

Available online at http://scik.org Commun. Math. Biol. Neurosci. 2023, 2023:137 https://doi.org/10.28919/cmbn/8317 ISSN: 2052-2541

DYNAMICS OF A FRACTIONAL-ORDER RUBELLA DISEASE MODEL WITH VERTICAL TRANSMISSION AND SATURATED INCIDENCE RATE

EMBUN FIVI ELIVINA*, WURYANSARI MUHARINI KUSUMAWINAHYU, MARSUDI

Department of Mathematics, Faculty of Mathematics and Natural Sciences,

State University of Brawijaya, Malang 65145, Indonesia

Copyright © 2023 the author(s). This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract. Rubella is one of the viruses responsible for rubella disease. If the rubella virus infects a pregnant woman during the first trimester of pregnancy, it causes CRS (the virus transmits vertically from mother to fetus). In this paper, we study the rubella disease model with a fractional-order derivative and saturated incidence rate. Infectious diseases have a history in their transmission dynamics, thus non-local operators such as fractional-order derivatives play a vital role in modeling the dynamics of such epidemics. First, we analyze the important mathematical features of the proposed model, such as the existence and uniqueness, the non-negativity and boundedness of solutions. Then, the equilibrium point, basic reproduction number, and stability of the equilibrium points are also investigated. The model has two equilibrium points, namely the disease-free equilibrium and endemic equilibrium. The disease-free equilibrium point always exists, while the endemic equilibrium point exists if $R_0 > 1$. The disease-free equilibrium point is locally asymptotically stable if the Routh-Hurwitz criterion is satisfied. Numerical simulation is done by using the Grunwald-Letnikov approximation method to confirm the results of analytical calculations.

Keywords: rubella; fractional-order; saturated incidence rate; equilibrium points; local stability.

2020 AMS Subject Classification: 92C60.

E-mail address: embunfive@student.ub.ac.id

^{*}Corresponding author

Received November 03, 2023

1. INTRODUCTION

Rubella virus is one of the viruses that cause a disease known as German measles or three-day measles [1]. Rubella is characterized by fever and the appearance of a mild rash that resembles the symptoms of measles [2]. Nearly 50% of people infected with rubella are asymptomatic. Symptoms typically begin on the face and spread to other parts of the body if a person experiences them [3]. Rubella infection is often considered a mild disease. However, if rubella virus infects a pregnant woman during the first trimester, it can lead to Congenital Rubella Syndrome (CRS), leading to significant pregnancy complications, including birth defects in babies, and in some cases, fetal death[4]. Rubella vaccination can prevent CRS cases. According to the recommendations of the Centers for Disease Control and Prevention (CDC), children should receive two doses of the MMR (Measles, Mumps and Rubella) vaccine [5].

Mathematical modeling is a valuable tool for depicting and analyzing dynamic behaviors, transmission mechanisms, predictive control strategies, and generating simulations of the spread of infectious diseases over time [6]. Many autors have studied the dynamics and transmission of rubella disease model see [7, 8, 9]. Tilahun et al. extended the model developed [8] by adding a protected subpopulations according to [5], individuals who receive two doses of the MMR vaccine will have active immunity, rendering them immune to the Rubella virus for life, and they are included in the protected populations [10].

The models developed by previous autors were constructed by using integer-order derivative approaches, which do not have memory effects for accurate predictions [11]. Fractional order derivative is a theory of fractional calculus with nonlocal properties, where the next state of the model depends not only on the current state but also on all previous states [12]. Memory effect plays a crucial role in disease transmission [13]. Many autors have studied fractional-order Rubella disease models, see references [14, 15, 16].

Incidence (rate of new infections) plays a very important role in modeling infectious diseases. Capasso and Serio [17] introduced the non-linear incidence rate is referred to as the saturated incidence rate, i.e., $\frac{\beta I}{1+cI}$, where βI Measuring the infection of the disease and $\frac{1}{1+cI}$ measuring the inhibiting effect from the behavioral change of the susceptible individuals. As far as we know, no studies have been conducted on mathematical models of a fractionalorder rubella disease with saurated incidence rate dynamics under the consideration of vertical transmission and second vaccine in their model. The remaining parts of this paper is organized as: In Section 2, we will show the formulation of fractional-order rubella disease model. Section 3 we analyze the important mathematical features of the proposed model, such as the existence and uniqueness, the non-negativity and boundedness of solutions. In Section 4, equilibrium point and basic reproduction number. In Section 5, local stability, in Section 6, numerical simulations. Finally, in Section 7, conclusions about our work.

2. MODEL FORMULATION

In this paper we consider an epidemiological model adapted from [11], by assuming the disease spread rate follows a saturated infection rate $\frac{\beta SI}{1+cI}$. Based on the behavior of rubella disease, total population size a given time *t*, denote by N(t), is divided into six compartment, namely: Sussceptible S(t), Vaccinated V(t), Protected P(t), Exposed E(t), Infected I(t), Recovered R(t). The susceptible compartment includes a group of individuals who have not yet contracted rubella but are vulnerable to becoming infected. The vaccinated compartment encompasses individuals who have received their first vaccine dose. The protected compartment encompasses individuals who have received the second doses of the MMR vaccine and possess active immunity, and will remain free from rubella throughout their lives [5]. The exposed compartment pertains to individuals who are susceptible and come into contact with infected individuals, including those who are asymptomatic carriers. The infected compartment includes those who have acquired temporary immunity. The proposed model using six compartment are shown in Figure 1.



FIGURE 1. Diagram of Rubella disease

From the compartment diagram, the proposed model is expressed in a differential equations is formulated in model (1).

(1)

$$\frac{dS}{dt} = \Lambda + \rho R + \omega V - \frac{\beta SI}{1 + cI} - (\sigma + \mu)S,$$

$$\frac{dV}{dt} = \sigma S - (\delta + \omega + \mu)V,$$

$$\frac{dP}{dt} = \delta V - \mu P,$$

$$\frac{dE}{dt} = \frac{\beta SI}{1 + cI} - (\vartheta + \mu)E,$$

$$\frac{dI}{dt} = \theta I + \vartheta E - (\gamma + \varepsilon + \mu)I,$$

$$\frac{dR}{dt} = \gamma I - (\rho + \mu)R,$$

with initial conditions $S(0) \ge 0$, $V(0) \ge 0$, $P(0) \ge 0$, $E(0) \ge 0$, $I(0) \ge 0$, and $R(0) \ge 0$, are positive. To include the memory effect in model (1), we apply fractional-order derivative $^{C}D_{t}^{\alpha}$, to get the following model.

$${}^{C}D_{t}^{\alpha}S = \Lambda + \rho R + \omega V - \frac{\beta SI}{1+cI} - (\sigma + \mu)S = H_{1}(X),$$

$${}^{C}D_{t}^{\alpha}V = \sigma S - (\delta + \omega + \mu)V = H_{2}(X),$$

$${}^{C}D_{t}^{\alpha}P = \delta V - \mu P = H_{3}(X),$$

$${}^{C}D_{t}^{\alpha}E = \frac{\beta SI}{1+cI} - (\vartheta + \mu)E = H_{4}(X),$$

(2)

$${}^{C}D_{t}^{\alpha}I = \theta I + \vartheta E - (\gamma + \varepsilon + \mu)I = H_{5}(X),$$
$${}^{C}D_{t}^{\alpha}R = \gamma I - (\rho + \mu)R = H_{6}(X),$$

with initial conditions $S(0) \ge 0$, $V(0) \ge 0$, $P(0) \ge 0$, $E(0) \ge 0$, $I(0) \ge 0$, and $R(0) \ge 0$, are positive.

In this article we study the dynamics of the model (2) with ${}^{C}D_{t}^{\alpha}$ is the Caputo operator. Parameter description of model (2) could be shown in Tabel 1.

Parameter	Description	Value	Source
Λ	Recruitment rate	374.125	[16]
β	Contact rate	0.004	[16]
ϑ	Exposure rate	0.85	[10]
ε	Death rate due to rubella disease	0.08	[10]
γ	Recovery rate	0.15	[10]
θ	Rate of infected infants	0.55	[10]
ρ	Rate of temporary immunity	0.01	[10]
ω	Waning rate of first vaccination dose	0.6	[10]
δ	Rate of second vaccination dose	1	[10]
μ	Natural death rate	0.4	[14]
σ	Rate of first vaccination dose	0.3	[15]
С	saturation constant	0.001	Assumed

TABLE 1. Parameter value for the numerical simulations

3. EXISTENCE AND UNIQUENESS, NON-NEGATIVITY AND BOUNDEDNESS SOLUTION

Firstly, we need to investigate the properties of solution of the model (2), such as the existence and uniqueness, non-negativity, and boundedness of the solutions. Hence we need to prove that model (2) satisfies the Lipschitz condition.

Theorem 1. Let $\Omega := \{(S,V,P,E,I,R) \in \mathbb{R}^6 : \max\{|S|,|V|,|P|,|E|,|I|,|R|\} \leq M\}$ and model (2) can be expressed as ${}^CD_t^{\alpha}X = \vec{H}(X)$ where X = (S,V,P,E,I,R) and $\vec{H}(X) =$

 $(H_1(X), H_2(X), H_3(X), H_4(X), H_5(X), H_6(X))$. $\vec{H}(X)$ satisfies Lipschitz condition, i.e, $\exists L > 0$ such that $|\vec{H}(X) - \vec{H}(\bar{X})| < L|X - \bar{X}|$, for $X, \bar{X} \in \Omega$.

Theorem 2. For each initial value $\{S_0, V_0, P_0, E_0, I_0, R_0\} \in \mathbb{R}^6_+$ in Ω , model (2) has a unique solution in $\Omega \times (0, \infty]$ for all t > 0.

Proof. Let $X, \overline{X} \in \Omega$, then

$$\begin{split} ||\vec{H}(X) - \vec{H}(\bar{X})|| &= |H_1(X) - H_1(\bar{X})| + |H_2(X) - H_2(\bar{X})| + |H_3(X) - H_3(\bar{X})| + |H_4(X) - H_4(\bar{X})| \\ &+ |H_5(X) - H_5(\bar{X})| + |H_6(X) - H_6(\bar{X})| \\ &\leq (2\beta M(1 + cM) + (2\sigma + \mu)|S - \bar{S}| + (2\delta + 2\omega + \mu)|V - \bar{V}| \\ &+ \mu|P - \bar{P}| + (2\vartheta + \mu)|E - \bar{E}| + (2(\beta M + \gamma) + \theta + \varepsilon + \mu)|I - \bar{I}| \\ &+ (2\rho + \mu)|R - \bar{R}| \\ &\leq L||X - \bar{X}|| \end{split}$$

where $L = \max \left\{ (2\beta M(1+cM) + (2\sigma + \mu), (2\delta + 2\omega + \mu), \mu, (2\vartheta + \mu), (2(\beta M + \gamma) + \theta + \varepsilon + \mu), (2\rho + \mu). \text{ Hence, } \vec{H}(X) \text{ satisfies Lipschitz's condition. Based on Lemma 2 in [18], for each initial value of <math>(S_0, V_0, P_0, E_0, I_0, R_0) \in \mathbb{R}^6_+$ in Ω , there is a unique solution in Ω of model (2) for all t > 0 and the Theorem 1 and 2 are well proven. \Box

Model (2) describe an epidemiological model in fractional order differential equations. Therefore, the solution of model (2) must be bounded and non-negative. Based on Theorem 5 in [19], model (2) with $\alpha = 1$ and locally Lipschitz, then the model (2) satisfies the positivity property.

Theorem 3. For all t > 0 solutions of model (2) are non-negative and uniformly bounded, with $(S(0) \ge 0, V(0) \ge 0, P(0) \ge 0, E(0) \ge 0, I(0) \ge 0, R(0) \ge 0.$

Proof.

$$\frac{dS(t)}{dt} = \Lambda + \rho R + \omega V - \frac{\beta IS(t)}{1 + cI} - (\sigma + \mu)S(t),$$
$$\frac{dS(t)}{dt} \ge -\frac{\beta SI}{1 + cI} - (\sigma + \mu)S,$$

DYNAMICS OF A FRACTIONAL-ORDER RUBELLA DISEASE MODEL

$$\int \frac{dS(t)}{S(t)} \ge -\int \left(\frac{\beta I(t)}{1+cI(t)} + \sigma + \mu\right) dt,$$

$$\ln|S(t)| \ge -\int \left(\frac{\beta I(t)}{1+cI(t)} + \sigma + \mu\right) dt + C,$$

$$S(t) \ge S(0)exp\left(-\int \left(\frac{\beta I(t)}{1+cI(t)} + \sigma + \mu\right) dt\right),$$

In the same process, the non-negativity of the other compartments are proved and we get the following solutions, $V(t) \ge V(0) \exp(-(\delta + \omega + \mu)t)$, $P(t) \ge P(0) \exp(-\mu t)$, $E(t) \ge E(0) \exp(-(\vartheta + \mu)t)$, $I(t) \ge I(0) \exp(-(\gamma + \mu + \varepsilon)t)$, $R(t) \ge R(0) \exp(-(\mu + \rho)t)$. Since the value of the exponential function is always positive, then S(t) > 0, V(t) > 0, P(t) > 0, E(t) > 0, I(t) > 0, R(t) > 0.

Now, we need to show the boundedness of solution. We denote N(t) as the total population, then

$$N(t) = S(t) + V(t) + P(t) + E(t) + I(t) + R(t)$$
$$= \Lambda - \mu N - (\varepsilon + \theta)I$$
$$\leq \Lambda - \mu N$$

Based on Lemma 3 in [20], we obtained

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right) E_{\alpha}[-\mu t^{\alpha}],$$

where E_{α} is the Mittag-Leffler function. Since $E_{\alpha}[-\mu t^{\alpha}]to0$ as $t \to \infty$, (see [21] Lemma 5 and Corollary 6). We have that N(t) is convergent to $\frac{\Lambda}{\mu}$ for $t \to \infty$. Therefore, all solutions of model (2) with non-negative initial conditions are confined to the region Φ , where

$$\Phi := \left\{ (S, V, P, E, I, R) \in \mathbb{R}^6 : N(t) \le \frac{\Lambda}{\mu} \right\}$$

4. EQUILIBRIUM POINT AND BASIC REPRODUCTION NUMBER

In this section, we determine the equilibrium point and basic reproduction number. To simplify he model (2), we use the new symbols as $\phi_1 = \sigma + \mu$, $\phi_2 = \delta + \omega + \mu$, $\phi_3 = \vartheta + \mu$, $\phi_4 = \gamma + \varepsilon + \mu$, $\phi_5 = \rho + \mu$. There are two equilibrium points, that are disease-free equilibrium point and endemic equilibrium point.

Disease-free equilibrium point
$$X^0 = \left(\frac{\Lambda\phi_2}{\phi_1\phi_2 - \sigma\omega}, \frac{\Lambda\sigma}{\phi_1\phi_2 - \sigma\omega}, \frac{\Lambda\sigma\delta}{(\phi_1\phi_2 - \sigma\omega)\mu}, 0, 0, 0\right)$$
, and

endemic equilibrium point $X^* = (S^*, V^*, P^*, E^*, I^*, R^*)$. where

$$S^{*} = \frac{(\phi_{2}\phi_{5}\Lambda + \phi_{2}\rho\gamma I^{*})(1 + cI^{*})}{\phi_{5}\left(\phi_{2}\beta I^{*} + (\phi_{1}\phi_{2} - \sigma\omega)(1 + cI^{*})\right)}, V^{*} = \frac{(\sigma\phi_{5}\Lambda + \sigma\rho\gamma I^{*})(1 + cI^{*})}{\phi_{5}\left(\phi_{2}\beta I^{*} + (\phi_{1}\phi_{2} - \sigma\omega)(1 + cI^{*})\right)}, V^{*} = \frac{\delta\sigma(\phi_{5}\Lambda + \sigma\rho\gamma I^{*})(1 + cI^{*})}{\mu\phi_{5}\left(\phi_{2}\beta I^{*} + (\phi_{1}\phi_{2} - \sigma\omega)(1 + cI^{*})\right)}, E^{*} = \frac{\beta(\phi_{2}\phi_{5}\Lambda + \phi_{2}\rho\gamma I^{*})}{\phi_{3}\phi_{5}\left(\phi_{2}\beta I^{*} + (\phi_{1}\phi_{2} - \sigma\omega)(1 + cI^{*})\right)}, I^{*} = \frac{\phi_{3}\phi_{4}\phi_{5}(\phi_{1}\phi_{2} - \sigma\omega)(1 + cI^{*})}{\phi_{3}\phi_{5}\left(\phi_{2}\beta I^{*} + (\phi_{1}\phi_{2} - \sigma\omega)(1 + cI^{*})\right)}, R^{*} = \frac{\gamma I^{*}}{\phi_{5}}$$

The endemic equilibrium point X^* exist when $\phi_3\phi_5(\phi_2\beta + c(\phi_1\phi_2 - \sigma\omega))(\phi_4 - \theta) - \vartheta\beta\phi_2\rho\gamma > 0$ and $R_0 > 1$, where $R_0 = \frac{\beta\vartheta\Lambda\phi_2 + \theta\phi_3(\phi_1\phi_2 - \sigma\omega)}{\phi_3\phi_4(\phi_1\phi_2 - \sigma\omega)}$. The basic reproduction number is obtained by using the next generation matrix method by [22].

5. STABILITY ANALYSIS

The model (2) is a nonlinear autonomus sytem. Local stability of disease-free equilibrium and endemic equilibrium points are performed by linearizing the nonlinear system.

$$(3) J(X^*) = \begin{bmatrix} -\frac{\beta I^*}{1+cI^*} - \phi_1 & \omega & 0 & 0 & -\frac{\beta S^*}{(1+cI^*)^2} & \rho \\ \sigma & -\phi_2 & 0 & 0 & 0 \\ 0 & \delta & -\mu & 0 & 0 & 0 \\ \frac{\beta I^*}{1+cI^*} & 0 & 0 & -\phi_3 & \frac{\beta S^*}{(1+cI^*)^2} & 0 \\ 0 & 0 & 0 & \vartheta & \theta - \phi_4 & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\phi_5 \end{bmatrix}$$

Theorem 4. The disease-free equilibrium point of the model (2) is locally asymtotically stable if $R_0 < 1$.

Proof. To prove the theorem (4) we compute the Jacobian matrix at disease-free equilibrium point (X^0) has the eigenvalues $\lambda_1 = -\mu < 0, \lambda_2 = -\phi_5 < 0$. Therefore for all values of $\alpha \in (0,1], \tan^{-1}\left(\frac{Im(\lambda_{1,2})}{Re(\lambda_{1,2})}\right) = \pi > \frac{\alpha\pi}{2}$, and the other four eigenvalues $\lambda_i, i = 2, 3, 4, 5, 6$ are

determined by two characteristic equations.

(4)
$$(\lambda^2 + d_1\lambda + d_2)(\lambda^2 + e_1\lambda + e_2) = 0$$

Next, we solve the following equation.

(5)
$$(\lambda^2 + d_1\lambda + d_2) = 0$$

where

$$d_1 = \phi_1 + \phi_2,$$
$$d_2 = \phi_1 \phi_2 - \sigma \omega.$$

Based on Proposition 1 (ii) in [23], the eigenvalues $\lambda_k, k = 3, 4$ will satisfy $|arg(\lambda_k)| = \pi > \frac{\alpha \pi}{2}$, if $d_1 > 0$ dan $d_2 > 0$.

The other two eigenvalues are determined by the following equation.

$$(\delta) \qquad (\lambda^2 + e_1\lambda + e_2) = 0$$

where

$$e_{1} = \phi_{3} + \phi_{4} - \theta,$$

$$e_{2} = \phi_{3}(\phi_{4} - \theta) - A\vartheta$$

$$A = \frac{\beta \Lambda \phi_{2}}{\phi_{1} \phi_{2} - \sigma \omega}$$

By using the same conditions as in the previous proof, the eigenvalues $\lambda_l, l = 5, 6$. will satisfy $|arg(\lambda_l)| = \pi > \frac{\alpha \pi}{2}$, if $e_1 > 0$ and $e_2 = \phi_3(\phi_4 - \theta) - A\vartheta > 0$, or $R_0 < 1$. According to Matignon's condition or Theorem 2 in [24] X^0 is locally asymptotically stable.

Theorem 5. The disease-free equilibrium point of the model (2) is locally asymtotically stable if $R_0 > 1$.

Proof. Stability of endemic equilibrium point X^* , by computing X^* in (3), let $B = \frac{\beta I^*}{1 + cI^*}$ and $D = \frac{\beta S^*}{(1 + cI^*)^2}$, the eigen values of the Jacobian matrix $J(X^*)$ can be written as (7) $(\lambda + \mu)(\lambda^5 + b_1\lambda^4 + b_2\lambda^3 + b_3\lambda^2 + b_4\lambda + b_5)$ where

$$\begin{split} b_1 &= B - \theta + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5, \\ b_2 &= B(-\theta + \phi_2 + \phi_3 + \phi_4 + \phi_5) - D\vartheta - \sigma\omega - \theta(\phi_1 + \phi_2 + \phi_3 + \phi_5) + \phi_1(\phi_2 + \phi_3 + \phi_4 + \phi_5) \\ &+ \phi_2(\phi_3 + \phi_4 + \phi_5) + \phi_3(\phi_4 + \phi_5) + \phi_4\phi_5, \\ b_3 &= B\theta(-\phi_2 - \phi_3 - \phi_5) + B\phi_2(\phi_3 + \phi_4 + \phi_5) + B\phi_3(\phi_4 + \phi_5) + B(\phi_4\phi_5) - D\vartheta(\phi_1 + \phi_2 + \phi_5) \\ &+ \sigma\omega(\theta - \phi_3 - \phi_4 - \phi_5) - \theta(\phi_1(\phi_2 + \phi_3 + \phi_5) + \phi_2(\phi_3 + \phi_5)\phi_3\phi_5) + \phi_1\phi_2(\phi_3 + \phi_4 + \phi_5), \\ &+ \phi_1\phi_3(\phi_4 + \phi_5) + \phi_4(\phi_1\phi_5 + \phi_2\phi_3) + \phi_2\phi_5(\phi_3 + \phi_4) + \phi_3\phi_4\phi_5, \\ b_4 &= -B\gamma\rho\vartheta - B\theta\phi_2(\phi_3 + \phi_5) - B\theta\phi_3\phi_5 + B\phi_2\phi_3(\phi_4 + \phi_5) + B\phi_4\phi_5(\phi_2 + \phi_3) + D\vartheta(\sigma\omega \\ &- \phi_1\phi_2 - \phi_1\phi_5 - \phi_2\phi_5) + \sigma\omega(\theta(\phi_3 + \phi_5) - \phi_3(\phi_4 + \phi_5) - \phi_4\phi_5) - \theta(\phi_1\phi_2(\phi_3 + \phi_5) \\ &+ \phi_3\phi_5(\phi_1 + \phi_2)) + \phi_1\phi_2(\phi_3\phi_4 + \phi_3\phi_5 + \phi_4\phi_5) + \phi_3\phi_4\phi_5(\phi_1 + \phi_2), \\ b_5 &= -B(\phi_2(\gamma\rho\vartheta + \theta\phi_3\phi_5) - \phi_3\phi_4\phi_5) + D\vartheta\phi_5(\sigma\omega - \phi_1\phi_2) + \sigma\omega\phi_3\phi_5(\theta - \phi_4) \\ &- \phi_1\phi_2\phi_3\phi_5(\theta - \phi_4). \end{split}$$

According to the Routh-Hurwitz criterion [25], we find that if, only if, the coefficients $b_k > 0$ for $k = 1, 2, 3, 4, 5, b_1b_2 - b_3 > 0, b_1b_2b_3 - b_1^2b_4 - b_3^2 > 0$, and $(b_1b_2 - b_3)(b_3b_4 - b_2b_5) - (b_5 - b_1b_4)^2 > 0$, then all roots of equations (7) have negative real parts. When $\alpha \in (0, 1]$ the endemic aquilibrium point is locally asymptotically stable if all roots of equation (7) satisfy $|arg(\lambda_k)| > \frac{\alpha\pi}{2}$.

6. NUMERICAL SIMULATIONS

In this section, we will present some numerical simulation results of the model (2) using the Grunwald-Letnikov scheme developed by Scherer [26]. Based on Grunwald Letnikov's explicit, the numerical equation form of model (2) is

$$D_{t}^{\alpha}S(t_{n+1}) = h^{\alpha}\left(\Lambda + \rho R_{n} + \omega V_{n} - \frac{\beta S_{n}I_{n}}{1 + cI_{n}}\right) + \sum_{k=1}^{n+1} (-1)^{k} \binom{\alpha}{k} S_{n+1-k} + \frac{(n+1)^{-\alpha}}{\Gamma(1-\alpha)} S_{0},$$
(8)
$$D_{t}^{\alpha}V(t_{n+1}) = h^{\alpha}(\sigma S_{n} - (\delta + \omega + \mu)V_{n}) + \sum_{k=1}^{n+1} (-1)^{k} \binom{\alpha}{k} V_{n+1-k} + \frac{(n+1)^{-\alpha}}{\Gamma(1-\alpha)} V_{0},$$

$$D_{t}^{\alpha}P(t_{n+1}) = h^{\alpha}(\delta V_{n} - \mu P_{n}) + \sum_{k=1}^{n+1} (-1)^{k} \binom{\alpha}{k} P_{n+1-k} + \frac{(n+1)^{-\alpha}}{\Gamma(1-\alpha)} P_{0},$$

$$D_{t}^{\alpha}E(t_{n+1}) = h^{\alpha} \left(\frac{\beta S_{n}I_{n}}{1 + cI_{n}} + (\vartheta + \mu)E_{n}\right) + \sum_{k=1}^{n+1} (-1)^{k} \binom{\alpha}{k} E_{n+1-k} + \frac{(n+1)^{-\alpha}}{\Gamma(1-\alpha)} E_{0},$$

$$D_{t}^{\alpha}I(t_{n+1}) = h^{\alpha}(\theta I_{n} + \vartheta E - (\gamma + \varepsilon + \mu)I_{n}) + \sum_{k=1}^{n+1} (-1)^{k} \binom{\alpha}{k} I_{n+1-k} + \frac{(n+1)^{-\alpha}}{\Gamma(1-\alpha)} I_{0},$$

$$D_{t}^{\alpha}R(t_{n+1}) = h^{\alpha}(\gamma I_{n} - (\rho + \mu)R_{n}) + \sum_{k=1}^{n+1} (-1)^{k} \binom{\alpha}{k} R_{n+1-k} + \frac{(n+1)^{-\alpha}}{\Gamma(1-\alpha)} R_{0}.$$

6.1. Equilibrium Points Simulation

Using the parameter values in Table 1 except $\beta = 0$, we get $R_0 = 0.873015 < 1$, the basic reproduction number shows that the endemic equilibrium point does not exist. By implementing parameter values in Table 1, and the initial value are set to be $N_1(0) = (360,90,135,125,95,130)$, $N_2(0) = (400,205,180,280,195,220)$, $N_3(0) =$ (380,105,150,95,105,170). We obtained $X^0 = (613.31,91.99,229.99,0,0,0)$ and we get the eigenvalues λ_i , i = 1,2,3,4,5,6 are $\lambda_i = (-0.4, -0.41, -2.13, -0.57, -1.29, -0.03)$. The simulation results are represented in Figure 2.



FIGURE 2. Phase potrait SVP and EIR using parameter in Table 1 with $\alpha = 0.9$ for $R_0 < 1$

Based on the Figure 2, we can observed that from the different initial values, the solution orbit in the S, V, P phase potrait and E, I, R phase potrait, converge towards X^0 . Therefore the infection does not exist and the rubella virus will not appear in the future.

To show the influence of memory effects, a simulation is conducted with parameter values

in Tabel 1 and for some values of α ($\alpha = 0.4, 0.6, 0.8, 0.9, 1$). The simulation results are represented in Figure 3.



FIGURE 3. Plots for $R_0 < 1$ corresponding to different values of $\alpha = 0.4, 0.6, 0.8, 0.9, 1$

We can observe from the Figure 3 that the curves of each compartment have the same trend when α is changed. However, A higher alpha value leads to faster convergence of the solution curves. That is to say the value of α has a crucial effect on the dynamics of the system.

Next, simulation using the parameter values in Table 1 and we get $R_0 = 3.522 > 1$. The basic reproduction number shows that the spread of Rubella disease always exists. Then implementing the parameter values in system (8), we get $X^* = (108.8, 16.33, 40.80, 254, 2701, 988.3)$. To show the stability of the equilibrium point, this simulation produces successive Routh-Hurwitz criterion values $b_1 = 7.358 > 0$, $b_1b_2 - b_3 = 112.4 > 0$, $b_1b_2b_3 - b_1^2b_4 - b_3^2 = 1528 > 0$, and $(b_1b_2 - b_3)(b_3b_4 - b_2b_5) - (b_5 - b_1b_4)^2 = 6694 > 0$. Therefore, the endemic equilibrium points are locally asymptotically stable. The simulation results are represented by Figure 4.

DYNAMICS OF A FRACTIONAL-ORDER RUBELLA DISEASE MODEL



FIGURE 4. Phase potrait SVP and EIR using parameter in Table 1 with $\alpha = 0.9$ for $R_0 > 1$

To show the influence of memory effects, a simulation is conducted with parameter values in Tabel 1 and for some values of α ($\alpha = 0.6, 0.8, 0.9, 1$). The simulation results are represented in Figure 5.



FIGURE 5. Plots for $R_0 > 1$ corresponding to different values of $\alpha = 0.6, 0.8, 0.9, 1$

7. CONCLUSIONS

We introduce the fractional-order rubella disease model with vertical transmission, consists of six compartmens: susceptible (S), vaccinated (V), protected (P), exposed (E), infected (I), and recovered (R). The incidence rate used is the saturated incidence rate. The properties of the model's solutions, including the existence and uniqueness of solutions, have been established. The model's solutions are consistently positive and bounded within the domain Φ . The disease-free equilibrium point always exists and is locally asymptotically stable if $R_0 < 1$ and satisfies the Routh-Hurwitz criteria. Conversely, if $R_0 > 1$, the endemic equilibrium point exists and is locally asymptotically stable if it satisfies the Routh-Hurwitz criterion. The numerical simulations conducted demonstrate results consistent with the dynamic analysis, with solution curves convergent to the equilibrium point.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

REFERENCES

- J.Y. Lee, D.S. Bowden, Rubella virus replication and links to teratogenicity, Clin. Microbiol. Rev. 13 (2000), 571–587. https://doi.org/10.1128/cmr.13.4.571.
- K. Kadek, S. Darmadi, Gejala rubela bawaan (kongenital) berdasarkan pemeriksaan serologis dan RNA virus, Indones. J. Clin. Pathol. Med. Lab. 13 (2018), 63–71. https://doi.org/10.24293/ijcpml.v13i2.885.
- [3] World Health Organization, Global measles and rubella strategic plan: 2012. (2012). https://www.who.int/pu blications/i/item/9789241503396.
- [4] J. Lessler, D.A.T. Cummings, Mechanistic models of infectious disease and their impact on public health, Amer. J. Epidemiol. 183 (2016), 415–422. https://doi.org/10.1093/aje/kww021.
- [5] G.B. Grant, S. Desai, L. Dumolard, et al. Progress toward rubella and congenital rubella syndrome control and elimination — worldwide, 2000–2018, MMWR Morb. Mortal. Wkly. Rep. 68 (2019), 855–859. https: //doi.org/10.15585/mmwr.mm6839a5.
- [6] M.Y. Li, An introduction to mathematical modeling of infectious diseases, Springer, Cham, 2018. https: //doi.org/10.1007/978-3-319-72122-4.
- [7] H.M. Yang, A.R.R. Freitas, Biological view of vaccination described by mathematical modellings: from rubella to dengue vaccines, Math. Biosci. Eng. 16 (2019), 3195–3214. https://doi.org/10.3934/mbe.2019159.

- [8] B.P. Prawoto, Abadi, R. Artiono, Dynamic of re-infection Rubella transmission model with vaccination, AIP Conf. Proc. 2264 (2020), 020005. https://doi.org/10.1063/5.0025674.
- [9] D. Amelia, H. Tasman, Mathematics model of measles and rubella with vaccination, J. Phys.: Conf. Ser. 1725 (2021), 012017. https://doi.org/10.1088/1742-6596/1725/1/012017.
- [10] G.T. Tilahun, T.M. Tolasa, G.A. Wole, Modeling the dynamics of rubella disease with vertical transmission, Heliyon. 8 (2022), e11797. https://doi.org/10.1016/j.heliyon.2022.e11797.
- [11] E. Bonyah, C.W. Chukwu, M.L. Juga, Fatmawati, Modeling fractional-order dynamics of Syphilis via Mittag-Leffler law, AIMS Math. 6 (2021), 8367–8389. https://doi.org/10.3934/math.2021485.
- [12] J. Alidousti, M.M. Ghahfarokhi, Dynamical behavior of a fractional three-species food chain model, Nonlinear Dyn. 95 (2018), 1841–1858. https://doi.org/10.1007/s11071-018-4663-6.
- [13] M. Saeedian, M. Khalighi, N. Azimi-Tafreshi, et al. Memory effects on epidemic evolution: The susceptibleinfected-recovered epidemic model, Phys. Rev. E. 95 (2017), 022409. https://doi.org/10.1103/physreve.95. 022409.
- [14] I. Koca, Analysis of rubella disease model with non-local and non-singular fractional derivatives, Int. J. Optim. Control, Theor. Appl. 8 (2017), 17–25. https://doi.org/10.11121/ijocta.01.2018.00532.
- [15] D. Baleanu, H. Mohammadi, S. Rezapour, A mathematical theoretical study of a particular system of Caputo–Fabrizio fractional differential equations for the Rubella disease model, Adv. Differ. Equ. 2020 (2020), 184. https://doi.org/10.1186/s13662-020-02614-z.
- [16] M.M. Al Qurashi, Role of fractal-fractional operators in modeling of rubella epidemic with optimized orders, Open Phys. 18 (2020), 1111–1120. https://doi.org/10.1515/phys-2020-0217.
- [17] V. Capasso, G. Serio, A generalization of the Kermack-McKendrick deterministic epidemic model, Math. Biosci. 42 (1978), 43–61. https://doi.org/10.1016/0025-5564(78)90006-8.
- [18] H.S. Panigoro, A. Suryanto, W.M. Kusumawinahyu, et al. Dynamics of an eco-epidemic predator-prey model involving fractional derivatives with power-law and Mittag-Leffler kernel, Symmetry. 13 (2021), 785. https: //doi.org/10.3390/sym13050785.
- [19] J. Cresson, A. Szafranska, Discrete and continuous fractional persistence problems the positivity property and applications, Commun. Nonlinear Sci. Numer. Simul. 44 (2017), 424–448. https://doi.org/10.1016/j.cn sns.2016.07.016.
- [20] H.L. Li, L. Zhang, C. Hu, et al. Dynamical analysis of a fractional-order predator-prey model incorporating a prey refuge, J. Appl. Math. Comput. 54 (2016), 435–449. https://doi.org/10.1007/s12190-016-1017-8.
- [21] S.K. Choi, B. Kang, N. Koo, Stability for Caputo fractional differential systems, Abstr. Appl. Anal. 2014 (2014), 631419. https://doi.org/10.1155/2014/631419.
- [22] J.M. Heffernan, R.J. Smith, L.M. Wahl, Perspectives on the basic reproductive ratio, J. R. Soc. Interface. 2 (2005), 281–293. https://doi.org/10.1098/rsif.2005.0042.

- [23] E. Ahmed, A.M.A. El-Sayed, H.A.A. El-Saka, On some Routh-Hurwitz conditions for fractional order differential equations and their applications in Lorenz, Rössler, Chua and Chen systems, Phys. Lett. A. 358 (2006), 1–4. https://doi.org/10.1016/j.physleta.2006.04.087.
- [24] D. Matignon, Stability results for fractional differential equations with applications to control processing, Comput. Eng. Syst. Appl. 2 (1996), 963–968.
- [25] R. Shi, Y. Li, C. Wang, Analysis of a fractional-order model for african swine fever with effect of limited medical resources, Fractal Fract. 7 (2023), 430. https://doi.org/10.3390/fractalfract7060430.
- [26] R. Scherer, S.L. Kalla, Y. Tang, et al. The Grünwald–Letnikov method for fractional differential equations, Computers Math. Appl. 62 (2011), 902–917. https://doi.org/10.1016/j.camwa.2011.03.054.