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# Numerical Analysis of Hepatitis A Transmission Using the Susceptible-Infected-Treatment-Recovery (SITR) Model with Vaccination Using 14th-Order Runge-Kutta Method

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**Abstract-** Hepatitis A remains a serious health issue in Indonesia, particularly in areas with poor sanitation. Poor sanitation significantly contributes to the transmission of Hepatitis A, as the disease is commonly spread through contaminated water and food. Inadequate waste management, lack of clean water access, and poor hygiene practices increase the likelihood of outbreaks, especially in densely populated areas. This study aims to analyze the dynamics of Hepatitis A transmission using the SITR (Susceptible-Infected-Treatment-Recovered) model, incorporating vaccination and treatment interventions. The methodology involves mathematical model construction, stability analysis, and numerical simulation using MATLAB's 14th-order Runge-Kutta method. Simulation results indicate a decline in the susceptible population (S) from 80% to nearly 0% due to high transmission rates ($\beta = 0.4286 - 0.9$and vaccination effectiveness ($\sigma = 0.008 - 0.01$). The infected population (I) decreases significantly through treatment interventions ($\eta = 0.1 - 0.3$and recovery rates ($\gamma = 0.0825 - 0.37$), while the recovered population (R) dominates up to 95% by the end of the simulation. The combination of vaccination, expanded treatment access, and improved care quality effectively suppresses disease spread, even under high transmission conditions. This study recommends multidimensional interventions such as mass vaccination, sanitation education, and rapid medical response for controlling Hepatitis A outbreaks.

## 1. Introduction

Hepatitis A is a type of hepatitis disease caused by a virus in the liver (Khairiah et al., 2017; MacKinney-Novelo et al., 2012; Naoumov, 2007).

Hepatitis A will be transmitted through close contact and the oral-fecal route, especially when food and drinks that have been contaminated with hepatitis A (Bae et al., 2014; Fiore, 2004). This virus belongs to the group of picornaviruses. Hepatitis A is easily transmitted to people who do not maintain hygiene because the virus attacks when people consume food or drinks contaminated with the feces of people who have been affected by hepatitis A (Harisma et al., 2018; Suni, 2019). There is an extraordinary incidence of hepatitis A experienced by developed countries, with a significant number of cases.

In 2003, there were 640 patients infected with hepatitis A that occurred in the states of Ohio and Pennsylvania in the United States. In 2008, 3.9 patients per 100,000 population were infected with hepatitis A in Europe. There are 300-500 cases of hepatitis A infection every year in Australia (Marantika, 2013). Indonesia is an endemic area for hepatitis A, where the virus is transmitted through close contact, especially objects at home, to children (Mentari & Besral, 2024). Indonesia has a high prevalence of hepatitis A cases, such as in 2016, when 7134 people died from hepatitis A worldwide (accounting for 0.5% of deaths due to the hepatitis virus) (World Health Organization, 2021). Based on data, developing countries that have poor sanitation and clean living practices have almost 90% of their population infected with hepatitis A before the age of 10 years (World Health Organization, 2021).

Mathematics is important in everyday life and various disciplines, which is realized through mathematical modeling. The result of this modeling process is referred to as a mathematical model. Mathematical models have been widely applied in various fields of science (Nurfitriana et al., 2019). The use of mathematical models will make it easier to understand a problem as well as find new methods for further development. For example, a problem that can be overcome with mathematical models is hepatitis A. There have been many previous studies that have discussed modeling the spread of hepatitis disease, including the spread of hepatitis A virus (HAV). In a study conducted by Nurfitriana et al. (2019), the model of the spread of hepatitis A disease was discussed by considering vaccination and sanitation factors. The model used in the study is the SIR (Susceptible-Infected-Recovered) model. In addition, other studies also use the SIR (Susceptible-Infected-Recovered) model to model the spread of hepatitis A in Jember Regency (Imamah et al., 2021). Another relevant research study is the stability of the mathematical model for hepatitis B virus transmission, which shows that the stability of the model is influenced by vaccination, treatment, and migration (Soleh et al., 2019). Furthermore, research by Ilahi & Fadilaturrohmah (2021) discusses the spread of hepatitis B with a treatment approach modeled using the SEIR (Susceptible, Exposed, Infected, and Recovered) model.

This study proposes a new mathematical model to analyze the dynamics of the spread of Hepatitis A disease by including factors that have not been widely explored in previous studies, namely the treatment factor or treatment for the infected population. The recovery process from symptoms of Hepatitis A virus infection (HAV) is often slow and can take several weeks or even months (World Health Organization, 2021). Therefore, treatment interventions are crucial because they can accelerate the healing process of individuals exposed to hepatitis A. This study aims to model the spread of Hepatitis A and interpret the model's results through numerical simulations using the Runge-Kutta Method of Order 14. In addition, this study also evaluates the effect of vaccination and treatment on the dynamics of the population infected with Hepatitis A. The model developed is the SITR (Susceptible-Infected-Treatment-Recovery) model. The analysis of this model was carried out to understand the dynamics of the spread of the disease and reduce the risk of increasing the population infected with hepatitis A.

This research is important because it offers a clearer and more complete understanding of how Hepatitis A spreads. It does this by adding both vaccination and treatment efforts into the traditional disease model. Most earlier studies only used SIR or SEIR models. However, the SITR model used in this study includes a treatment stage, making it more realistic, especially for areas where healthcare access and treatment quality affect how quickly people recover. Also, using a more accurate method like the 14th-order Runge-Kutta, this research can simulate how the disease spreads in more detail. These simulations can help health officials create better plans to control outbreaks. Overall, this study can support public health decisions, especially in places with a high risk of infection and limited medical facilities.

## 2. Methods

This research applies a library research method conducted from November to December 2024. This research was conducted by four people in the Laboratory of the Faculty of Teacher Training and Education, University of Jember. The procedure of this research is as follows: (1) The method used in this study is a documentation-based approach, where the SITR mathematical model is adopted from

previous research; (2) Data collection relies on secondary sources, specifically from prior studies that have formulated and analyzed the SITR model; (3) To solve the mathematical model, a numerical method is applied to analyze the dynamics of Hepatitis A transmission; (4) The numerical method employed is the 14th-order Runge-Kutta method, as previously examined by Fatahillah et al. (2021); (5) The formulation of the 14th-order Runge-Kutta method is then converted into a computational algorithm. An algorithm generally refers to a systematic sequence of steps to solve a problem. (6) This research uses MATLAB R2009a as the software tool to execute the algorithm and perform simulations.
The selection of the 14th-order Runge-Kutta numerical method in this study is based on the need for accuracy and stability of the results of the simulation of the SITR model, which is nonlinear and dynamic. This method can solve differential equation systems with high precision, especially for long time intervals, making it suitable for predicting the dynamics of infectious diseases over a certain period (Arif et al., 2024). Compared to the low-order Runge-Kutta method (such as order 4), the 14th-order method provides a much smaller truncation error, so the simulation results are closer to an analytical solution (Audu et al., 2025). All algorithms are tested in stages to ensure no computational errors, including sensitivity tests to parameter variations. The study also assumes a closed population without migration and homogeneity of interaction levels, although it has limitations in representing geographic diversity. However, such assumptions are necessary to simplify the complexity of the model without compromising the primary objective of health intervention policy analysis.

## 3. Results and Discussion

### (a) Result

The model used for the spread of Hepatitis A disease by vaccination is the SITR model, developed through the division of the population into four groups based on reference articles related to mathematical modeling of infectious diseases. The population includes Susceptible (S), a group of individuals who are healthy but vulnerable to being infected with Hepatitis A; Infected (I), a group of individuals currently infected with Hepatitis A; Treatment (T), a group of individuals undergoing treatment; and Recovery (R), a group of individuals who have recovered and are immune from Hepatitis A. The assumptions used in constructing the SITR model for the spread of Hepatitis A disease are as follows. Populations are constant.

a. The birth rate and death rate are the same, in that the individuals born are Susceptible (S) groups, and each individual who dies from all groups has a proportional rate with other groups.
b. Individuals who have hepatitis A or *an infection will undergo* the *treatment process*.
c. The death event in *the Susceptible* and *Recovery* group was a natural death, while the death in *the Infected* and *Treatment* class was yesterday caused by hepatitis A infection.
d. Vaccination of newborns or children
e. The Susceptible individual group is susceptible to hepatitis A infection.
f. Hepatitis A is contagious when susceptible individuals contact infected individuals or when the virus contacts susceptible individuals.
g. Individuals will recover from hepatitis A after receiving *treatment*
h. In this event, only one disease spreads, namely hepatitis A.
i. Individuals who recover have the possibility of being able to re-infectte.

Based on these assumptions, the mathematical model for spreading Hepatitis A disease is obtained, as shown in Figure 1.

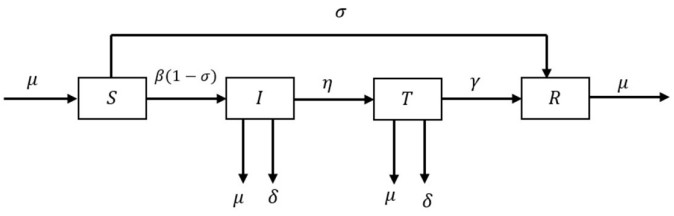


**Figure 1.** SITR model diagram for Hepatitis A spread with vaccination

Based on Figure 1, the system of differential equations for each compartment is obtained as follows:

$$\frac{dS}{dt} = \mu - (1-\sigma)(\beta)S\frac{I}{N} - \sigma S - \mu S$$

$$\frac{dI}{dt} = (1-\sigma)(\beta)S\frac{I}{N} - \eta I - \delta I - \mu I$$

$$\frac{dT}{dt} = \eta I - \gamma T - \delta T - \mu T$$

$$\frac{dR}{dt} = \gamma T + \sigma S - \mu R$$

The equation can be simplified to make it easier to analyze the model.

$$s = \frac{S}{N};\ i = \frac{I}{N};\ t = \frac{T}{N};\ r = \frac{R}{N}$$

So that the equation of the occupants is obtained.

$$\frac{ds}{dt} = \mu - (1-\sigma)(\beta)si - \sigma s - \mu s$$

$$\frac{di}{dt} = (1-\sigma)(\beta)si - \eta i - \delta i - \mu i$$

$$\frac{dt}{dt} = \eta i - \gamma t - \delta t - \mu t$$

$$\frac{dr}{dt} = \gamma t + \sigma s - \mu r$$

Information:

$\beta$ : The rate of transmission of an infected individual to the suspect
$\mu$ : Rate of births or deaths that are not the result of hepatitis A infection (naturally)
$\delta$ : Death rate due to hepatitis A infection
$\gamma$ : The rate of recovery of an individual after undergoing treatment for hepatitis A infection
$\sigma$ : Vaccination effectiveness level
$\eta$ : Proportion of infected individuals who receive treatment
$S$: Number of vulnerable individuals
$I$ : Individuals affected by hepatitis A
$R$: Recovered individuals who will be immune to hepatitis A
$T$: Infected individuals who are undergoing treatment
$N$: The number of the human population (Sidik et al., 2022).

Numerical Simulation of SITR Mathematical Models Using Runge-Kutta Order 14. The simulation was carried out using the parameter values contained in Table 1. To view population dynamics based on these parameter values, which can be seen in Table 1, the simulation was performed using MATLAB software with N = 100 and initial values: s(0) = 0.8, i(0) = 0.2, t(0) = 0, r(0) = 0.

**Table 1.** Value each parameter

| Parameter | Description | Parameter Values | |
|---|---|---|---|
| | | $T_1$ | $T_2$ |
| $\mu$ | Rate of births or deaths that are not the result of hepatitis A infection (naturally) | 0.002 | 0.002 |
| $\delta$ | Death rate due to hepatitis A infection | 0.0025 | 0.003 |
| $\sigma$ | Vaccination effectiveness level | 0.008 | 0.01 |
| $\beta$ | The rate of transmission of an infected individual to the suspect | 0.4286 | 0.9 |
| $\gamma$ | The rate of recovery of an individual after undergoing treatment for hepatitis A infection | 0.0825 | 0.37 |
| $\eta$ | Proportion of infected individuals who receive treatment | 0.3 | 0.1 |

(Sidik et al., 2022)

After determining the parameter values in Table 1, the program was created using the Runge Kutta Order 14 method in MATLAB. It included the SITR model for the spread of hepatitis A disease by vaccination using the Runge-Kutta order 14 method and the initial values and parameters used. A numerical solution in the form of a graph of the number of each individual was obtained.

The following is a MATLAB program to conduct a numerical simulation of the SITR model on the spread of hepatitis A disease by vaccination using the Runge-Kutta method of order 14.

```
clear all;
clc;
```

```
% Parameter
mu = 0.002;
delta = 0.0025;
sigma = 0.008;
beta = 0.4286;
gamma = 0.0825;
eta = 0.3;

% Kondisi awal
s0 = 0.8;
i0 = 0.2;
t0 = 0.0;
r0 = 0.0;

% Rentang waktu dan langkah iterasi
h = 0.01;
tfinal = 100;
n = ceil(tfinal/h);

% Inisialisasi solusi
t = zeros(1, n+1);
S = zeros(1, n+1);
I = zeros(1, n+1);
T = zeros(1, n+1);
R = zeros(1, n+1);

t(1) = 0;
S(1) = s0;
I(1) = i0;
T(1) = t0;
R(1) = r0;

% Fungsi model SITR
fS = @(S, I) mu - (1 - sigma) * beta * S * I - sigma * S - mu * S;
fI = @(S, I) (1 - sigma) * beta * S * I - eta * I - delta * I - mu * I;
fT = @(I, T) eta * I - gamma * T - delta * T - mu * T;
fR = @(S, T, R) gamma * T + sigma * S - mu * R;

disp('Iterasi         t           S           I           T           R');

for i = 1:n
    % K1
    k1S = h * fS(S(i), I(i));
    k1I = h * fI(S(i), I(i));
    k1T = h * fT(I(i), T(i));
    k1R = h * fR(S(i), T(i), R(i));
    % K2
    k2S = h * fS(S(i) + k1S/2, I(i) + k1I/2);
    k2I = h * fI(S(i) + k1S/2, I(i) + k1I/2);
    k2T = h * fT(I(i) + k1I/2, T(i) + k1T/2);
    k2R = h * fR(S(i) + k1S/2, T(i) + k1T/2, R(i) + k1R/2);
    % k3
    k3S = h * fS(S(i) + (5/27) * k1S, I(i) + (5/27) * k1I);
    k3I = h * fI(S(i) + (5/27) * k1S, I(i) + (5/27) * k1I);
    k3T = h * fT(I(i) + (5/27) * k1I, T(i) + (5/27) * k1T);
    k3R = h * fR(S(i) + (5/27) * k1S, T(i) + (5/27) * k1T, R(i) + (5/27) * k1R);
    % k4
    k4S = h * fS(S(i) + (1/9) * k1S + (1/3) * k3S, I(i) + (1/9) * k1I + (1/3) * k3I);
    k4I = h * fI(S(i) + (1/9) * k1S + (1/3) * k3S, I(i) + (1/9) * k1I + (1/3) * k3I);
    k4T = h * fT(I(i) + (1/9) * k1I + (1/3) * k3I, T(i) + (1/9) * k1T + (1/3) * k3T);
    k4R = h * fR(S(i) + (1/9) * k1S + (1/3) * k3S, T(i) + (1/9) * k1T + (1/3) * k3T, R(i) + (1/9) *
k1R + (1/3) * k3R);
    % k5
    k5S = h * fS(S(i) + (1/7) * k1S + (2/3) * k4S, I(i) + (1/6) * k1I + (2/3) * k4I);
    k5I = h * fI(S(i) + (1/7) * k1S + (2/3) * k4S, I(i) + (1/6) * k1I + (2/3) * k4I);
    k5T = h * fT(I(i) + (1/7) * k1I + (2/3) * k4I, T(i) + (1/6) * k1T + (2/3) * k4T);
    k5R = h * fR(S(i) + (1/7) * k1S + (2/3) * k4S, T(i) + (1/6) * k1T + (2/3) * k4T, R(i) + (1/6) *
k1R + (2/3) * k4R);
    % k6
    k6S = h * fS(S(i) + (5/12) * k1S - (25/16) * k3S + (27/16) * k4S, I(i) + (5/12) * k1I - (25/16) *
k3I + (25/16) * k4I);
    k6I = h * fI(S(i) + (5/12) * k1S - (25/16) * k3S + (27/16) * k4S, I(i) + (5/12) * k1I - (25/16) *
k3I + (25/16) * k4I);
    k6T = h * fT(I(i) + (5/12) * k1I - (25/16) * k3I + (27/16) * k4I, T(i) + (5/12) * k1T - (25/16) *
k3T + (25/16) * k4T);
    k6R = h * fR(S(i) + (5/12) * k1S - (25/16) * k3S + (27/16) * k4S, T(i) + (5/12) * k1T - (25/16) *
k3T + (25/16) * k4T, R(i) + (5/12) * k1R - (25/16) * k3R + (25/16) * k4R);
    % k7
    k7S = h * fS(S(i) + (1/20) * k1S + (1/4) * k4S + (1/5) * k5S - (1/12) * k6S, I(i) + (1/20) * k1I +
(1/4) * k4I + (1/5) * k5I - (1/12) * k6I);
    k7I = h * fI(S(i) + (1/20) * k1S + (1/4) * k4S + (1/5) * k5S - (1/12) * k6S, I(i) + (1/20) * k1I +
(1/4) * k3I + (1/5) * k5I - (1/12) * k6I);
    k7T = h * fT(I(i) + (1/20) * k1I + (1/4) * k4I + (1/5) * k5I - (1/12) * k6I, T(i) + (1/20) * k1T +
(1/4) * k4T + (1/5) * k5T - (1/12) * k6T);
    k7R = h * fR(S(i) + (1/20) * k1S + (1/4) * k4S + (1/5) * k5S - (1/12) * k6S, T(i) + (1/20) * k1T +
(1/4) * k3T + (1/5) * k5T - (1/12) * k6T, R(i) + (1/20) * k1R + (1/4) * k4R + (1/5) * k5R - (1/12) *
k6R);
    % k8
```

```
    k8S = h * fS(S(i) + (1/12) * k1S - (1/5) * k4S + (1/6) * k5S + (1/20) * k6S, I(i) + (1/12) * k1I -
(1/5) * k4I + (1/2) * k5I + (1/20) * k6I);
    k8I = h * fI(S(i) + (1/12) * k1S - (1/5) * k4S + (1/6) * k5S + (1/20) * k6S, I(i) + (1/12) * k1I -
(1/5) * k4I + (1/2) * k5I + (1/20) * k6I);
    k8T = h * fT(I(i) + (1/12) * k1I - (1/5) * k4I + (1/6) * k5I + (1/20) * k6I, T(i) + (1/12) * k1T -
(1/5) * k4T + (1/2) * k5T + (1/20) * k6T);
    k8R = h * fR(S(i) + (1/12) * k1S - (1/5) * k4S + (1/6) * k5S + (1/20) * k6S, T(i) + (1/12) * k1T -
(1/5) * k4T + (1/2) * k5T + (1/20) * k6T, R(i) + (1/12) * k1R - (1/5) * k4R + (1/2) * k5R + (1/20) *
k6R);
    % k9
    k9S = h * fS(S(i) - (1/15) * k1S + (1/4) * k4S - (1/7) * k5S + (3/26) * k7S, I(i) - (1/15) * k1I +
(1/4) * k4I - (1/7) * k5I + (3/26) * k7I);
    k9I = h * fI(S(i) - (1/15) * k1S + (1/4) * k4S - (1/7) * k5S + (3/20) * k7S, I(i) - (1/15) * k1I +
(1/4) * k4I - (1/7) * k5I + (3/26) * k7I);
    k9T = h * fT(I(i) - (1/15) * k1I + (1/4) * k4I - (1/7) * k5I + (3/20) * k7I, T(i) - (1/15) * k1T +
(1/4) * k4T - (1/7) * k5T + (3/26) * k7T);
    k9R = h * fR(S(i) - (1/15) * k1S + (1/4) * k4S - (1/7) * k5S + (3/20) * k7S, T(i) - (1/15) * k1T +
(1/4) * k4T - (1/7) * k5T + (3/26) * k7T, R(i) - (1/15) * k1R + (1/4) * k4R - (1/7) * k5R + (3/26) *
k7R);
    % k10
    k10S = h * fS(S(i) + (3/40) * k1S - (5/9) * k5S + (4/15) * k7S + (1/10) * k8S, I(i) + (3/40) * k1I
- (5/9) * k5I + (4/15) * k7I + (1/10) * k8I);
    k10I = h * fI(S(i) + (3/40) * k1S - (5/9) * k5S + (4/15) * k7S + (1/10) * k8S, I(i) + (3/40) * k1I
- (5/9) * k5I + (4/15) * k7I + (1/10) * k8I);
    k10T = h * fT(I(i) + (3/40) * k1I - (5/9) * k5I + (4/15) * k7I + (1/10) * k8I, T(i) + (3/40) * k1T
- (5/9) * k5T + (4/15) * k7T + (1/10) * k8T);
    k10R = h * fR(S(i) + (3/40) * k1S - (5/9) * k5S + (4/15) * k7S + (1/10) * k8S, T(i) + (3/40) * k1T
- (5/9) * k5T + (4/15) * k7T + (1/10) * k8T, R(i) + (3/40) * k1R - (5/9) * k5R + (4/15) * k7R +
(1/10);
    % k11
    k11S = h * fS(S(i) - (7/18) * k1S + (2/5) * k6S - (5/12) * k9S + (1/7) * k10S, I(i) - (7/18) * k1I
+ (2/5) * k6I - (5/12) * k9I + (1/7) * k10I);
    k11I = h * fI(S(i) - (7/18) * k1S + (2/5) * k6S - (5/12) * k9S + (1/7) * k10S, I(i) - (7/18) * k1I
+ (2/5) * k6I - (5/12) * k9I + (1/7) * k10I);
    k11T = h * fT(I(i) - (7/18) * k1I + (2/5) * k6I - (5/12) * k9I + (1/7) * k10I, T(i) - (7/18) * k1T
+ (2/5) * k6T - (5/12) * k9T + (1/7) * k10T);
    k11R = h * fR(S(i) - (7/18) * k1S + (2/5) * k6S - (5/12) * k9S + (1/7) * k10S, T(i) - (7/18) * k1T
+ (2/5) * k6T - (5/12) * k9T + (1/7) * k10T, R(i) - (7/18) * k1R + (2/5) * k6R - (5/12) * k9R + (1/7);
    % k12
    k12S = h * fS(S(i) + (1/8) * k1S - (1/3) * k7S + (1/4) * k10S + (3/16) * k11S, I(i) + (1/8) * k1I
- (1/3) * k7I + (1/4) * k10I + (3/16) * k11I);
    k12I = h * fI(S(i) + (1/8) * k1S - (1/3) * k7S + (1/4) * k10S + (3/16) * k11S, I(i) + (1/8) * k1I
- (1/3) * k7I + (1/4) * k10I + (3/16) * k11I);
    k12T = h * fT(I(i) + (1/8) * k1I - (1/3) * k7I + (1/4) * k10I + (3/16) * k11I, T(i) + (1/8) * k1T
- (1/3) * k7T + (1/4) * k10T + (3/16) * k11T);
    k12R = h * fR(S(i) + (1/8) * k1S - (1/3) * k7S + (1/4) * k10S + (3/16) * k11S, T(i) + (1/8) * k1T
- (1/3) * k7T + (1/4) * k10T + (3/16) * k11T, R(i) + (1/8) * k1R - (1/3) * k7R + (1/4) * k10R + (3/7);
    % k13
    k13S = h * fS(S(i) + (1/10) * k1S + (1/6) * k8S - (1/5) * k11S + (3/8) * k12S, I(i) + (1/10) * k1I
+ (1/6) * k8I - (1/5) * k11I + (3/8) * k12I);
    k13I = h * fI(S(i) + (1/10) * k1S + (1/6) * k8S - (1/5) * k11S + (3/8) * k12S, I(i) + (1/10) * k1I
+ (1/6) * k8I - (1/5) * k11I + (3/8) * k12I);
    k13T = h * fT(I(i) + (1/10) * k1I + (1/6) * k8I - (1/5) * k11I + (3/8) * k12I, T(i) + (1/10) * k1T
+ (1/6) * k8T - (1/5) * k11T + (3/8) * k12T);
    k13R = h * fR(S(i) + (1/10) * k1S + (1/6) * k8S - (1/5) * k11S + (3/8) * k12S, T(i) + (1/10) * k1T
+ (1/6) * k8T - (1/5) * k11T + (3/8) * k12T, R(i) + (1/10) * k1R + (1/6) * k8R - (1/5) * k11R + (3/8);
    % k14
    k14S = h * fS(S(i) + (1/20) * k1S + (1/7) * k11S - (1/12) * k12S + (1/5) * k13S, I(i) + (1/20) *
k1I + (1/7) * k11I - (1/12) * k12I + (1/5) * k13I);
    k14I = h * fI(S(i) + (1/20) * k1S + (1/7) * k11S - (1/12) * k12S + (1/5) * k13S, I(i) + (1/20) *
k1I + (1/7) * k11I - (1/12) * k12I + (1/5) * k13I);
    k14T = h * fT(I(i) + (1/20) * k1I + (1/7) * k11I - (1/12) * k12I + (1/5) * k13I, T(i) + (1/20) *
k1T + (1/7) * k11T - (1/12) * k12T + (1/5) * k13T);
    k14R = h * fR(S(i) + (1/20) * k1S + (1/7) * k11S - (1/12) * k12S + (1/5) * k13S, T(i) + (1/20) *
k1T + (1/7) * k11T - (1/12) * k12T + (1/5) * k13T, R(i) + (1/20) * k1R + (1/7) * k11R - (1/12) * k12R
+ (1/5);

% Update solusi
S(i+1) = S(i) + (k1S + 2*k2S + 2*k3S + 2*k4S + 2*k5S + 2*k8S + 2*k9S + 2*k10S + 2*k11S + 2*k12S +
2*k13S + k14S)/14;
I(i+1) = I(i) + (k1I + 2*k2I + 2*k3I + 2*k4I + 2*k5I + 2*k8I + 2*k9I + 2*k10I + 2*k11I + 2*k12I +
2*k13I + k14I)/14;
T(i+1) = T(i) + (k1T + 2*k2T + 2*k3T + 2*k4T + 2*k5T + 2*k8T + 2*k9T + 2*k10T + 2*k11T + 2*k12T +
2*k13T + k14T)/14;
R(i+1) = R(i) + (k1R + 2*k2R + 2*k3R + 2*k4R + 2*k5R + 2*k8R + 2*k9R + 2*k10R + 2*k11R + 2*k12R +
2*k13R + k14R)/14;
t(i+1) = t(i) + h;

    fprintf('%6d   %8.2f   %8.5f   %8.5f   %8.5f   %8.5f\n', i, t(i), S(i), I(i), T(i), R(i));
end

% Plot solusi per kategori
figure(1);
subplot(2,2,1)
plot(t, S, '-k', 'linewidth', 2);
xlabel('Waktu', 'FontSize', 7);
ylabel('Jumlah Populasi', 'FontSize', 7);
legend('Susceptible (Rentan)', 'FontSize', 7)
```

```
subplot(2,2,2)
plot(t, I, '-r', 'linewidth', 2);
xlabel('Waktu', 'FontSize', 7);
ylabel('Jumlah Populasi', 'FontSize', 7);
legend('Infected (Terinfeksi)', 'FontSize', 7)

subplot(2,2,3)
plot(t, T, '-g', 'linewidth', 2);
xlabel('Waktu', 'FontSize', 7);
ylabel('Jumlah Populasi', 'FontSize', 7);
legend('Treatment (Dalam Perawatan)', 'FontSize', 7)

subplot(2,2,4)
plot(t, R, '-b', 'linewidth', 2);
xlabel('Waktu', 'FontSize', 7);
ylabel('Jumlah Populasi', 'FontSize', 7);
legend('Recovered (Pulih)', 'FontSize', 7)

% Plot semua solusi dalam satu grafik
figure(2);
plot(t, S, '-k', 'linewidth', 2); hold on;
plot(t, I, '-r', 'linewidth', 2);
plot(t, T, '-g', 'linewidth', 2);
plot(t, R, '-b', 'linewidth', 2);
title('Dinamika Populasi Model SITR', 'Color', [0 0 1], 'FontWeight', 'bold', 'FontSize', 12);
ylabel('Jumlah Populasi', 'Color', [0 0 1], 'FontWeight', 'bold', 'FontSize', 8);
xlabel('Waktu (t)', 'Color', [0 0 1], 'FontWeight', 'bold', 'FontSize', 8);
legend('Susceptible (Rentan)', 'Infected (Terinfeksi)', 'Treatment (Dalam Perawatan)', 'Recovered
(Pulih)', 'FontSize', 10)
grid on;
```

The MATLAB program is run to obtain graphs for each susceptible, infected, treatment, and recovery group as shown in Figure 2.

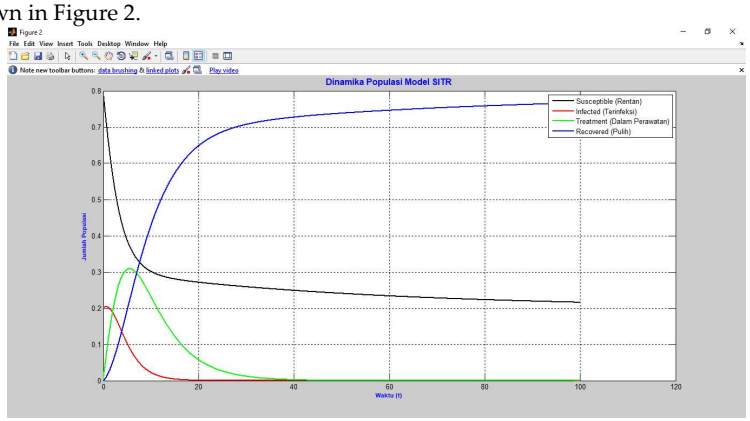


**Figure 2.** Graph of MATLAB program simulation results

The results of numerical simulation of the SITR model using the Runge-Kutta Order 14 method describe the dynamics of the spread of Hepatitis A influenced by vaccination and treatment interventions. Based on Figure 2, the vulnerable population (S) experienced a drastic decrease from 80% to almost 0%, mainly due to high virus transmission ($\beta$(0.4286 in $T_1$ and 0.9 in $T_2$and the effectiveness of vaccination ($\sigma = 0.008 - 0.01$) which directly shifted vulnerable individuals to the recovered category (R). Meanwhile, the infected population (I) initially jumped to 20% (as per the initial conditions), but dropped significantly over time thanks to treatment interventions ($\eta = 0.3$ at $T_1$ and 0.1 at $T_2$) that moved infected individuals to the treatment group (T), as well as natural mortality ($\mu = 0.002$) and disease mortality ($\delta = 0.0025 - 0.003$). In the $T_1$ scenario, a wider coverage of treatment (high $\eta$) resulted in a stable decrease in infection cases, while in T2. However, transmission was faster ($\beta = 0.9$), a high cure rate ($\gamma = 0.37$) succeeded in suppressing the number of active cases. The population under treatment (T) peaked with the decline I, then shrank due to recovery ($\gamma = 0.0825$ at $T_1$ and 0.37 at $T_1$), while the recovered population (R) dominated to 90–95% at the end of the simulation, driven by vaccination and recovery from treatment. A comparison of the two scenarios shows that a combination of vaccination, expanded access to treatment, and improved quality of care is the main key in controlling the outbreak, even in high transmission conditions. These results align with previous studies that emphasized the importance of multidimensional interventions that include mass vaccination, sanitation education, and rapid medical response to break the chain of Hepatitis A spread, especially in areas with poor sanitation and high risk of transmission.

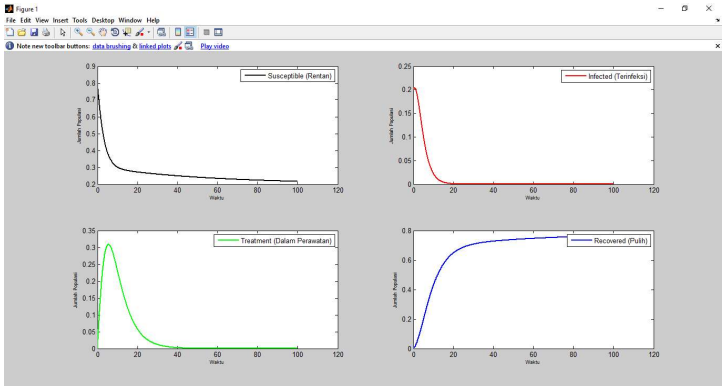


**Figure 3.** Graph of the simulation results of each group

Based on the graphs in Figure 3 generated from the MATLAB program, the SITR model successfully describes the dynamics of the spread of Hepatitis A disease in the population. The graph of susceptible groups shows that the number of vulnerable individuals has decreased sharply over time. This reflects the disease transmission process, in which individuals initially vulnerable to the Hepatitis A virus are exposed through contact with infected individuals or contaminated environments. This decline in vulnerable populations occurs rapidly in the early phases of simulations, which indicates a high rate of disease transmission if there are no effective prevention efforts. The infected graph shows that infected individuals increased sharply initially, but then declined drastically over time. This decrease indicates the effectiveness of interventions such as medical care and vaccination in suppressing the spread of the disease. The treatment graph supports this, where the number of individuals in treatment increases rapidly at the beginning of the simulation, indicating a significant treatment effort. However, this number then decreased because individuals in treatment managed to recover and moved to the recovered group. The recovered graph shows a significant increase in recovered individuals, eventually reaching a stable value. This illustrates the success of treatment and recovery in dealing with individuals infected with Hepatitis A. With proper treatment, infected individuals can recover completely, so the number of recovered individuals dominates at the end of the simulation. This condition shows the importance of prompt and appropriate medical intervention and prevention efforts through vaccination and environmental hygiene education to control the spread of the disease.

## (b) Discussion

The results of this study reinforce the basic theory of the epidemiological group model, especially the SITR model, which states that interventions such as vaccination and treatment can change the dynamics of disease spread (Toaha et al., 2024). The decline in the vulnerable population (S) to near zero, which indicates that vaccination ($\sigma$) Significantly reduces the proportion of vulnerable individuals, especially when integrated with sanitation policies (Nurfitriana et al., 2019; Utazi et al., 2023). However, this study builds on previous models by including the Treatment (T) group, which shows that increased treatment ($\eta$) and recovery ($\gamma$Rates reduce the infected population (I) and accelerate the transition of the recovery (R) group. This is based on research on the Hepatitis B model, where medical intervention ($\eta$) and quality of care ($\gamma$) are the determining factors for recovery (Soleh et al., 2019; Wodajo & Mekonnen, 2022). These findings also support the theory that health systems with rapid responses to active cases can suppress the rate of transmission, even in scenarios with high infection rates ($\beta = 0.9$) (Singh et al., 2024).

Based on a health policy perspective, the simulation results align with the WHO recommendation, emphasizing the importance of mass vaccination and increased access to treatment for infectious diseases such as Hepatitis A (World Health Organization, 2021). The effectiveness of vaccination (σ) in diverting vulnerable individuals directly to the recovery category (R) shows that this strategy can be a long-term solution to achieve herd immunity. (Rasmussen, 2020). However, this study also revealed that the success of the intervention depends not only on vaccination coverage, but also on the quality of treatment ($\gamma$). As observed in the T2 scenario, although treatment coverage is low ($\eta = 0.1$Improved treatment quality= (0.37) can compensate for the high transmission rate. These findings reinforce Ilahi and Fadilaturrohmah's argument that a combination of preventive (vaccination) and curative (treatment) interventions is the optimal approach in outbreak control, especially in areas with limited health resources.. (Ilahi & Fadilaturrohmah, 2021).

In addition to highlighting the roles of vaccination and treatment, the simulation results demonstrate that the group of individuals undergoing treatment (T) plays a significant role in the transition dynamics from infected to recovered status. In the early stages of the simulation, the T group increases in size as the number of individuals in the I group decreases, reflecting the effectiveness of referral mechanisms and access to treatment. However, over time, the number of individuals in the T group also declines due to the success of treatment, which contributes to the growth of the recovered population (R). These findings emphasize that the availability and capacity of healthcare facilities are key indicators of intervention success (Sidik et al., 2022). In scenarios with a high $\gamma$ value, the transition from T to R occurs more rapidly, indicating that access and the speed and quality of treatment determine how effectively an outbreak can be managed (Wodajo & Mekonnen, 2022). Thus, this result provides strong evidence that a responsive, integrated, and high-quality healthcare system is crucial in effectively breaking the chain of Hepatitis A transmission (Singh et al., 2024; World Health Organization, 2021).

## 4. Conclusion

This study analyzed the dynamics of the spread of Hepatitis A using the SITR (Susceptible-Infected-Treatment-Recovered) model by considering vaccination and treatment interventions. The Runge-Kutta Order 14 method is used to perform numerical simulations in MATLAB. The simulation results show that the vulnerable population (S) has experienced a drastic decrease from 80% to almost 0% due to the high rate of transmission ($\beta$) and vaccination effectiveness ($\sigma$). The infected population (I) initially increased, but then dropped significantly thanks to treatment interventions ($\eta$) and recovery rates ($\gamma$). The recovered population (R) dominated up to 95% at the end of the simulation, demonstrating the success of the combination of vaccination and treatment in suppressing the spread of the disease. The combination of vaccination, expanding access to care, and improving the quality of care has proven effective in controlling Hepatitis A outbreaks, even in high transmission conditions. The study recommends multidimensional interventions, including mass vaccination, sanitation education, and rapid medical response, to break the chain of Hepatitis A spread, especially in areas with poor sanitation and high risk of transmission. These findings reinforce the importance of preventive and curative approaches in outbreak control and confirm that the success of an intervention depends not only on vaccination coverage but also on the quality of care provided. This study has several limitations. The SITR model assumes a constant population size and does not account for migration, fluctuations, or regional variations in treatment and vaccination effectiveness. Additionally, the model does not incorporate external factors like environmental conditions or socioeconomic variables, and the 14th-order Runge-Kutta method requires significant computational resources, limiting its real-time application.

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