An epidemic model with age structured of rubella virus: threshold and stability

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Abstract. This study aimed to explore the dynamic behaviour of rubella virus model which considers age-structured. It was not only to figure out the stability analysis of the model but also the judgement of the result through numerical simulation. Firstly, we constructed model for the disease spread based on the characteristic of rubella virus. The basic model of the disease spread (SEIR) had been used as a fundamental model. In the beginning, human population have been divided into two age groups; children and adults. Furthermore, based on the characteristic of rubella, we separated each group into four sub populations, i.e., Susceptible, Exposed, Infected, and Recovered. The transmission routes of the disease were formulated mathematically based on the real problem. In the second step, two disease equilibrium points had been obtained. There were a disease-free equilibrium and an endemic equilibrium. Moreover, by using Next Generation Matrix method, the basic reproduction number also had been determined. Finally, stability analysis had been done to explore the persistence of the disease. This results also had been supported by the numerical simulation.

1. Introduction
Rubella, which is well-known as German measles disease, is a type of disease that spreads not only in tropical countries but also in sub-tropics. The disease was first discovered in the mid-eighteenth. De Bergen in 1752 and Orlow in 1758 confirmed the first clinical description of rubella in 1740 made by Friedrich Hoofman. The disease is caused by the rubella virus which belongs to the togavirus group and comes from the rubivirus type which is included in the RNA virus class. The genetic material of this virus is in the form of nucleic acids with single or double twist chains [1], [2]. Body fluids is released by a person with rubella disease can spread through the air, especially when a person coughs or sneezes. It takes place through the respiratory tract which can then live and multiply in the nasopharynx and regional lymph nodes for 4 - 7 days. The incubation period for rubella disease ranges from 14 to 21 days with symptoms such as low-grade fever (37.2°C) and maculopapular rash accompanied by enlarged lymph nodes behind the ears, back of the neck and sub occipital [3], [4].

Rubella disease not only affects children but also adults. The consequences given by this disease are low if it infects children. In general, children will only have a mild fever or do not even feel a change in their body. However, if this disease infects pregnant women, then the virus can cause miscarriage in the fetus or the fetus who is born with congenital rubella syndrome (CRS). Children with this congenital syndrome are born with heart defects (Patent ductus arteriosus, atrial septal defect, ventricular septal defect, pulmonary valve stenosis), eye disorders (congenital cataracts, congenital glaucoma, pigmentary retinopathy), hearing disorders, disorders of the nervous system (Mental retardation, microcephalia,
meningoencephalitis), and other disorders (purpura, splenomegaly, jaundice present within 24 hours of birth, radiolucent bone) [5].

Since 2017, the Indonesian government through the Directorate General of Disease Prevention and Control of the Ministry of Health is campaigning for the use of the MR vaccine to prevent the spread of rubella [6]. The government hopes that by 2020, Indonesia will be able to eliminate the measles virus and control rubella. In line with the expectations of the Indonesian government, it is necessary to make a mathematical model that can be used to predict the presence of rubella. The model is constructed by considering the characteristics of the virus, the spread pattern of the rubella virus, and the medical symptoms that appear when a person is infected by rubella.

This study aimed to explore the dynamic behavior of rubella virus which takes into account age-structured of the population. In the first step, we proposed a model for the spread of disease by dividing women population into two age groups, such as children and adults. Each group was contained susceptible sub-population, exposed sub-population, infected sub-population, and recovered sub-population. Furthermore, we calculated the threshold of the disease spread known as a basic reproduction number \( R_0 \) to figure out the condition that the disease will be exist or extinct. In the next step, we determined the equilibria of the system and analyzed their stability, and then finally, we simulated the data numerically to compare the results.

2. Mathematical model

The model was constructed from the basic model of SEIR disease spread which was first developed by Kermack-McKendrick in 1927 [7]. The model was another refinement of the one that had been studied by Abadi, Artiono, and Prawoto [9]. We proposed a mathematical model with the compartments shown in Figure 1. The women population was divided into two age groups, namely children and adult, while each group was also divided into 4 sub-populations, such Susceptible \( (S(t)) \), Exposed \( (E(t)) \), Infected \( (I(t)) \), and Recovered \( (R(t)) \). Some assumptions used were as follows. a) The population is closed and constant with the birth rate equal to the death rate, namely \( \alpha \), b) Vaccines are applied to children population only, c) The vaccine is permanent, individuals who are successfully vaccinated will be immune to rubella with an effectiveness level of \( \theta \), d) Rubella does not cause death of individuals, e) Individual children can transmit rubella to adult and vice versa with the rate of transmission from child to child, child to adult, adult to adult, and adult to child are same, namely \( \beta \), f) Rubella requires incubation time in the infected individual then becomes an infective individual with an infection rate of \( \gamma \), g) Populations recovering from rubella cannot be re-infected or resistant to rubella with a healing rate of \( \delta \), h) Each individual child will turn into an adult at a rate of \( \eta \).

![Figure 1. Compartment Model of Rubella with an age-structured](image)

Regard to the diagram, mathematical model is described as follows

\[
\begin{align*}
\text{Birth Rate: } & \alpha \quad \text{Death Rate: } \alpha N \\
\text{Susceptible (S): } & (1 - \theta) \frac{\beta S(t) E(t)}{N} \\
\text{Exposed (E): } & \delta S(t) + \frac{\beta S(t) E(t)}{N} \\
\text{Infected (I): } & \frac{\beta S(t) E(t)}{N} \\
\text{Recovered (R): } & \delta I(t) + \frac{\gamma I(t)}{N} \\
\end{align*}
\]
Simplify the model by these rescallings:

\[
\begin{align*}
S_a &= S_a, \quad E_a = E_a, \quad I_a = I_a, \quad R_a = R_a, \quad S_d = S_d, \quad E_d = E_d, \quad I_d = I_d, \quad R_d = R_d
\end{align*}
\]

\[
\begin{align*}
\frac{dS_a}{dt} &= \alpha - (1 - \theta)\beta S_a(I_a + I_d) - (\alpha + \theta + \eta)S_a \\
\frac{dE_a}{dt} &= (1 - \theta)\beta S_a(I_a + I_d) - (\alpha + \eta + \delta)E_a \\
\frac{dI_a}{dt} &= \delta E_a - (\alpha + \gamma + \eta)I_a \\
\frac{dR_a}{dt} &= \gamma I_a - (\alpha + \eta)R_a - \alpha R_a \\
\frac{dS_d}{dt} &= \eta S_a - \beta S_d(I_a + I_d) - \alpha S_d \\
\frac{dE_d}{dt} &= \beta S_d(I_a + I_d) + \eta E_a - (\alpha + \delta)E_d \\
\frac{dI_d}{dt} &= \delta E_d + \eta I_a - (\alpha + \gamma)I_d \\
\frac{dR_d}{dt} &= \gamma I_a + \eta R_a - \alpha R_d
\end{align*}
\]

where,

\[
\begin{align*}
\alpha &: \text{quantifying the rate of birth and death per unit time per person} \\
\beta &: \text{quantifying the rate of infection per unit time per person} \\
\theta &: \text{quantifying the rate of the efficacy of the vaccine (0 < \theta < 1)} \\
\delta &: \text{quantifying the rate of incubation per unit time per person} \\
\gamma &: \text{quantifying the rate of maturity per unit time per person} \\
\eta &: \text{quantifying the rate of recovery per unit time per person}
\end{align*}
\]

All parameters were assumed to be positive.

Disease Free Equilibrium and Endemic Equilibrium were determined by setting the right-hand sides of equation (1) equal zero, to obtain the followings.
1. Disease-Free Equilibrium

\[ DFE := (S_\alpha E_\alpha I_\alpha R_\alpha S_d E_d I_d R_d) = \left( \frac{\alpha}{\theta + \alpha + \eta}, 0, 0, \frac{\alpha \theta}{(\theta + \alpha + \eta)(\alpha + \eta)}, \frac{\eta}{\theta + \alpha + \eta}, 0, 0, \frac{\theta \eta}{(\theta + \alpha + \eta)(\alpha + \eta)} \right) \]

2. Endemic Equilibrium

\[ EE := (S_\alpha E_\alpha I_\alpha R_\alpha S_d E_d I_d R_d) \]

where,

\[ S_\alpha^* = \frac{\alpha}{(1 - \theta) \beta (I_\alpha^* + I_d^*) + (\alpha + \theta + \eta)} \]

\[ E_\alpha^* = \frac{(1 - \theta) \beta (I_\alpha^* + I_d^*)}{(\alpha + \eta + \delta)} \]

\[ I_\alpha^* = \frac{\delta E_\alpha^*}{(\alpha + \gamma + \eta)} \]

\[ R_\alpha = \gamma I_\alpha^* + \theta S_\alpha^* \]

\[ S_d^* = \frac{\eta S_\alpha^*}{(\alpha + \eta)} \]

\[ E_d^* = \frac{\beta S_d (I_\alpha^* + I_d^*)}{(\alpha + \delta) - \eta} \]

\[ I_d^* = \frac{\delta E_d^* + \eta I_\alpha^*}{(\alpha + \gamma)} \]

\[ R_d^* = \frac{\gamma I_\alpha^* + \eta R_\alpha}{\alpha} \]

3. Results and Discussion

a. Basic reproduction number

Furthermore, by using next generation matrix (NGM), we found the threshold of the disease as follow. Firstly, we constructed NGM on the population which can spread the disease only, \((E_\alpha, I_\alpha, E_d, I_d)\). It gave us these equations:

\[ \frac{dE_\alpha}{dt} = (1 - \theta) \beta S_\alpha (I_\alpha + I_d) - (\alpha + \eta + \delta) E_\alpha \]

\[ \frac{dI_\alpha}{dt} = \delta E_\alpha - (\alpha + \gamma + \eta) I_\alpha \]

\[ \frac{dE_d}{dt} = \beta S_d (I_\alpha + I_d) + \eta E_\alpha - (\alpha + \delta) E_d \]

\[ \frac{dI_d}{dt} = \delta E_d + \eta I_\alpha - (\alpha + \gamma) I_d \]

Let \( x = (E_\alpha, I_\alpha, E_d, I_d)^T \). \( F \) is a transmission vector and \( V \) is a transition vector. \( F \) and \( V \) can be written as follows.

\[ F = \begin{bmatrix} F_1 \\ F_2 \\ F_3 \\ F_4 \end{bmatrix} = \begin{bmatrix} (1 - \theta) \beta S_\alpha (I_\alpha + I_d) \\ \beta S_d (I_\alpha + I_d) \\ \eta E_\alpha - (\alpha + \delta) E_d \\ 0 \end{bmatrix} \] and \( V = \begin{bmatrix} V_1 \\ V_2 \\ V_3 \\ V_4 \end{bmatrix} = \begin{bmatrix} -(\alpha + \eta + \delta) E_\alpha \\ \delta E_\alpha - (\alpha + \gamma + \eta) I_\alpha \\ \eta E_\alpha - (\alpha + \delta) E_d \\ \delta E_d + \eta I_\alpha - (\alpha + \gamma) I_d \end{bmatrix} \]

Therefore,
\[
\Sigma = \begin{bmatrix}
(\alpha + \eta + \delta) & 0 & 0 & 0 \\
\delta & -(\alpha + \eta) & 0 & 0 \\
\eta & 0 & -(\alpha + \delta) & 0 \\
0 & \eta & \delta & -(\alpha + \gamma)
\end{bmatrix}
\]

Moreover,

\[
\Sigma^{-1} = \frac{-1}{(\alpha + \eta + \delta)} \begin{bmatrix}
0 & 0 & 0 \\
\frac{1}{(\alpha + \eta + \delta)} & 0 & 0 \\
\eta & \frac{1}{(\alpha + \delta)} & 0 \\
\eta & \frac{1}{(\alpha + \gamma + \eta)} & \frac{1}{(\alpha + \gamma)}
\end{bmatrix}
\]

NGM matrix can be found by this formula.

\[
A = -T\Sigma^{-1}
\]

such that

\[
A = -T\Sigma^{-1} = \begin{bmatrix}
\frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \\
\frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \\
\frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \\
\frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \\
\frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} & \frac{(1-\theta)\beta_S \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)}
\end{bmatrix}
\]

Substituting the disease free equilibrium, we have into matrix A, to have

\[
A = \begin{bmatrix}
A_{11} & A_{12} & A_{13} & A_{14} \\
0 & 0 & 0 & 0 \\
A_{31} & A_{32} & A_{33} & A_{34}
\end{bmatrix}
\]

where

\[
A_{11} = \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)}
\]

\[
a_{12} = \frac{1}{\alpha + \eta + \delta} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)}
\]

\[
a_{13} = \frac{1}{\alpha + \eta + \delta} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)}
\]

\[
a_{14} = \frac{1}{\alpha + \eta + \delta} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)} \frac{(1-\theta)\beta_\alpha \delta}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)}
\]
\[a_{14} = \frac{(1-\theta)\beta \theta + \alpha + \eta}{(\alpha + \gamma)^\eta} \]
\[a_{31} = \frac{\beta \theta + \alpha + \eta \delta}{(\alpha + \gamma + \eta)(\alpha + \eta + \delta)} + \frac{\beta \theta + \alpha + \eta \eta \delta(2\alpha + \gamma + \eta)}{(\alpha + \eta + \delta)(\alpha + \gamma + \eta)(\alpha + \gamma)^{(\alpha + \gamma)}} \]
\[a_{32} = \frac{\beta \theta + \alpha + \eta \eta}{(\alpha + \gamma + \eta)(\alpha + \gamma)} \]
\[a_{33} = \frac{\beta \theta + \alpha + \eta \delta}{(\alpha + \gamma + \eta)(\alpha + \gamma)} \]
\[a_{34} = \frac{-\beta \theta + \alpha + \eta}{(\alpha + \gamma)} \]

The largest eigenvalue or spectral radius of \(FV^{-1}\) is the basic reproduction number of the model \([8]\), i.e.:
\[R_0 = \frac{\beta \delta(\alpha + \eta - \alpha \theta)}{(\alpha + \gamma)(\alpha + \delta)(\theta + \alpha + \eta)} \]

**b. Stability analysis**

We analyzed the stability of the system around the disease free equilibrium \(E^0 = (S_a^0, E_a^0, I_a^0, R_a^0, S_d^0, E_d^0, I_d^0, R_d^0)\) and endemic equilibrium \(E^* = (S_a^*, E_a^*, I_a^*, R_a^*, S_d^*, E_d^*, I_d^*, R_d^*)\). It had been done by linearization of the system (1).

Suppose,
\[\frac{dS_a}{dt} = f_1 = (S_a, E_a, I_a, R_a, S_d, E_d, I_d, R_d) = \alpha - (1-\theta)\beta S_a(I_a + I_d) - (\alpha + \theta + \eta)S_a \]
\[\frac{dE_a}{dt} = f_2 = (S_a, E_a, I_a, R_a, S_d, E_d, I_d, R_d) = (1-\theta)\beta S_a(I_a + I_d) - (\alpha + \eta + \delta)E_a \]
\[\frac{dI_a}{dt} = f_3 = (S_a, E_a, I_a, R_a, S_d, E_d, I_d, R_d) = \delta E_a - (\alpha + \gamma + \eta)I_a \]
\[\frac{dR_a}{dt} = f_4 = (S_a, E_a, I_a, R_a, S_d, E_d, I_d, R_d) = \gamma I_a - (\alpha + \eta)R_a + \theta S_a \]
\[\frac{dS_d}{dt} = f_5 = (S_a, E_a, I_a, R_a, S_d, E_d, I_d, R_d) = \eta S_a - \beta S_d(I_a + I_d) - \alpha S_d \]
\[\frac{dE_d}{dt} = f_6 = (S_a, E_a, I_a, R_a, S_d, E_d, I_d, R_d) = \beta S_d(I_a + I_d) + \eta E_a - (\alpha + \delta)E_d \]
\[\frac{dI_d}{dt} = f_7 = (S_a, E_a, I_a, R_a, S_d, E_d, I_d, R_d) = \delta E_d + \eta I_a - (\alpha + \gamma)I_d \]
\[\frac{R_d}{dt} = f_8 = (S_a, E_a, I_a, R_a, S_d, E_d, I_d, R_d) = \gamma I_d + \eta R_a - \alpha R_d \]

By linearization in the disease free equilibrium, the Jacobian matrix had been obtained, as follows.
\[
J = \begin{bmatrix}
-(1-\theta)\beta(I_a + I_d) - (\alpha + \theta + \eta) & 0 & -(1-\theta)\beta S_a & 0 & 0 & 0 & 0 & -(1-\theta)\beta S_a \\
(1-\theta)\beta(I_a + I_d) & -(\alpha + \eta + \delta) & (1-\theta)\beta S_a & 0 & 0 & 0 & 0 & (1-\theta)\beta S_a \\
0 & 0 & -\alpha + \eta + \delta & 0 & 0 & 0 & 0 & 0 \\
\theta & 0 & 0 & \gamma & -\alpha + \eta & 0 & 0 & 0 \\
\eta & 0 & -\beta S_d & 0 & -\beta(I_a + I_d) - \alpha & 0 & -\beta S_d & 0 \\
0 & \eta & \beta S_d & 0 & \beta(I_a + I_d) & -(\alpha + \delta) & \beta S_d & 0 \\
0 & 0 & \eta & 0 & 0 & \delta & -\alpha + \eta & 0 \\
0 & 0 & 0 & 0 & \eta & 0 & \gamma & -\alpha \\
\end{bmatrix}
\]
Furthermore, substitute the disease free equilibrium into the jacobian matrix such that the eigen value of jacobian matrix can be determined, as follows.

\[
\begin{bmatrix}
-(\alpha + \theta + \eta) & 0 & -(1 - \theta)\beta \frac{\alpha}{\theta + \alpha + \eta} & 0 & 0 & 0 & -(1 - \theta)\beta \frac{\alpha}{\theta + \alpha + \eta} & 0 \\
0 & -(\alpha + \eta + \delta) & (1 - \theta)\beta \frac{\alpha}{\theta + \alpha + \eta} & 0 & 0 & 0 & (1 - \theta)\beta \frac{\alpha}{\theta + \alpha + \eta} & 0 \\
0 & \delta & -(\alpha + \gamma + \eta) & 0 & 0 & 0 & 0 & 0 \\
\theta & 0 & \gamma & -(\alpha + \eta) & 0 & 0 & 0 & 0 \\
\eta & 0 & -\beta \frac{\eta}{\theta + \alpha + \eta} & 0 & -\alpha & 0 & -\beta \frac{\eta}{\theta + \alpha + \eta} & 0 \\
0 & \eta & \beta \frac{\eta}{\theta + \alpha + \eta} & 0 & 0 & -(\alpha + \delta) & \beta \frac{\eta}{\theta + \alpha + \eta} & 0 \\
0 & 0 & \eta & 0 & 0 & \delta & -(\alpha + \gamma) & 0 \\
0 & 0 & 0 & \eta & 0 & 0 & \gamma & -\alpha \\
\end{bmatrix}
\]

Again, it was not easy to determine the eigenvalue of the jacobian matrix due to the complexity of equations, however it is still possible to show the result numerically. By using some parameter, the eigenvalue of the jacobian matrix can be seen in the following.

\[
\lambda_1 = -1.30000, \quad \lambda_5 = -0.339325 \\
\lambda_2 = -0.70000, \quad \lambda_6 = -0.80000 \\
\lambda_3 = -0.40000, \quad \lambda_7 = -1.16067 \\
\lambda_4 = -0.40000, \quad \lambda_8 = -1.30000 \\
\]

These results showed that the system near the disease free equilibrium was stable for the parameter used. Furthermore, we linearized in the endemic equilibrium, the Jacobian matrix had been obtained, as follows.

\[
\begin{bmatrix}
-(1 - \theta)\beta (I^e_1 + I^e_2) - (\alpha + \theta + \eta) & 0 & -(1 - \theta)\beta S^e_1 & 0 & 0 & 0 & -(1 - \theta)\beta S^e_2 & 0 \\
(1 - \theta)\beta (I^e_1 + I^e_2) & -(\alpha + \eta + \delta) & (1 - \theta)\beta S^e_1 & 0 & 0 & 0 & (1 - \theta)\beta S^e_2 & 0 \\
0 & \delta & -(\alpha + \gamma + \eta) & 0 & 0 & 0 & 0 & 0 \\
\theta & 0 & \eta & -(\alpha + \eta) & 0 & 0 & 0 & 0 \\
\eta & 0 & -\beta S^e_1 & 0 & -(\alpha + \delta) & -\beta S^e_2 & 0 & 0 \\
0 & \eta & \beta S^e_1 & 0 & \beta (I^e_1 + I^e_2) & -(\alpha + \delta) & \beta S^e_2 & 0 \\
0 & 0 & \eta & 0 & 0 & \delta & -(\alpha + \gamma) & 0 \\
0 & 0 & 0 & \eta & 0 & 0 & \gamma & -\alpha \\
\end{bmatrix}
\]

Again, it was not easy to determine the eigenvalue of the jacobian matrix due to the complexity of equations, however it is still possible to show the result numerically. By using some parameter, the eigenvalue of the jacobian matrix can be seen in the following.

\[
\lambda_1 = -1.00000, \quad \lambda_5 = -0.80000 \\
\lambda_2 = -0.70000, \quad \lambda_6 = -1.00000 \\
\lambda_3 = -0.10000, \quad \lambda_7 = -7.36921 \\
\lambda_4 = -0.10000, \quad \lambda_8 = -0.13692 \\
\]

These results showed that the system near the endemic equilibrium was also stable for the parameter used.

c. Numerical simulation

Finally, we simulated data numerically to figure out the consistency of the results obtained above with numerical solution. The simulation had been done for some initial condition near the disease free equilibrium and endemic equilibrium. By using parameters value set in Table 1, our simulation can be seen in Figure 2.
Table 1. Parameter Value

| Parameter | Parameter value |
|-----------|-----------------|
| $\eta$    | 0.3-0.6         |
| $\delta$  | 0.3-0.6         |
| $\gamma$  | 0.1             |
| $\beta$   | 0.5-0.9         |
| $\theta$  | 0.3-0.6         |
| $\alpha$  | 0.1-0.4         |

Figure 2. Numerical simulation (a) near the disease free equilibrium and (b) near endemic equilibrium

In Figure 2 (a) it can be seen that any initial conditions near the disease free equilibrium will go up to the critical point. Susceptible children, recovered children, susceptible adults, and recovered adults will exist for long time period ($t \to \infty$), while exposed children, infected children, exposed adults, and infected adults will be extinct. This solution gives the same result as analytical solution. Meanwhile, in Figure 2 (b) for any initial conditions near the endemic equilibrium also give the same result. For time goes to infinity, an exposed children, infected children, exposed adults, and infected adults will be exist. Even though, it just give a small number of people, this result also can be used to justify that numerical and analytical solution have the same behaviour.

This simulation also confirm that once an infected person found in the population then the disease will be stay in that population forever. This last result will affect to the incidence of infants with CSR, as we have shown in the results in [9].

4. Conclusions

The rubella mathematical model developed using the age structure had a threshold value for the spread of disease, $R_0 = \frac{\beta \delta (\alpha + \theta - \mu \delta)}{(\alpha + \gamma)(\theta + \alpha + \eta)\eta}$. This study also produced a disease-free equilibrium and endemic equilibrium. Moreover, the analysis of the model's stability at the equilibrium point was carried out numerically using some known parameters. It had been done numerically due to the complexity of equation. With the choice of parameters it had been obtained that all eigenvalues are negative and it means that these two equilibria were stable. These results were confirmed by the numerical simulations that have been carried out. It is interesting to compare between the number of infected adult women in this model with those in the model in [9], since it will lead to the conclusion about the effectiveness of the vaccination program when it is implemented to child-bearing age women population [9] or to the younger ones, as shown in this study.
5. References

[1] Baleanu D, Mohammadi H, and Rezapour S 2020 A mathematical theoretical study of a particular system of Caputo–Fabrizio fractional differential equations for the Rubella disease model. Adv Differ Equ 2020, 184 (2020). https://doi.org/10.1186/s13662-020-02614-z

[2] Jin-Sook Wang, Hye Min Lee, Su Jin Kim, Jun-Sub Kim, Chun Kang, Chae won Jung, Hye kyung In, Dong Hee Seo, Dong Han Lee, Yoon-Seok Chung 2020 Laboratory confirmation of congenital rubella syndrome in South Korea in 2017: A genomic epidemiological investigation, Vaccine, 2020, ISSN 0264-410X, https://doi.org/10.1016/j.vaccine.2020.08.064.

[3] Ki-Hyun Kim, Ehsanul Kabir, Shamin Ara Jahan 2018 Airborne bioaerosols and their impact on human health, Journal of Environmental Sciences, Volume 67, 2018, Pages 23-35, ISSN 1001-0742, https://doi.org/10.1016/j.jes.2017.08.027.

[4] Athira P. S, Hanna Simon, T. Sivakumar 2019 Congenital Rubella Syndrome-A Case Report, International Journal of Research & Review (www.ijrrjournal.com) 421 Vol.6; Issue: 11; November 2019, E-ISSN: 2349-9788; P-ISSN: 2454-2237

[5] Demeke Mekonnen 2017 Clinically confirmed congenital rubella syndrome: the role of echocardiography, Ethiopian Journal of Health Sciences, DOI: 10.4314/ejhs.v27i2.13, 2017-03-15

[6] Elizabeth Siti Herini, Gunadi, Agung Triono, Asal Wahyuni Erlin Mulyadi, Niprida Mardin, Rusipah, Yati Soenarto, Susan E. Reef 2017 Hospital-based surveillance of congenital rubella syndrome in Indonesia, Eur J Pediatr (2017) 176:387–393, DOI 10.1007/s00431-017-2853-8

[7] Kermack, W. O. and McKendrick, A. G. 1927 Contributions to the mathematical theory of epidemics, part I. Proc. Roy. Soc. Edin. A, 115, 710-721.

[8] van den Driessche P., Watmough J. 2008 Further Notes on the Basic Reproduction Number. In: Brauer F., van den Driessche P., Wu J. (eds) Mathematical Epidemiology. Lecture Notes in Mathematics, vol 1945. Springer, Berlin, Heidelberg. https://doi.org/10.1007/978-3-540-78911-6 6

[9] Abadi, R. Artiono, B. P. Prawoto 2019 The Effects of Vaccination to the Dynamics of Rubella Virus with Seasonality, Vol. 2020. https://doi.org/10.28919/cmbn/4422.

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