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Effect of Population Density on the Model of the Spread of Measles

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Abstract

This study is expected to contribute to the health sector, specifically to describe the dynamics of the measles spread through the models that have been analyzed. One of the factors that became the focus of this study was reviewing the influence of population density on measles spread. The initial step formulated the model and then determined the primary reproduction number (R_0) and analyzed the stability of the model equilibrium point. The results of the analysis of this model show that there are two conditions for the value of (R_0) which is a requirement that the existence of two model equilibrium points as well as local stability is needed, namely $R_0 < 1$ and $R_0 > 1$. When $R_0 < 1$, there exists a unique equilibrium point, called the non-endemic equilibrium point denoted by E_0. Conversely, when $R_0 > 1$, there are two equilibrium points, namely E_0 and the endemic equilibrium point characterized by E^*. The results of local stability analysis show that when $R_0 < 1$, the equilibrium point E_0 is stable asymptotic locally. It means that if $R_0 < 1$ hold, then in a long time there will not be a spread of disease in the susceptible and vaccinated sub-population, or in other words, the outbreak of the disease will stop. Conversely, when $R_0 > 1$ equilibrium point E^* is stable asymptotic locally. It means that if $R_0 > 1$, then measles disease is still in the environment for an infinite time with the condition of the proportions of each sub-population approach to S^*, V^*, I^* and R^*.

INTRODUCTION

Environmental factors can affect the spread of an illness so that they can affect public health. Dangerous diseases include infectious diseases. One of the problems of health in Indonesia is contagious diseases. Indonesia is a tropical island country because it is located in the equator area. Indonesia has a very high environmental temperature, so that this temperature is ideal for growing a microorganism [1]. These microorganisms are very small-sized viruses that can be viewed using microscope tools [2]. Measles disease is one of the problems in Indonesia. Measles disease is a contagious disease caused by *Paramyxovirus*. Efforts to prevent its spread can be made by giving vaccines to susceptible individuals infected with measles.

In the human body and healthy animal's inserted bacteria or pathogenic viruses that have been weakened is the meaning of vaccination. Vaccinations are expected to form an immune system to combat these pathogenic bacteria or viruses. The problem of disease spread phenomenon is often modeled in the form of mathematical equations, including SIR and SIRS models [3][4]. Individuals who are faced can be assumed as new sub-populations in the SIR model. The addition of the V (Vaccination) sub-population of the SIR epidemic model indicates the number of individuals who have experienced the vaccination process, so one of the development models of the SIR epidemic model is the SVIR epidemic model. The population is divided into four subpopulations, S (Susceptible), V (Vaccination), I (Infected), and R (Recovered) [5]. The number of individuals susceptible to disease is denoted by S (Susceptible), the

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number of individuals who have undergone a vaccination process denoted by V (Vaccination), the number of infected individuals denoted by I (Infected), and the number of individuals recovering from the disease denoted by R (Recovered). Mathematical models about the SVIR epidemic are widely discussed in several scientific articles, including [6],[7], [8][9], [10], [7], [11], [12],[13],[14], [15], [16], [17], [18], [19]and[20]. Models discussed in the article [6],[7], [8][9], [10], [7], [11], [12] and[13] is a continuous model of SIR epidemic with the addition of vaccination compartment. The model is then called the SVIR [13], Later discussed by Marentek in his thesis [14] in 2011. In the year 2014, in the article [15], Haryati applied the SVIR model to analyze the spread of measles disease in Semarang. In his article [17] in 2017, he discusses the SVIR model [13] with the addition of deadly disease assumptions. In the same year, Hidayati discussed the model of SVIR [13] with the addition of an event factor saturated in the population. The SVIR model [13] focuses on a constant number of populations until 2018; Harianto added an unconstant assumption of the model's population and discussed it in the article [18].

Furthermore, Aryani implemented the SVIR Model [13] to analyze diphtheria disease spread in Indonesia [20]. Discussion of this article is a continuation of the paper [17] in 2017 Written by Harianto, et al. The difference of this article with previous reports is the modification of the model in adding the influence of density in the population. This article is expected to contribute to the health sector in particular to describe the dynamic spread of measles disease through models that have been analyzed.

METHODS

This study was conducted with a mathematical approach that refers to several references (literary studies). The stages in this study are as follows:

Step 1 : Article collection and relevant information related to measles disease (literature study),
Step 2 : Determination of assumptions as a reference for the restriction of problems and the process of drafting a measles epidemic model with various facts in a region.
Step 3 : Formulation of problems with mathematical descriptions of models and studies of the model.
Step 4 : Create the numerical simulation and its interpretation.

RESULTS AND DISCUSSION

Populations can be divided into four subpopulations, such as the susceptible subpopulation (S), sub-populations that are vaccinated (V), sub-population infected (I), and sub-population recovered (R). The size used for each of these sub-populations in this model is proportions. The proportion of each sub-population will undoubtedly change or depend on time. In other words, the balance of each sub-population is a function of time variables. Variable time is denoted by t. To solve this problem mathematically, letting S (t) is a proportion of the susceptible subpopulation, V (t) is a proportion of sub-population that is vaccinated, I (t) is a proportion of sub-population infected, and R (t) is the proportion of sub-population recovered at the time t. It is assumed that S (t), V (t), I (t), and R (t) is differentiable of time. This model is formed by adding one parameter as a wide-sized area of the disease of measles populated by the population. These parameters are used to help analyze the population density dependence of the spread of measles disease dynamics. The phenomenon of spreading measles that is modeled need to be limited and defined by the following assumptions:

1. The population is not constant, and the community is not closed,
2. The birth rate is the same for each subpopulation,
3. Individuals who are born in each subpopulation enter into susceptible sub-population,
4. Birth rate equals death rate,

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5. Types of disease is a deadly disease,
6. The disease can be permanently recovered,
7. The incubation period is relatively short,
8. The vaccine is given to someone suspect of measles,
9. Immunity to the disease is obtained over time, resulting in a permanent recovered,
10. A person who has been given a vaccine can be infected with measles before gaining immunity,
11. Contamination between infected people with susceptible people leads to the transmission of measles,
12. The distribution of homogeneous populations across the region is not broad.

From the assumptions outlined above, the following are given explanations of some of the notation used in the model.

1) $\frac{dS}{dt}$ = rate of susceptible sub-population,
2) $\frac{dV}{dt}$ = rate of vaccinated sub-population,
3) $\frac{dI}{dt}$ = rate of infected sub-population,
4) $\frac{dR}{dt}$ = rate of recovered sub-population,
5) S = proportion of susceptible sub-population, with $S(t)$ is positive for all t,
6) V = proportion of vaccinated sub-population, with $V(t)$ is positive for all t,
7) I = proportion of infected sub-population, with $I(t)$ is positive for all t,
8) R = proportion of recovered sub-population, with $R(t)$ is positive for all t,
9) μ = Natural birth rate with μ is positive,
10) β = Transmission rate between infected sub-populations with susceptible sub-population, with β is positive,
11) β_1 = Transmission rate between infected sub-populations with vaccinated sub-population, with β_1 is positive,
12) γ = Recovery rate of infectious measles, with γ is positive,
13) γ_1 = Recovery rate after being given a vaccine, with γ is positive,
14) α = Vaccine rate on susceptible sub-population, with α is positive,
15) ω = Death rate of Infectious measles, with ω is positive,
16) A = area of human-populated, with A is positive.

The following is given a transmission diagram of the SVIR epidemic model of measles disease.

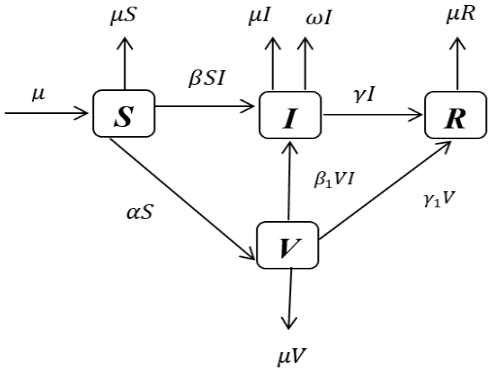

Figure 1. Transfer diagram of the SVIR model for measles disease

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Based on the SVIR transfer diagram with the assumptions given, the following are given SVIR models in the form of a differential equation system.

$$\begin{aligned}\frac{dS}{dt} &= \mu - \mu S - \frac{\beta SI}{A} - \alpha S \\ \frac{dV}{dt} &= \alpha S - \frac{\beta_1 VI}{A} - \gamma_1 V - \mu V \\ \frac{dI}{dt} &= \frac{\beta SI}{A} + \frac{\beta_1 VI}{A} - \gamma I - \mu I - \omega I \\ \frac{dR}{dt} &= \gamma_1 V + \gamma I - \mu R\end{aligned} \qquad (1)$$

with initial conditions $S^0, V^0, I^0, R^0 \geq 0$, for $t = 0$ and $S + V + I + R = 1$ for all non-negative t.

Equilibria and Basic Reproduction Number

Because the last equation does not affect the other equation, then the above system can be eliminated to:

$$\begin{aligned}\frac{dS}{dt} &= \mu - \mu S - \frac{\beta SI}{A} - \alpha S \\ \frac{dV}{dt} &= \alpha S - \frac{\beta_1 VI}{A} - \gamma_1 V - \mu V \\ \frac{dI}{dt} &= \frac{\beta SI}{A} + \frac{\beta_1 VI}{A} - \gamma I - \mu I - \omega I\end{aligned} \qquad (2)$$

Furthermore, the local stability analysis of the equilibria of the SVIR model is analyzed in the neighbourhood of the equilibria. The initial step is to determine the equilibrium point of the system (2). It is mentioned that the dynamics of the SVIR model has two equilibrium points i.e. the non-endemic and endemic equilibrium point [13]. The non-endemic equilibrium point is a representation of a state without being infected by an infectious environment. While the endemic equilibrium point is a representation of some people in the environment still infected with disease. It means that the disease will even spread because there are still several infected people (I is positive) for $t \to \infty$ in an environment.

The equilibria of the system (2) obtained when

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dI}{dt} = 0,$$

Consequently:

$$\mu - \mu S - \frac{\beta SI}{A} - \alpha S = 0 \qquad (3)$$

$$\alpha S - \frac{\beta_1 VI}{A} - \gamma_1 V - \mu V = 0 \qquad (4)$$

$$\frac{\beta SI}{A} + \frac{\beta_1 VI}{A} - n\, I = 0 \qquad (5)$$

According to equation (5), we get $I\left(\frac{\beta S}{A} + \frac{\beta_1 V}{A} - n\right) = 0$, then $I = 0$ or $\frac{\beta S}{A} + \frac{\beta_1 V}{A} - n = 0$, with $n = \gamma + \mu + \omega$. Thus, we have two case, i.e.:

Case 1. $I = 0$,

This case is called the necessary condition to obtain non-endemic equilibrium points. Note that:

From Equation (3) we get $\mu - \mu S - \alpha S = 0 \Leftrightarrow \boldsymbol{S = \frac{\mu}{\alpha + \mu}}$

From Equation (4) we get $\alpha S - \gamma_1 V - \mu V = 0 \Leftrightarrow \boldsymbol{V = \frac{\alpha \mu}{(\alpha + \mu)(\gamma_1 + \mu)}}$

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Thus, the non-endemic equilibrium point is $E_0 = (S_0, V_0, I_0) = \left(\frac{\mu}{\alpha+\mu}, \frac{\alpha\mu}{(\alpha+\mu)(\gamma_1+\mu)}, 0\right)$.

Case 2. $I > 0$,

This case is called a necessary condition to obtain the endemic equilibrium point. We know that

From Equation (3) we get $\mu - \mu S - \frac{\beta SI}{A} - \alpha S = 0 \Leftrightarrow \boldsymbol{S^*} = \frac{A\mu}{(A\alpha+A\mu+\beta I^*)}$

From Equation (4) we get $\alpha S - \frac{\beta_1 VI}{A} - \gamma_1 V - \mu V = 0 \Leftrightarrow \boldsymbol{V^*} = \frac{A^2\alpha\mu}{(A\alpha+A\mu+\beta I^*)(A\gamma_1+A\mu+\beta_1 I^*)}$

According to equation (5), if $I \neq 0$, then we have

$$\frac{\beta S}{A} + \frac{\beta_1 V}{A} - \gamma - \mu - \omega = 0,$$

such that by substituted S^* and V^*, we get:

$$\frac{\mu\beta}{(A\alpha + A\mu + \beta I^*)} + \frac{A\alpha\mu\beta_1}{(A\alpha + A\mu + \beta I^*)(A\gamma_1 + A\mu + \beta_1 I^*)} - \mu + \gamma + \omega = 0$$

$$\Leftrightarrow \mu + \gamma + \omega - \frac{\mu\beta}{(A\alpha + A\mu + \beta I^*)} - \frac{A\alpha\mu\beta_1}{(A\alpha + A\mu + \beta I^*)(A\gamma_1 + A\mu + \beta_1 I^*)} = 0$$

If we let:

$A_1 = (\gamma + \mu + \omega)\beta_1\beta > 0$

$A_2 = A(\gamma + \mu + \omega)((\alpha + \mu)\beta_1 + (\gamma_1 + \mu)\beta) - A\beta_1\beta\mu$

$A_3 = A^2(\gamma + \mu + \omega)(\alpha + \mu)(\gamma_1 + \mu) > 0$

$$K = \frac{\beta\mu}{A(\alpha + \mu)(\mu + \gamma + \omega)} + \frac{\alpha\beta_1\mu}{A(\alpha + \mu)(\mu + \gamma_1)(\mu + \gamma + \omega)}$$

Then we get:

$$A_1 I^{*2} + A_2 I^* + A_3(1 - K) = 0 \qquad (6)$$

Where we have the roots of the equation (6), i. e.

$$I_{1,2}{}^* = \frac{-A_2 \pm \sqrt{A_2{}^2 - 4A_1A_3(1 - K)}}{2A_1}$$

Clearly that $I^* > 0$ such that $K > 1$ must hold.

So, we get the endemic equilibrium i.e. $E^* = (S^*, V^*, I^*)$

With $S^* = \frac{A\mu}{(A\alpha+A\mu+\beta I^*)}$, $V^* = \frac{A^2\alpha\mu}{(A\alpha+A\mu+\beta I^*)(A\gamma_1+A\mu+\beta_1 I^*)}$ and I^* is the positive root of equation (6).

Basic reproduction number R_0 be a condition for the existence of the endemic equilibrium point of the system (2). Basic reproduction number R_0 can be obtained by specifying the positivity condition of the proportion of sub-population infected.

Reconsider equation (6); the endemic equilibrium point is a positive root because of the equibrium point is the proportion of sub-populations that in real life is positive. Positive roots are only hold when K>1. Thus, it was concluded that the existence of the endemic equilibrium point depends on the value of K, therefore, the K parameter can be defined as basic reproduction number, i. e.:

$$R_0 = \frac{\beta\mu}{A(\alpha + \mu)(\mu + \gamma + \omega)} + \frac{\alpha\beta_1\mu}{A(\alpha + \mu)(\mu + \gamma_1)(\mu + \gamma + \omega)}$$

Clearly that the existence of the non-endemic equilibrium points does not depend on R_0. Reviewed from the R_0 can be concluded that if $R_0 \leq 1$, then there exists a unique equilibrium point of system (2), that isthe non-endemic equilibrium point denoted by E_0. However, if $R_0 > 1$, then there are two equilibrium point of system (2), that is E_0 and the endemic equilibrium point denoted by E^*. We obtain that

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$E^* = (S^*, V^*, I^*) = \left(\frac{A\mu}{(\alpha+\mu+\beta I^*)}, \frac{A^2\alpha\mu}{(\alpha+\mu+\beta I)(\mu+\gamma_1+\beta_1 I^*)}, I^*\right)$, with I^* is positive root of the following equation

$$A_1 I^{*2} + A_2 I^* + A_3(1 - R_0) = 0,$$

where

$A_1 = (\gamma + \mu + \omega)\beta_1\beta > 0$

$A_2 = A(\gamma + \mu + \omega)((\alpha + \mu)\beta_1 + (\gamma_1 + \mu)\beta) - A\beta_1\beta\mu$

$A_3 = A^2(\gamma + \mu + \omega)(\alpha + \mu)(\gamma_1 + \mu) > 0$

Stability Analysis

The result of local stability analysis of the equilibrium point of the system (2) is given in the following theorems.

Theorem 1.

Define

$$R_0 = \frac{\beta\mu}{A(\alpha+\mu)(\mu+\gamma+\omega)} + \frac{\alpha\beta_1\mu}{A(\alpha+\mu)(\mu+\gamma_1)(\mu+\gamma+\omega)}$$

1) If the basic reproduction number less than one $(R_0 < 1)$, then the non-endemic equilibrium point E_0 is locally asymptotically stable. However, if the basic reproduction number more than one $(R_0 > 1)$, then the endemic equilibrium point E_0 is not stable.
2) The endemic equilibrium point E^* islocally asymptotically stable, if the basic reproduction number more than one $(R_0 > 1)$.

Proof**.**

1) The following is Jacobian matrix (linearization method) of system (2)

$$J(S,V,I) = \begin{pmatrix} -\mu - \alpha - \frac{\beta I}{A} & 0 & -\frac{\beta S}{A} \\ \alpha & -\mu - \gamma_1 - \frac{\beta_1 I}{A} & -\frac{\beta_1 V}{A} \\ \frac{\beta I}{A} & \frac{\beta_1 I}{A} & \frac{\beta S}{A} + \frac{\beta_1 V}{A} - \mu - \gamma - \omega \end{pmatrix}$$

Hence Jacobian matrix in $\boldsymbol{E_0} = (\boldsymbol{S_0}, \boldsymbol{V_0}, \boldsymbol{I_0}) = \left(\frac{\boldsymbol{\mu}}{\boldsymbol{\alpha+\mu}}, \frac{\boldsymbol{\alpha\mu}}{(\boldsymbol{\alpha+\mu})(\boldsymbol{\gamma_1+\mu})}, \mathbf{0}\right)$ is:

$$J(E_0) = \begin{pmatrix} -\mu - \alpha & 0 & -\frac{\beta S_0}{A} \\ \alpha & -\mu - \gamma_1 & -\frac{\beta_1 V_0}{A} \\ 0 & 0 & \frac{\beta S_0}{A} + \frac{\beta_1 V_0}{A} - \mu - \gamma - \omega \end{pmatrix}$$

The characteristic equation of $J(E_0)$ can be written as

$$[-(\mu + \alpha) - \lambda][-(\mu + \gamma_1) - \lambda]\left[\frac{\beta S_0}{A} + \frac{\beta_1 V_0}{A} - n - \lambda\right] = 0$$

where:

$$\lambda_1 = -\mu - \alpha < 0$$
$$\lambda_2 = -\mu - \gamma_1 < 0$$
$$\lambda_3 = \frac{\beta S_0}{A} + \frac{\beta_1 V_0}{A} - n = n(R_0 - 1)$$

Clearly that all Eigen value of $J(E_0)$ are negative when $R_0 < 1$. Consequently, E_0 is locally asymptotically stable[21]. However, when $R_0 > 1$ there exists positive Eigen value of $J(E_0)$ hence E_0 is not stable.

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1) Obviously, when $R_0 > 1$, we obtained that E^*, hence Jacobian matrix around $E^* = (S^*, V^*, I^*)$ can be written as:

$$J(E^*) = \begin{pmatrix} -\mu - \alpha - \frac{\beta I^*}{A} & 0 & -\frac{\beta S^*}{A} \\ \alpha & -\mu - \gamma_1 - \frac{\beta_1 I^*}{A} & -\frac{\beta_1 V^*}{A} \\ \frac{\beta I^*}{A} & \frac{\beta_1 I^*}{A} & \frac{\beta S^*}{A} + \frac{\beta_1 V^*}{A} - \mu - \gamma - \omega \end{pmatrix}$$

The element j_{11} of $J(E^*)$ ekuivalent with $-\mu - \alpha - \frac{\beta I^*}{A} = -\frac{\mu}{S^*}$ and the element entri j_{22} of $J(E^*)$ ekuivalent with $-\mu - \gamma_1 - \frac{\beta_1 I^*}{A} = -\frac{\alpha S^*}{V^*}$. We know that $\frac{\beta S^*}{A} + \frac{\beta_1 V^*}{A} - \mu - \gamma - \omega = 0$, hence $J(E^*)$ can be written as:

$$J(E^*) = \begin{pmatrix} -\frac{\mu}{S^*} & 0 & -\frac{\beta S^*}{A} \\ \alpha & -\frac{\alpha S^*}{V^*} & -\frac{\beta_1 V^*}{A} \\ \frac{\beta I^*}{A} & \frac{\beta_1 I^*}{A} & 0 \end{pmatrix}$$

The characteristic equation of $J(E^*)$, that is

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$$

where:

$$a_1 = \frac{\mu}{S^*} + \frac{\alpha S^*}{V^*} > 0$$

$$a_2 = \frac{\alpha\mu}{V^*} + \frac{{\beta_1}^2 V^* I^*}{A^2} + \frac{\beta^2 S^* I^*}{A^2} > 0$$

$$\boldsymbol{a_3 = \frac{\alpha\beta_1\beta S^* I^*}{A^2} + \frac{\alpha\beta^2 {S^*}^2 I^*}{V^* A^2} + \frac{\mu{\beta_1}^2 V^* I^*}{S^* A^2}} > 0$$

We have

$$a_1 a_2 - a_3 = \frac{\alpha\mu^2}{S^* V^*} + \frac{(\mu + \beta I^*)\beta^2 S^* I^*}{A^2} + \frac{\alpha^2 \mu S^*}{{V^*}^2} + \frac{\alpha I^* S^* (\beta - \beta_1)^2}{A^2} + \frac{\alpha\beta_1\beta S^* I^*}{A^2} > 0$$

Hence according to Routh-Hurwitz criterion [22], $a_i > 0,\ i = 1,2,3$ dan $a_1 a_2 - a_3 > 0$ are hold, then all the Eigen values of $J(E^*)$ have negative real parts. So, $E^* = (S^*, V^*, I^*)$ is locally asymptotically stable [21]. Therefore, the proof is complete.

According to the results that have been obtained, the interpretation is as follows:

1. If $R_0 < 1$ and the initial condition (S^0, V^0, I^0) at the neighborhood of $E_0 = (S_0, V_0, I_0)$, then for $t \to \infty$, the solution from the system (1) will move towards the $E_0 = (S_0, V_0, I_0)$. This means that if $R_0 < 1$, then for the number of susceptible, vaccinated, and infected individuals approaching $E_0 = (S_0, V_0, I_0)$, Then the disease does not spread, and the number of infected sub-populations decreases towards zero for an indefinite period. It means that measles disease tends to disappear in disease. It is called stable asymptotic local around the equilibrium point $E_0 = (S_0, V_0, I_0)$.
2. If $R_0 > 1$ and the initial condition (S^0, V^0, I^0) at the neighborhood of $E^* = (S^*, V^*, I^*)$, then for $t \to \infty$, the solution from the system (1) will move towards the $E^* = (S^*, V^*, I^*)$. This means that if $R_0 > 1$, then Measles disease still remains in the environment for an infinite time with the value S, V, I approaching $E^* = (S^*, V^*, I^*)$. It is called stable asymptotic local around the equilibrium point $E^* = (S^*, V^*, I^*)$.

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Interpretation of analysis results is similar to the discussion in [13], [14], [15], [16], [17], [18], [19] and [20] that the primary reproduction number plays an important role to know the dynamics of the spread of disease. However, every primary reproduction number obtained varies depending on the parameters of the model formed. In this discussion, the population density factor influences the primary reproduction number. It can be seen from the presence of the population density parameter that appears on the primary reproduction number.

Numerical Results

The following solutions are provided from the system (1) with a numerical approach. The approach is numerically given in the form of graphs of sub-population susceptible, vaccinated, infected, and recovered versus time. All parameter values used are estimates. The stability of the equilibrium point of the system (1) is noted based on the R_0 value, which is $R_0 > 1$ and $R_0 < 1$. In this simulation, three broad-size areas are used to review population density influence each subpopulation's proportion. The assumed area size is 20 km^2, 200 km^2, and 2000 km^2.

Assumed parameter values for the case $R_0 < 1$, with $\mu = 0{,}03;\ \beta = \beta_1 = 5;\ \gamma = 0{,}1;\ \gamma_1 = 0{,}1;\ \alpha = 0{,}2;\ \omega = 0{,}05$. The solution of the System (1) with the given parameters presented in Figure 2. For a total area of 20 km^2, within 0 to 5 months the proportion of the infected sub-population increased above 0.1 then after 5 months decreased to 0. These results are shown in Figure 2 below.

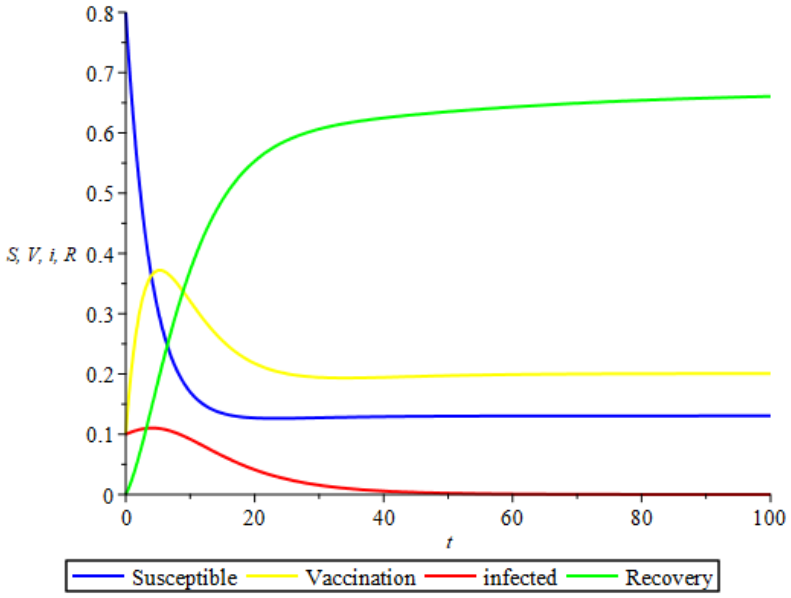

Figure 2. Dynamics of the proportion of sub-population S, V, I and R versus time (t) for a total area of 20 km^2

The graph in Figure 3 shows a total area of 200 km^2, the proportion of sub-populations infected from the initial state of 0.1 decreased to 0. The chart also applies to an area of 2000 km^2; the infected population has decreased to 0. The three-area comparison of the region shows that the area is an influential factor in the dynamic spread of measles disease. The more dense the area then the proportion of the infected subpopulation will increase to a specific time limit. Further, the proportion of sub-population infected decreases until the spread will stop for an infinite time.

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While in the area with an area above 200 km^2, the proportion of the infected sub-population will continue to decline, even the spread will cease for an infinite time.

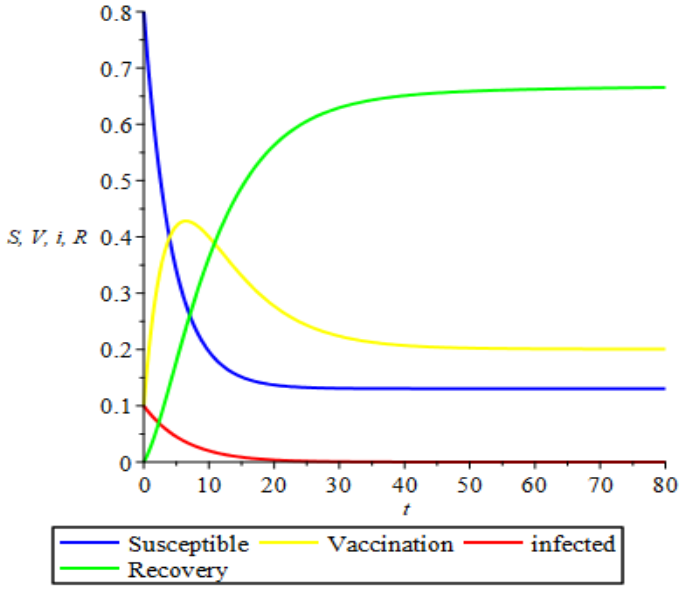

Figure 3.Dynamics of the proportion of sub-population S, V, I and R versus time (t) for a total area of 200 km^2 dan 2000 km^2

CONCLUSIONS

The analysis results of this model indicate that there are two case R_0 value, which is $R_0 > 1$ and $R_0 < 1$. Each case of the R_0 value determines the local stability analysis of the equilibrium point of the model. Furthermore, the R_0 parameter is a necessary condition for the existence of equilibrium point and stability analysis of the model. When $R_0 < 1$, there only a unique equilibrium point called the non-endemic equilibrium point denoted by E_0. However, when $R_0 > 1$ there are two equilibrium point, namely E_0 and the endemic equilibrium point denoted by E^*.The results of local stability analysis show that when $R_0 < 1$, then the non-endemic equilibrium point E_0 is locally asymptotically stable. This means that if $R_0 < 1$ hold, then measles disease does not spread, and the number of infected subpopulations decreases towards zero. This means that the spread of measles disease will stop. However, E^* is locally asymptotically stable when $R_0 > 1$. It This means that if $R_0 > 1$, then measles disease is still in the environment for an infinite time. The proportion of each sub-population in this situation is S^*, V^*, I^* dan R^*.The area inhabited by the population is an influential factor in the dynamic spread of measles disease. The more dense the residential areas of the population then the proportion of infected sub populations will increase to a certain time limit. Furthermore, the proportion of sub-population is infected down until the spread will stop for infinite time. In the area above 200 km^2, the proportion of the infected sub-population will continue to decline even the spread will stop for an infinite time.

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