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SEPIR Model of Skin Cancer Caused by Exposure to Ultraviolet Light

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Abstract

Skin cancer is a disease that occurs due to a change in the nature of normal skin cells into abnormal skin cells, where these cells will divide into abnormal forms in an unconditioned manner due to DNA damage. This research explains the stability of the SEPIR model in skin cancer caused by exposure to ultraviolet light. The population is divided into five subpopulations, namely, susceptible (S), latent period with early symptoms (E), pre-cancer (P), infected (I), and recovered from skin cancer (R). Based on the model analysis using Jacobian matrix and eigen value, there is one equilibrium point free and one endemic equilibrium point for skin cancer and the basic reproduction number $\boldsymbol{R_0}$*. The results of the stability test of the equilibrium test using basic reproduction number* $\boldsymbol{R_0}$ *showed that if* $\boldsymbol{R_0 < 1}$*, then the equilibrium point free from skin cancer is asymptotically stable and if* $\boldsymbol{R_0 > 1}$ *the equilibrium point endemic to skin cancer is asymptotically stable.*

Keywords: *Basic Reproduction Number, Eigen Value, Jacobian Matrix, SEPIR Model.*

1. INTRODUCTION

One of the diseases that causes death worldwide is cancer. According to the 2018 Basic Health Research of the Ministry of Health of the Republic of Indonesia, the level of people affected by cancer in Indonesia itself has also increased quite high, which in 2013 increased by 1.4 thousand people then in 2018 rose to 1.79 thousand of people [1]**.** One of the cancer diseases that attack the community is skin cancer.

Skin Cancer is a genetic term for a large group of diseases that are characterized by abnormal cell development that crosses the boundary and quickly spreads to other organs. In other words, skin cancer is a disease that arises due to abnormal cell development in the body and over time these cells will turn into cancer cells in the body. Therefore, it can be concluded that it is true that skin cancer is a disease that causes major deaths worldwide [2]. Skin cancer is one of the cancers that has the highest malignancy rate. Skin cancer even occupies the third position according to the results of Rikesdas (2007) as the hottest cancer after cervical cancer and breast cancer found in Indonesia [3]**.** Some researchers who have conducted studies have found that skin cancer becomes very aggressive to people with white skin. This is still predicted because of their frequent exposure to sunlight, but it does not rule out the possibility that people with brown skin or other skin colors are at risk of developing this type of cancer. This skin cancer is also very fatal for sufferers in addition to causing death if not followed up because Indonesia has a fairly high level of sun exposure throughout the year because it is located along the equator [4]**.**

The mathematical model is made based on several assumptions which will then be analyzed and the model will be represented to the problem being discussed [5]**.** There are several forms of mathematical models, one of which is the SEIR form that has been studied by several researchers. Then, in this problem, the SEIR mathematical model represents skin cancer, especially due to exposure to ultraviolet light. As in previous research conducted by Side et al. [6] produce a mathematical model of the SEIR form of skin cancer due to ultraviolet light exposure in South Sulawesi by obtaining the stability value of the basic reproductive number $(R_0) < 1$ which concluded that a person infected with skin cancer cannot cause other people to get the same disease in the province.

In [6] it states that symptoms that belong to group E (Exposed) or individuals, who have symptoms but are not yet infected, have not been explained in detail. Therefore the authors want to develop the work in [6] in which this development there are additional variables and parameters. In the addition of variables, there is the addition of pre-cancer variables (P) and

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the addition of parameters, namely $\propto (alfa)$ defined the rate of latent individuals becoming pre-cancerous individuals. In this pre-cancerous individual, there are second-level symptoms of the latent period (*exposure*), which in this pre-cancerous period can also be called the condition of solar keratosis. Solar keratosis will cause rough and scaly patches of varying colors on the face and hands due to sun exposure. This research is corroborated by the authors in [3] which explains and describes in detail about skin cancer and research from the work in [5] which discusses SEIR modeling in stages. Furthermore, in [1] it also explains the addition of the Pre (P) variable to the model discussed.

2. RESEARCH METHODS

This research is a literature study. Definition of skin from [7] states that skin is very important in the human body and plays an important role in social society and mathematical models can solve real problems that exist in society. The assumptions made to solve real problems in mathematics can produce several models such as the SIR model [8] and SEIR model [9]**.** After the model is obtained, it will then be analyzed to find the stability of the resulting model using the Jacobian matrix and eigenvalues and basic reproduction numbers. The following theorem is related to the stability of the model.

Theorem 1.

a. If all eigenvalues of the Jacobian matrix $J(f(x))$ have negative real parts, then the equilibrium point is asymptotically stable.
b. If any eigenvalue of the Jacobian matrix $J(f(x))$ has a positive real part, then the equilibrium point is unstable [10].

Next, a discussion related to basic reproductive numbers is given. Basic reproduction number (R_0) is the average number of infected individuals which is caused by one infected individual during its infection period in the entire vulnerable population. There are several provisions in this basic reproduction number, namely, if $R_0 < 1$ it can be interpreted that the disease will only infect less than one vulnerable individual in a population so that the disease will disappear from the population. Then, if $R_0 > 1$, it means that infected individuals will be able to infect more than vulnerable individuals. Meanwhile, if $R_0 = 1$ then the infected individual will infect or transmit to at least one other individual [9].

Theorem 2

a. If $R_0 < 1$ then the disease-free equilibrium point is asymptotically stable in the sense that the disease will disappear.
b. If $R_0 > 1$ then the equilibrium point of the endemic disease is asymptotically stable in the sense that the disease will outbreak [11].

The steps in this research are:

(i) Determining model assumptions and defining model parameters viz:
   a. Assuming a closed population, the total population size is constant. N.
   b. The diseases discussed are only skin diseases caused by ultraviolet exposure.
   c. It is assumed that the birth rate and death rate are the same.
   d. The incubation period (latency period) of skin cancer is called the skin cancer formation period, which starts from individuals who have symptoms but are not infected (E) then becomes individuals who have entered pre-cancer symptoms (P).
   e. There is no death from skin cancer. This means that skin cancer is curable through treatment.

(ii) Reconsider the model in Side et al. [6] which will be developed by adding Pre-Cancer (P) variables with parameters α.

(iii) Analysis of the resulting model and model stability.

3. RESULTS AND DISCUSSION

In this study, the SEPIR model in skin cancer is divided into five classes, namely, Susceptible Class (S), Exposed Class (E), Pre-Cancer Class (P), Infected Class (I) and Recovered Class (R). Based on the

assumptions that have been made, the SEPIR model of skin cancer due to ultraviolet light exposure is obtained with a transfer diagram model as shown in Figure 1.

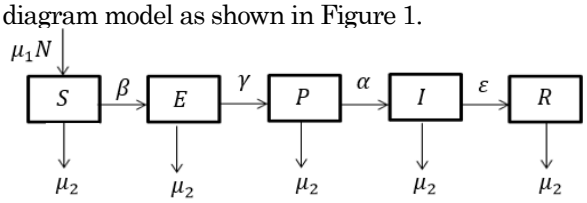

Figure 1. SEPIR mathematical model of skin cancer due to ultraviolet light exposure

The variables and parameters used in this model can be seen in Table 1.

Table 1. Variables and Model Parameters

Variables/ parameters	Definition/description
S	Number of individuals susceptible to skin cancer
E	Number of individuals experiencing early symptoms but not yet infected
P	Number of individuals who have symptoms of solar keratosis that result in skin cancer
I	Number of individuals infected with skin cancer
R	Number of individuals cured of skin cancer
μ_1	Birth rate
μ_2	Natural death rate
β	The rate at which susceptible individuals become individuals who have early symptoms but are not yet infected with skin cancer
α	The rate of individuals who have early symptoms but are not yet infected with skin cancer to pre-cancerous individuals (having symptoms of solar keratosis that result in skin cancer)
γ	Rate of individuals infected with skin cancer
ε	The cure rate of each individual infected with skin cancer
N	Total population

Based on the assumptions, transfer diagram of the model and Table 1, the differential equation system of the SEPIR model of skin cancer due to ultraviolet light exposure is obtained, namely

$$\begin{aligned} \frac{dS}{dt} &= \mu_1 N - \mu_2 S - \beta S \\ \frac{dE}{dt} &= \beta S - \mu_2 E - \alpha E \\ \frac{dP}{dt} &= \alpha E - \mu_2 P - \gamma P \\ \frac{dI}{dt} &= \gamma P - \mu_2 I - \varepsilon I \\ \frac{dR}{dt} &= \varepsilon I - \mu_2 R \end{aligned} \tag{1}$$

where $N(t) = S(t) + E(t) + P(t) + I(t) + R(t)$ is the total population.

Then, Equation (1) can be simplified using scaling by simplifying the notation, suppose $s := \frac{S}{N}, S = sN$; $e = \frac{E}{N}$, $E = eN$; $p = \frac{P}{N}$, $P = pN$; $i = \frac{I}{N}$, $I = iN$; $r = \frac{R}{N}$, $R = rN$. By using scaling, the system of differential equations (1), can be simplified into a system of differential Equation (2) as follows:

$$\begin{aligned} \frac{ds}{dt} &= \mu_1 - \mu_2 s - \beta s \\ \frac{de}{dt} &= \beta s - \mu_2 e - \alpha e \\ \frac{dp}{dt} &= \alpha e - \mu_2 p - \gamma p \\ \frac{di}{dt} &= \gamma p - \mu_2 i - \varepsilon i \\ \frac{dr}{dt} &= \varepsilon i - \mu_2 r \end{aligned} \tag{2}$$

Furthermore, the sum of system (2) is obtained $N(t) = \mu_1/\mu_2$. Thus, if t increases then

$$\lim_{t\to\infty} N(t) = \frac{\mu_1}{\mu_2}$$

or $N(t) \to \mu_1/\mu_2$ as $t \to \infty$, meaning that the human population will reach the capacity limit μ_1/μ_2. If $N_0 > \mu_1/\mu_2$ then $N(t)$ monotonically decreases towards the capacity limit μ_1/μ_2 and if $N_0 < \mu_1/\mu_2$ then $N(t)$ monotonically increasing towards the capacity limit μ_1/μ_2. So it can be explained that the human population N(t) over a long period of time will go to the capacity limit μ_1/μ_2. Because $N(t) \to \mu_1/\mu_2 \; as \; t \to \infty$, then the feasible region

$$\Omega = \left\{(S,V,I,R) : S,V,I,R \geq 0 \; ; S + V + I + R = \frac{\mu_1}{\mu_2}\right\} \tag{3}$$

is the positive invariant of Model (1). Therefore, the model region is within Ω .

3.1. Model Equilibrium Point

The equilibrium points in Equation (2) are obtained by equating Equation (2) to zero, namely, $ds/dt = 0, de/dt = 0, dp/dt = 0, di/dt = 0,$ and $dr/dt = 0$. From Equation (2), there are two equilibrium points, namely the disease-free equilibrium point and the endemic equilibrium point. The disease-free equilibrium point occurs if there are no diseases or disease symptoms in the population, namely $e = p = i = 0$.

Based on Equation (2), the disease-free equilibrium point of the SEPIR model is obtained, namely $E_0 = (s,e,p,i,r) = \left(\frac{\mu_1}{\mu_2}, 0,0,0,0\right)$. While the endemic equilibrium point occurs if there are symptoms and diseases in the population, $e = p = i \neq 0$. Based on Equation (2), the endemic equilibrium point of the disease is obtained, namely $E_\varepsilon = (s^*, e^*, p^*, i^*, r^*) = \left(\frac{\mu_1}{(\mu_2+\beta)}, \frac{\beta s}{(\mu_2+\alpha)}, \frac{\alpha e}{(\mu_2+\gamma)}, \frac{\gamma p}{(\mu_2+\varepsilon)}, \frac{\varepsilon i}{\mu_2}\right)$.

3.2. Basic Reproduction Number

The basic reproduction number is obtained by determining the eigenvalues of the Jacobian matrix of the system of equations (model). From Equation (2), the Jacobian matrix is obtained as follows:

$$J(f(x)) = \begin{bmatrix} -\mu_2 - \beta & 0 & 0 & 0 & 0 \\ \beta & -\mu_2 - \alpha & 0 & 0 & 0 \\ 0 & \alpha & -\mu_2 - \gamma & 0 & 0 \\ 0 & 0 & \gamma & -\mu_2 - \varepsilon & 0 \\ 0 & 0 & 0 & \varepsilon & -\mu_2 \end{bmatrix}$$

Furthermore, it yields

$$\begin{aligned} (-\mu_2 - \beta)(-\mu_2 - \alpha)(-\mu_2 - \gamma)(-\mu_2 - \varepsilon)(-\mu_2) &= 0 \\ ({\mu_2}^2 + \mu_2\alpha + \mu_2\beta + \beta\alpha)(-\mu_2 - \gamma) &= 0 \\ (-{\mu_2}^3 - {\mu_2}^2\gamma - {\mu_2}^2\alpha - \mu_2\alpha\gamma - {\mu_2}^2\beta - \mu_2\beta\gamma - \beta\alpha\mu_2 - \beta\alpha\gamma)(-\mu_2 - \varepsilon)(-\mu_2) &= 0 \\ {\mu_2}^5 + {\mu_2}^4\varepsilon + {\mu_2}^4\gamma + {\mu_2}^3\gamma\varepsilon + {\mu_2}^4\alpha + {\mu_2}^3\alpha\varepsilon + {\mu_2}^3\alpha\gamma + {\mu_2}^2\alpha\gamma\varepsilon + {\mu_2}^4\beta + {\mu_2}^3\beta\varepsilon + {\mu_2}^3\beta\gamma + {\mu_2}^2\beta\gamma\varepsilon \\ + {\mu_2}^3\beta\alpha + {\mu_2}^2\beta\alpha\varepsilon + {\mu_2}^2\beta\alpha\gamma + \mu_2\beta\alpha\gamma\varepsilon &= 0 \end{aligned}$$

So, the basic reproduction number R_0 is obtained as follows:

$$R_0 = \mu_2\beta\alpha\gamma\varepsilon + (\beta\alpha\gamma + \beta\alpha\varepsilon + \beta\gamma\varepsilon + \alpha\gamma\varepsilon){\mu_2}^2 + (\beta\alpha + \beta\gamma + \beta\varepsilon + \alpha\gamma + \alpha\varepsilon + \gamma\varepsilon){\mu_2}^3 + (\beta + \alpha + \gamma + \varepsilon){\mu_2}^4 + {\mu_2}^5 \quad (4)$$

3.3. Equilibrium Point Stability of the Model

To determine the stability of the equilibrium point of the model, here the author uses numerical simulation to see the stability of the model. This simulation is carried out with maple18 software, the data will be given in Table 2.

Table 2. Preliminary Data

Variables	Value	Source
N	8819500	Journal [6]
S	8818990	Journal [6]
E	98	Assumption
P	95	Assumption
I	188	Journal [6]
R	129	Journal [6]

Source: Side et al. (2021)

The parameters that will be used in this model can be seen in Table 3.

Table 3. Parameters of SEPIR Model on Skin Cancer Disease

Variables	Parameter Value 1	Parameter Value 2	Parameter Value 3	Parameter Value 4
μ_1	0,083	0,083	0,083	0,083
μ_2	0,083	0,083	0,083	0,083
β	0,000022	0,000022	0,000022	0,000022
α	0,98	0,98	0,98	0,98
γ	0,97	0,97	0,97	0,97
ε	0,1	0,5	0,9	0,7

Source: Side et al. (2021)

Then, the parameter values can be substituted into

$$\frac{ds}{dt} = 0{,}083 - 0{,}083s - 0{,}000022s$$

$$\frac{de}{dt} = 0{,}000022s - 0{,}083e - 0{,}98e$$

$$\frac{dp}{dt} = 0{,}98e - 0{,}083p - 0{,}97p$$

$$\frac{di}{dt} = 0{,}97p - 0{,}083i - \varepsilon i$$

$$\frac{dr}{dt} = \varepsilon i - 0{,}083r$$

Then, skin cancer has a basic reproduction number value (R_0) which is:

$$R_0 = \mu_2\beta\alpha\gamma\varepsilon + (\beta\alpha\gamma + \beta\alpha\varepsilon + \beta\gamma\varepsilon + \alpha\gamma\varepsilon){\mu_2}^2 + (\beta\alpha + \beta\gamma + \beta\varepsilon + \alpha\gamma + \alpha\varepsilon + \gamma\varepsilon){\mu_2}^3 + (\beta + \alpha + \gamma + \varepsilon){\mu_2}^4 + {\mu_2}^5$$

Then, the parameter value can be determined by substituting the value of each variable in each parameter in Table 3 to R_0 obtained as follows,

Parameter 1	$R_0 = 0{,}001314230881$
Parameter 2	$R_0 = 0{,}004496778275443$
Parameter 3	$R_0 = 0{,}007582046389073$
Parameter 4	$R_0 = 0{,}006039412332243$

Because these parameters have $R_0 < 1$, then according to Theorem 2 it can be interpreted that a person infected with skin cancer will not cause other people to get the same disease, meaning that there will be no outbreak in the population. Then, because the basic reproduction

value $R_0 < 1$ then this shows that cancer in the region has not increased and there is no outbreak.

Furthermore, to see the endemic state of skin cancer, the parameter values will be selected $\alpha, \gamma > 1$ and other parameter values are the same as in Table 3, where each parameter value is assumed in Table 4.

Table 4. SEPIR Model Parameters in the Endemic State of Skin Cancer Disease

Variables	Parameter 1	Parameter 2	Parameter 3	Parameter 4
μ_1	0,083	0,083	0,083	0,083
μ_2	0,083	0,083	0,083	0,083
β	0.000022	0.000022	0.000022	0.000022
α	500	500	500	500
γ	0.5	0.5	0.7	0.9
ε	0,1	0,5	0,9	0,7

In the same way in finding the value R_0 in the disease-free state, the value of R_0 for the endemic disease for Table 4 as follows.

Parameter 1 $R_0 = 1{,}08$
Parameter 2 $R_0 = 4{,}02$
Parameter 3 $R_0 = 5{,}48$
Parameter 4 $R_0 = 6{,}95$

Known value $R_0 > 1$ for the parameters $\alpha > 1$ dan < 1 , then according to Theorem 2 it can be interpreted that the endemic skin cancer disease has occurred in the population.

3.4. Numerical Simulation of Model

3.4.1. Numerical Simulation of Models for $R_0 < 1$

With respect to the parameter values ε parameter, where ε This parameter states the recovery rate of each infected individual due to treatment. Then the simulation will be carried out as follows.

Simulation for Susceptible Population

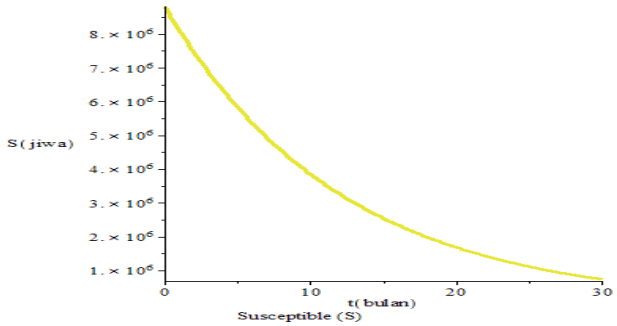

Figure 2 Simulation for Susceptible Population with $\varepsilon = 0.1, 0.5, 0.9$ and 0.7

From Figure 2, it can be explained that the population or the number of individuals susceptible to skin cancer with a cure rate of $\varepsilon = 0.1, 0.5, 0.9$ and 0.7 decreased from its starting point of 8818990 people in month 0 and disappeared in month-30. Then, the susceptible population is not affected by the cure rate parameter ε.

Simulation for Exposed Population

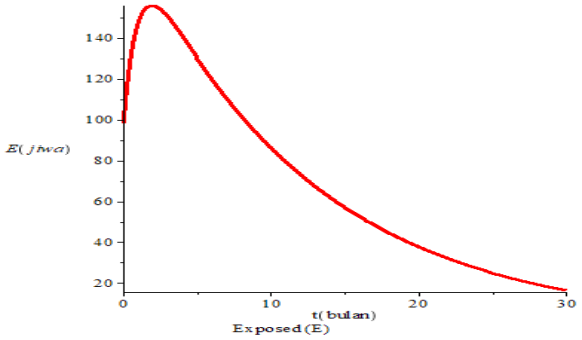

Figure 3 Simulation for Exposed Population With $\varepsilon = 0.1, 0.5, 0.9$ and 0.7

In Figure 3, it can be seen that the population of individuals who show early symptoms of skin cancer due to exposure to ultraviolet light (exposed) has increased from the 0th month, namely 98 people, then experienced a very drastic decline in the 30th month. Then, in this population the cure rate ε does not affect.

Simulation for Pre-Cancer Population

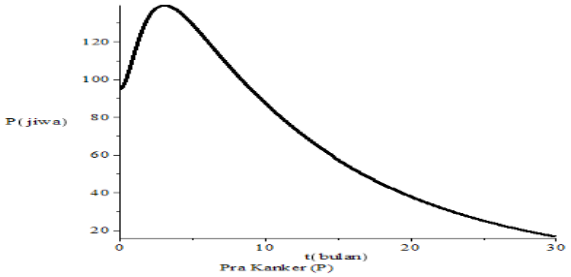

Figure 4 Simulation for Pre-Cancer Population With $\boldsymbol{\varepsilon = 0.1, 0.5, 0.9}$ **and** $\boldsymbol{0.7}$

It can be seen in Figure 4 that the population of individuals who have experienced severe symptoms towards pre-cancer called Pre-cancer (P) in skin cancer has increased from its starting point of 95 people and then decreased in the 30th month. Then, the pre-cancer population is not influenced by the cure rate parameter ε.

Simulation for Infected Population

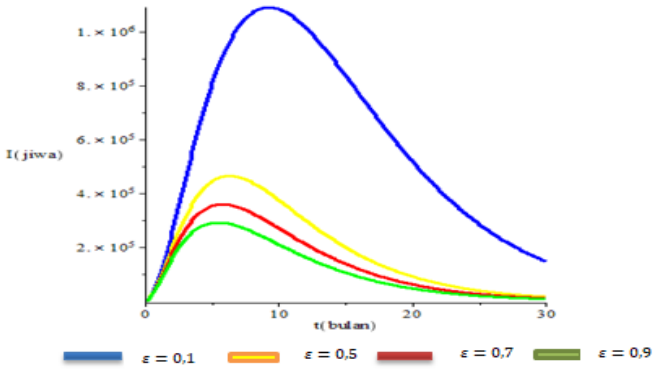

Figure 5 Simulation for Infected Population

It can be seen in Figure 5 that the total population of infected individuals has increased from month 0 with a starting point of 188 people by looking at each recovery rate. ε which is the greater ε the larger it is, the smaller the infected population.

Simulation for Recovered Population

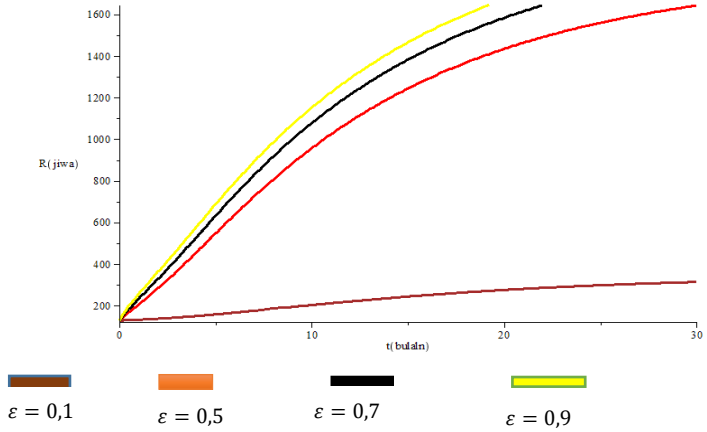

Figure 6. Simulation for Recovered Population

It can be seen from Figure 6 that the population of individuals who have recovered from skin cancer due to ultraviolet light exposure has increased drastically from month 0 to month 30, namely with an initial point of 129 people with each recovery rate ε. So the greater the value of the recovery rate ε, then the more the population is cured in the region.

3.4.1. Numerical Simulation of Model for $R_0 > 1$

Numerical simulations of the model for $R_0 < 1$ which means the endemic equilibrium point is asymptotically stable.

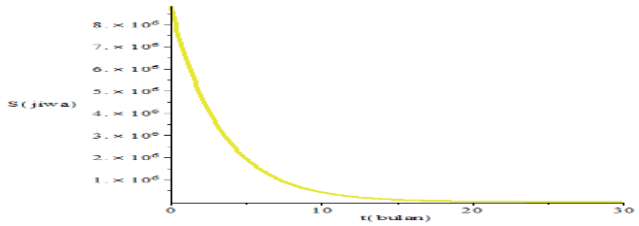

Figure 7. Simulation for Susceptible Population with $\boldsymbol{\varepsilon = 0.1, 0.5, 0.7, 0.9}$

It can be seen in Figure 7 that the vulnerable population S from each recovery rate increases from month 0 to month 30 the population decreases. In this population, the cure rate ε does not affect the spread of skin cancer in the population.

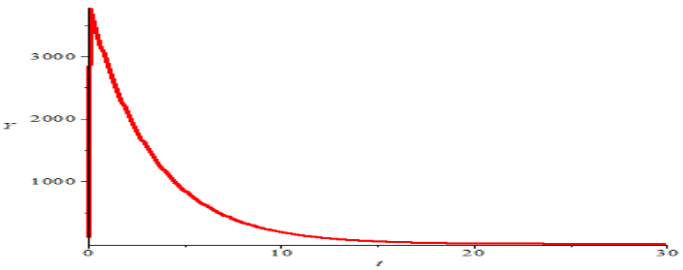

Figure 8. Simulation for Exposed Population with $\boldsymbol{\varepsilon = 0.1, 0.5, 0.7, 0.9}$

It can be seen in Figure 8 that every month the population that has the initial symptoms of E will continue to exist and grow every month and will disappear in the 30th month. In this population, the cure rate $\boldsymbol{\varepsilon}$ does not affect the spread of skin cancer in that population.

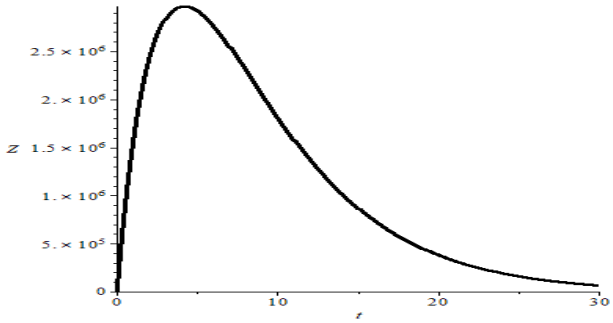

Figure 9. Simulation for Pre-Cancer Population With $\boldsymbol{\varepsilon = 0.1, 0, 5, 0.7, 0.9}$

It can be seen from Figure 9 that skin cancer outbreaks in the Pre-Cancer population with a high increase and then decreases in the 30th month. In this population, the cure rate ε does not affect the spread of skin cancer in the population.

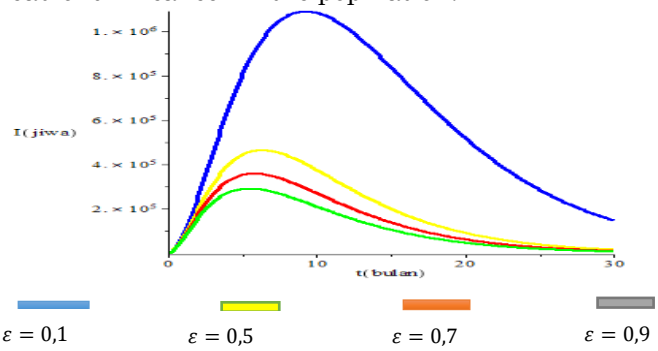

Figure 10. Simulation for Infected Population

In Figure 10, it can be seen that skin cancer continues to develop or outbreak in the population infected with the disease with each cure rate and disappears in the 30th month.

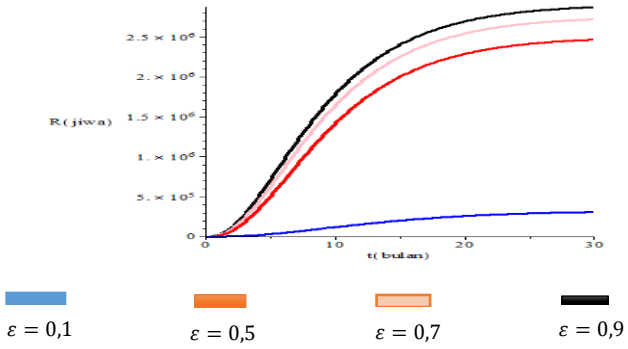

Figure 11. Simulation for Recovered Population

In Figure 11, it can be seen that the population cured of skin cancer continues to increase from month 0 to month 30, where the greater the value of the cure rate, the greater the cure rate. ε then the cure rate of the disease is getting bigger.

CONCLUSION

Based on the research that has been done, it can be concluded as follows that the SEPIR model of skin cancer due to ultraviolet light exposure has two equilibrium points, namely the disease-free equilibrium point and the disease-endemic equilibrium point E_0 and disease endemic equilibrium point E_ε. Then for stability at the disease-free equilibrium point E_0 is asymptotically stable and the disease endemic equilibrium point is E_ε is also asymptotically stable based on its basic reproduction number (R_0).

The value of R_0 in each parameter in the disease-free state E_0 is asymptotically stable if $R_0 < 1$ while for the disease endemic equilibrium point E_ε is asymptotically stable if $R_0 > 1$, it can be stated that the greater the cure rate ε in each infected individual, the recovered class will increase due to the treatment carried out and vice versa in the infected class will decrease.

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