5)$$

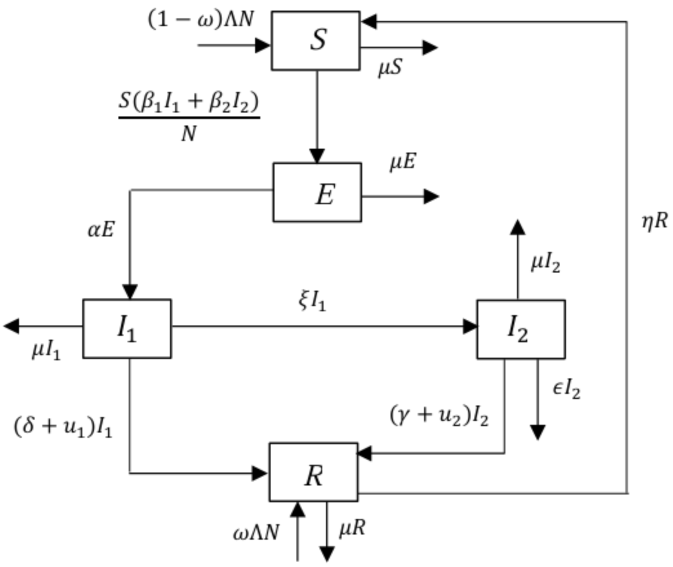


Figure 2. The compartment diagram of pneumonia spreading up in toddlers when controls are included.

with initial condition $s(0) = s_0, e(0) = e_0, i_1(0) = i_{10}, i_2(0) = i_{20}, r(0) = r_0$, and the final time limits on the control variables u_1 and u_2 are as follows.

$$0 \le u_1(t) \le 1, \quad 0 \le u_2(t) \le 1, \quad t \in [0, t_f]$$

3.5. *Controllability*

Based on the results of the Jacobi matrix calculation, the matrix A is obtained, i.e.

$$A = \begin{pmatrix} j_1 & 0 & -\beta_1 s & -\beta_2 s & \eta \\ j_2 & j_3 & \beta_1 s & \beta_2 s & 0 \\ 0 & \alpha & j_4 & 0 & 0 \\ 0 & 0 & \xi & j_5 & 0 \\ 0 & 0 & \delta & \gamma & j_6 \end{pmatrix},$$

$$\begin{aligned} j_1 &= -(\beta_1 i_1 + \beta_2 i_2 + \mu), \\ j_2 &= \beta_1 i_1 + \beta_2 i_2, \\ j_3 &= -(\mu + \alpha), \\ j_4 &= -(\delta + \xi + \mu), \\ j_5 &= -(\mu + \epsilon + \gamma), \\ j_6 &= -(\mu + \eta). \end{aligned}$$

Or it is written as

$$A = \begin{pmatrix} d_{11} & 0 & d_{13} & d_{14} & d_{15} \\ d_{21} & d_{22} & d_{23} & d_{24} & 0 \\ 0 & d_{32} & d_{33} & 0 & 0 \\ 0 & 0 & d_{43} & d_{44} & 0 \\ 0 & 0 & d_{53} & d_{54} & d_{55} \end{pmatrix}.$$

Meanwhile, matrix B can be written as follows:

$$B = \begin{pmatrix} \frac{\partial f_1}{\partial u_1} & \frac{\partial f_1}{\partial u_2} \\ \frac{\partial f_2}{\partial u_1} & \frac{\partial f_2}{\partial u_2} \\ \frac{\partial f_3}{\partial u_1} & \frac{\partial f_3}{\partial u_2} \\ \frac{\partial f_4}{\partial u_1} & \frac{\partial f_4}{\partial u_2} \\ \frac{\partial f_5}{\partial u_1} & \frac{\partial f_5}{\partial u_2} \end{pmatrix}$$

Or it is expressed as

$$B = \begin{pmatrix} 0 & 0 \\ 0 & 0 \\ -i_1 & 0 \\ 0 & -i_2 \\ i_1 & i_2 \end{pmatrix}.$$

Next, by performing the calculation of the controllability matrix

$$M_c = [B, AB, A^2B, A^3B, A^4B],$$

$$M_c = \begin{pmatrix} 0 & 0 & f_{11} & f_{12} & g_{11} & g_{12} & h_{11} & h_{12} & g_{11} & g_{12} & i_{11} & i_{12} \\ 0 & 0 & f_{21} & f_{22} & g_{21} & g_{22} & h_{21} & h_{22} & g_{21} & g_{22} & i_{21} & i_{22} \\ e_{31} & 0 & f_{31} & 0 & g_{31} & g_{32} & h_{31} & h_{32} & g_{31} & g_{32} & i_{31} & i_{32} \\ 0 & e_{42} & f_{41} & f_{42} & g_{41} & g_{42} & h_{41} & h_{42} & g_{41} & g_{42} & i_{41} & i_{42} \\ e_{51} & e_{52} & f_{51} & f_{52} & g_{51} & g_{52} & h_{51} & h_{52} & g_{51} & g_{52} & i_{51} & i_{52} \end{pmatrix}.$$

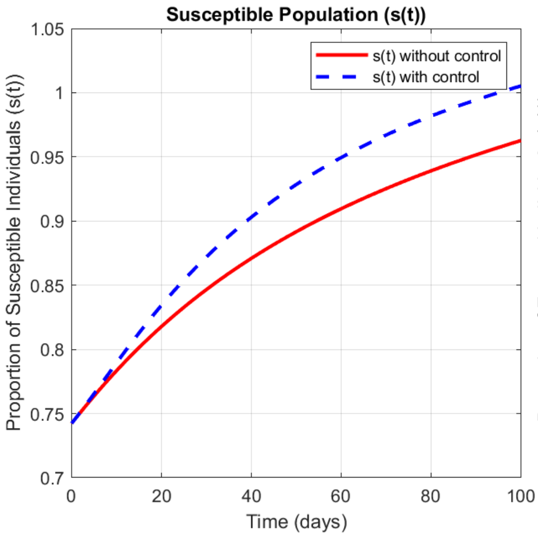
Based on the controllability matrix above, it is obtained that $\text{rank}(M_c) = 5$, so that the system can be said to be controlled.

3.5. *Pontryagin's Minimum Principle*

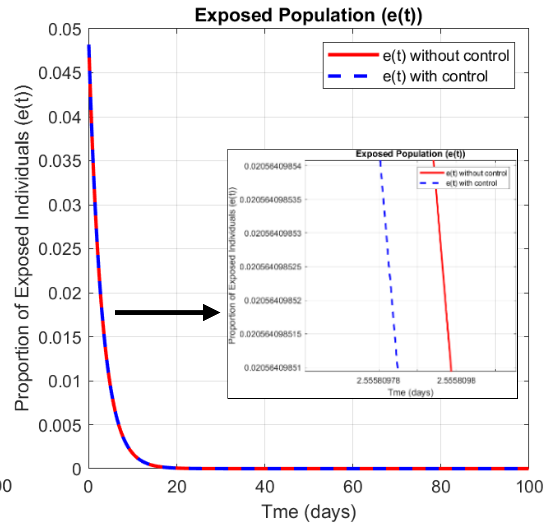
We further minimize the number of subpopulations infected by pneumonia, both mild pneumonia and severe pneumonia. So the objective function can be defined as follows.

$$J(u_1, u_2) = \min \int_{t_0}^{t_f} \left(A_1 i_1 + A_2 i_2 + \frac{1}{2}(A_3 u_1^2 + A_4 u_2^2) \right) dt,$$

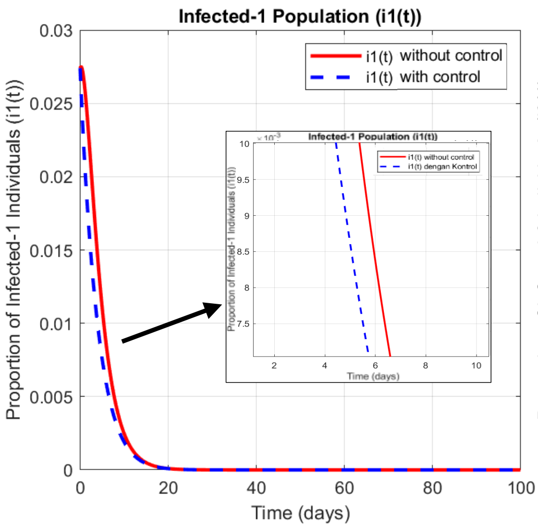
where is A_1 the weight of the effectiveness of the control to minimize the population sufferers of mild pneumonia (i_1), A_2 is the weight of the effectiveness of the control to minimize the sufferers of severe pneumonia (i_2), A_3 is the weight of the cost of first stage of treatment (u_1), and A_4 is the weight of the cost of second stage of treatment (u_2).



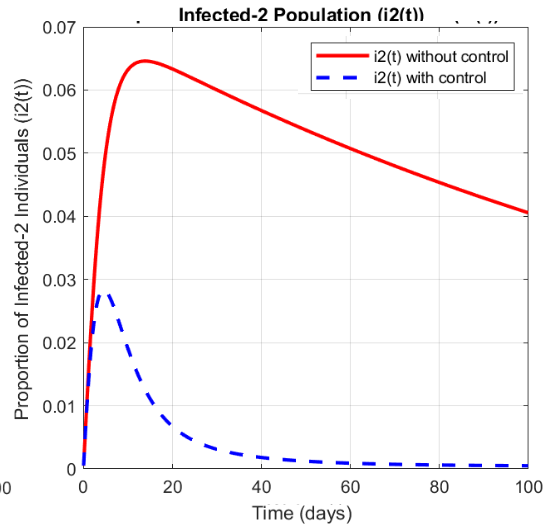
(a) Susceptible Population (s(t))



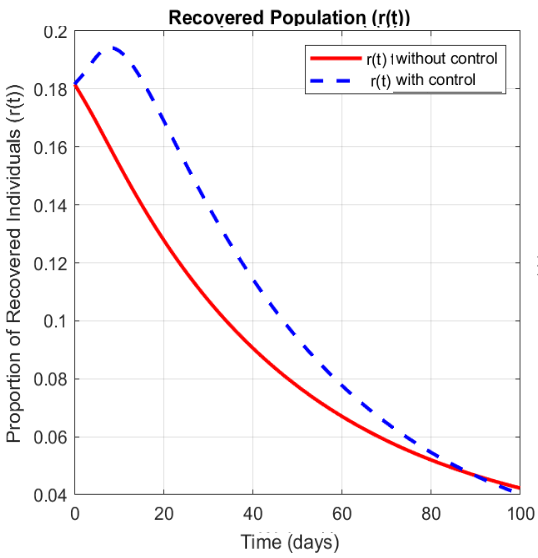
(b) Exposed Population (e(t))



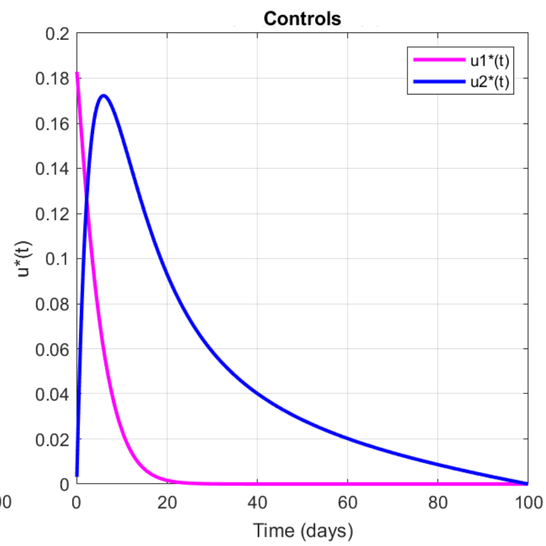
(c) Infected-1 Population (i1(t))



(d) Infected-2 Population (i2(t))



(e) Recovered Population (r(t))



(f) Controls (u*(t))

Figure 3. Comparison graph for each population before control and after control.

We further the Pontryagin's Minimum Principle for solving the problem of the optimal control. The steps taken are:

1. Formulate the Hamiltonian function and obtained the following:

$$\begin{aligned}\mathcal{H} =& A_1 i_1 + A_2 i_2 + \frac{1}{2}(A_3 u_1^2 + A_4 u_2^2)\\ &+ \lambda_s \left((1-\omega)\Lambda - s(\beta_1 i_1 + \beta_2 i_2) + \eta r - \mu s\right)\\ &+ \lambda_e \left(s(\beta_1 i_1 + \beta_2 i_2) - (\mu+\alpha)e\right)\\ &+ \lambda_{i1} \left(\alpha e - (\delta + u_1) i_1 - \xi i_1 - \mu i_1\right)\\ &+ \lambda_{i2} \left(\xi i_1 - (\gamma + u_2) i_2 - \epsilon i_2 - \mu i_2\right)\\ &+ \lambda_r \left(\omega\Lambda + (\delta + u_1) i_1 + (\gamma + u_2) i_2 - (\mu+\eta) r\right).\end{aligned}$$

2. Formulate the optimal control obtained from stationary conditions, and obtained as follows:

$$u_1^* = \min\left(\max\left(0, \frac{(\lambda_{i1} - \lambda_r) i_1}{A_3}\right), 1\right),$$

$$u_2^* = \min\left(\max\left(0, \frac{(\lambda_{i2} - \lambda_r) i_2}{A_4}\right), 1\right),$$

3. Determine the new optimal Hamiltonian function $\mathcal{H}$ using u_1^* and u_2^*, and obtained as follows:

$$\begin{aligned}\mathcal{H} =& A_1 i_1 + A_2 i_2 + \frac{1}{2}\left(A_3\left(\frac{\lambda_{i_1} i_1 - \lambda_r i_1}{A_3}\right)^2\right.\\ &\left. + A_4\left(\frac{\lambda_{i_2} - \lambda_r i_2}{A_4}\right)^2\right) + \lambda_s(\Lambda(1-\omega) - s(\beta_1 i_1\\ &+ \beta_2 i_2) + \eta r - \mu s) + \lambda_e(s(\beta_1 i_1 + \beta_2 i_2) - (\mu+\alpha)e)\\ &+ \lambda_{i_1}\left(\alpha e - \delta i_1 - \left(\frac{\lambda_{i_1} i_1 - \lambda_r i_1}{A_3}\right) i_1 - \xi i_2 - \mu i_2\right)\\ &+ \lambda_{i_2}\left(\xi i_1 - \gamma i_2 - \left(\frac{\lambda_{i_2} i_2 - \lambda_r i_2}{A_4}\right) i_2 - \epsilon i_2 - \mu i_2\right)\\ &+ \lambda_r(\Lambda\omega + \delta i_1 + \left(\frac{\lambda_{i_1} i_1 - \lambda_r i_1}{A_3}\right) i_1 + \gamma i_2\\ &+ \left(\frac{\lambda_{i_2} i_2 - \lambda_r i_2}{A_4}\right) i_2 - (\mu+\eta) r)\end{aligned}$$

4. Solve the state and costate equations to obtain the optimal system equation. State equation is defined as follows:

$$\begin{aligned}\dot{s} &= \frac{\partial \mathcal{H}^*}{\partial \lambda_s} = \Lambda(1-\omega) - s(\beta_1 i_1 + \beta_2 i_2) + \eta r - \mu s,\\ \dot{e} &= \frac{\partial \mathcal{H}^*}{\partial \lambda_e} = s(\beta_1 i_1 + \beta_2 i_2) - (\mu+\alpha)e,\\ \dot{i}_1 &= \frac{\partial \mathcal{H}^*}{\partial \lambda_{i_1}} = \alpha e - (\delta + u_1^*) i_1 - \xi i_1 - \mu i_1,\\ \dot{i}_2 &= \frac{\partial \mathcal{H}^*}{\partial \lambda_{i_2}} = \xi i_1 - (\gamma + u_2^*) i_2 - \epsilon i_2 - \mu i_2,\\ \dot{r} &= \frac{\partial \mathcal{H}^*}{\partial \lambda_r} = \Lambda\omega + (\delta + u_1^*) i_1 + (\gamma + u_2^*) i_2 - (\mu+\eta) r.\end{aligned}$$

Costate equation is expressed as follows:

$$\begin{aligned}\dot{\lambda}_s &= \lambda_s(\beta_1 i_1 + \beta_2 i_2) + \lambda_s\mu - \lambda_e(\beta_1 i_1 + \beta_2 i_2),\\ \dot{\lambda}_e &= \lambda_e\mu + \lambda_e\alpha - \lambda_{i_1}\alpha,\\ \dot{\lambda}_{i_1} &= -A_1 + \lambda_s s\beta_1 - \lambda_e s\beta_1 + \lambda_{i_1}\delta + \lambda_{i_1} u_1^* + \lambda_{i_1}\xi\\ &\quad + \lambda_{i_1}\mu - \lambda_{i_2}\xi - \lambda_r\delta - \lambda_r u_1^*,\\ \dot{\lambda}_{i_2} &= -A_2 + \lambda_s s\beta_2 - \lambda_e s\beta_2 + \lambda_{i_2}\mu + \lambda_{i_2}\epsilon + \lambda_{i_2}\gamma\\ &\quad + \lambda_{i_2} u_2^* - \lambda_r\gamma - \lambda_r u_2^*,\\ \dot{\lambda}_r &= -\lambda_s\eta + \lambda_r\mu + \lambda_r\eta.\end{aligned}$$

4. Numerical Results

Next, numerical simulation is carried out using the initial values and parameter values listed in both Tables 1 and 2. We conduct numerical calculation by implementing the 4th order Runge-Kutta forward-backward sweep method using Matlab software. In this numerical simulation, a comparison graph of the model will be displayed before being given control and after being given control for each subpopulation with the final time being 100 days. Figure 3 below is the result of graphical analysis of the numerical simulation results.

1. Susceptible Population ($s(t)$)
   Based on Figure 3a, it can be seen that the proportion of susceptible individuals before being given control and after being given control has increased quite significantly. The increase in both graphs can be caused by the addition of a susceptible population from newborn individuals, individuals who have not received complete vaccinations, and recovered individuals who return to being susceptible individuals, the number of which is greater than the number of natural deaths in the susceptible population. The proportion of susceptible individuals after being given control experienced a higher increase compared to the proportion of susceptible individuals before being given control. This is due to a fairly high increase in the proportion of individuals who recovered after being given control which also resulted in an increase in the proportion of susceptible individuals after being given control. This is what causes the proportion of susceptible individuals after being given control to be greater than the proportion of susceptible individuals before being given control.
2. Exposed Population ($e(t)$)
   Based on Figure 3b, it is obtained that the population graph exposed before being given control and after being given control experienced a significant decrease from $t = 0$ to $t = 20$ days. This decrease could occur due to the high rate of movement of exposed individuals to individuals infected with mild pneumonia. After time t = 20 days, both graphs will begin to stabilize with the proportion of exposed individuals approaching 0. This decrease can occur due to the high movement of exposed individuals to individuals infected with mild pneumonia. After 20 days, both graphs will begin to stabilize with the proportion of exposed individuals approaching 0. This means that after 20 days, the proportion of exposed individuals will gradually run out or in other words there are no more exposed individuals.
3. Population Infected with Mild Pneumonia ($i_1(t)$)
   Based on Figure 3c, it can be seen that both graphs experienced a significant decline from $t = 0$ to $t = 20$ days. This decrease could occur due to the large number of movements

from individuals infected with mild pneumonia to individuals infected with severe pneumonia or individuals who recovered. Based on this figure, it can also be seen that the proportion of individuals infected with mild pneumonia before being given control is higher than the proportion of individuals infected with mild pneumonia after being given control. This shows that the first stage of treatment control u_1 is very influential in reducing the population infected with mild pneumonia by increasing the recovery rate of individuals infected with mild pneumonia. After 20 days, both graphs will begin to stabilize with the proportion of individuals infected with mild pneumonia approaching 0, indicating that as time goes by the number of individuals infected with mild pneumonia will decrease until there are no more individuals infected with mild pneumonia.

4. Population Infected with Severe Pneumonia ($i_2(t)$)
Based on Figure 3d, it can be seen that the proportion of individuals infected with severe pneumonia before being given control increased significantly from the initial point up to $t = 13$ days with the proportion of individuals reaching $i_2(13) = 0.0645$. This can occur due to the large number of movements from individuals infected with mild pneumonia to individuals infected with severe pneumonia as a result of the lack of first-stage treatment control for individuals infected with mild pneumonia, which causes the rate of disease progression to be greater. After that, the graph before being given control will experience a decrease due to input from the proportion of individuals with mild pneumonia which is also decreasing and also deaths due to pneumonia that do not receive immediate treatment. Meanwhile, the proportion of individuals infected with severe pneumonia after being given control increased from the initial point to $t = 4,5$ days $i_2(4,5) = 0.0283$, with the proportion of individuals achieving this increase still lower than the increase in the population before control. After that, the graph after being given control slowly began to decline until at = 80 days it was found that the proportion of individuals infected with severe pneumonia after being given control began to be in a stable state approaching 0, which means there were no longer any individuals infected with severe pneumonia. Thus, it can be said that the second stage of treatment control (u_2) is very effective in reducing individuals infected with severe pneumonia increases the recovery rate of individuals infected with severe pneumonia. So, in this case, the control provided causes more and more individuals infected with severe pneumonia to become recovered individuals.

5. Recovered Population ($r(t)$)
Based on Figure 3e, it can be seen that the proportion of individuals who recovered before being given control decreased quite significantly from the initial point up to $t = 100$ days. Meanwhile, the graph of the proportion of individuals who recovered after being given control increased from the initial point until it reached a maximum point at $t = 8$ days with the proportion of individuals reaching $r(8) = 0,1942$. This increase could occur due to the relatively high proportion of individuals infected with severe pneumonia who have recovered from pneumonia. After that, the graph after being controlled will experience a decline of up to $t = 100$ days. This decrease is in line with input from individuals infected with mild and severe pneumonia which also decreases over time. Based on Figure 3e, it can also be seen that the graph of the population recovering after being given control was higher than the graph before being given control. This shows that control of the first stage of treatment (u_1) and control in the form of second stage of treatment (u_2) are able to contribute to reducing individuals infected with mild and severe pneumonia, thereby causing more individuals to recover from pneumonia. Apart from that, the increase in the recovered population is also obtained from the proportion of individuals after being given the vaccine. However, if you look at the two graphs of the recovered population at the end of the observation, it is found that the proportion of individuals who recovered after being given control was lower than the proportion of individuals who recovered before being given control. This can occur due to the influence of individual immunity or the influence of antibiotics or other drugs during infection which can also influence susceptibility to pneumonia. Therefore, this results in an increase in the proportion of susceptible individuals after being given control.

6. Control Admission
Figure 3f shows that the u_1 and u_2 controls have different administration intensities. The graph above shows that the intensity of the first stage of treatment control (u_1) is given maximally at the beginning of time, reaching $u_1(0) = 0.1828$ or around 18%. After that, the intensity of the first stage of treatment control (u_1) will decrease until $t = 20$ days. This decrease occurs because the rate of recovery of individuals infected with mild pneumonia is increasing so that more and more individuals infected with mild pneumonia are cured thanks to the first stage of treatment control. After 20 days, the first stage of treatment control (u_1) will be in a stable condition approaching 0. This shows that after 20 days there are no individuals infected with mild pneumonia so that the first stage of treatment control (u_1) is not needed. The intensity of the second stage of treatment control (u_2) given to the population infected with severe pneumonia has increased sharply since the beginning of the observation time until it reaches its maximum value at $t = 5.8$ days, reaching $u_2(5.8) = 0.1722$ or around 17%. This increase occurred because the proportion of individuals infected with severe pneumonia also increased. Then, after $t = 5.8$ days, the intensity of the provision of second-stage control treatment (u_2) slowly began to decrease along with the entry of individuals infected with severe pneumonia which also decreased. This decrease occurred because the rate of recovery of individuals infected with severe pneumonia increased so that more and more individuals infected with severe pneumonia recovered thanks to the second-stage control treatment (u_2). Thus, it can be concluded that the provision of first-stage control treatment (u_1) and second-stage control treatment (u_2) is very effective in efforts to overcome pneumonia in toddlers.

5. Conclusion

This study makes a significant contribution to efforts to control pneumonia in toddlers, particularly in East Java, through a mathematical modeling and optimal control approach. Numerical simulation results indicate that control with first- and second-stage treatment is effective in reducing the number of individuals infected with pneumonia, both in the mild and severe categories. Implementing a timely and sustainable treatment strategy not only reduces the number of infections but also significantly increases the recovery rate. These numerical solutions demonstrate the potential for real-world application in planning public health interventions, particularly in developing structured treatment policies to reduce the burden of pneumonia in vulnerable age groups such as toddlers.

Author Contributions. **Widodo, B.**: Carry out all activities from preparation to report completion. Develop a concept of mathematical model and methodology of computer simulation of the spread of pneumonia in toddlers in East Java, Indonesia, using Pontryagin's minimum principle. Supervise undergraduate students in this research. Make draft the conclusions of this research. Original draft preparation and review and editing a paper for publication in a reputable international journal. **Kamiran, K.**: Carry out all activities from preparation to report completion. Develop a methodology of computer simulation of the spread of pneumonia in toddlers in East Java, Indonesia, using Pontryagin's minimum principle. Supervise undergraduate students in this research. Original draft preparation and review and editing a paper for publication in a reputable international journal. **Syahputri, D.**: Carry out all activities from preparation to report completion. Develop a mathematical model and computer simulation of The Spread of Pneumonia in Toddlers in East Java, Indonesia Using the Pontryagin's Minimum Principle. Develop a numerical method to solve the mathematical model and computer simulation of The Spread of Pneumonia in Toddlers in East Java, Indonesia Using the Pontryagin's Minimum Principle. Create an algorithm and computer program, analysis and investigation for the numerical solution of the numerical simulation model of The Spread of Pneumonia in Toddlers in East Java, Indonesia Using the Pontryagin's Minimum Principle. Taking validation of the numerical solution. Design the conclusions of this research. Assist in creating the research logbook report. Assist in creating the financial report. Assist in creating the Progress Report and Final Research Report. Create the Final Project Book.

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