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Batik Motif "Sulur Harapan": Data Visualization from Mathematical Model Simulation of Skin Cancer in South Sulawesi Province

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Abstract: As part of Indonesia's cultural heritage, batik continues to evolve through the integration of art and research. This study developed the "Sulur Harapan" batik motif based on data visualization obtained from a SEIR mathematical model simulation of skin cancer spread in South Sulawesi Province. To identify the dynamic patterns of disease spread and trends in the number of individuals in each population class, epidemiological data related to skin cancer cases affected by ultraviolet exposure were analyzed using mathematical modeling methods. The simulation results show that variations in the recovery rate play an important role in suppressing the number of infected people, and an increase in the recovery rate significantly accelerates the development of the disease. Philosophically, the dynamic patterns generated from this simulation are translated into vine motifs, which are symbols of growth, balance, and hope. This motif not only serves as an aesthetic work but also functions as a visual educational tool to inform people about the risks and ways to prevent skin cancer. Therefore, the "Sulur Harapan" batik serves as a way to integrate culture, science, and public health using a mathematical modeling approach.

Keywords: Batik Sulur Harapan, Data Visualization, Mathematical Model, Skin Cancer, South Sulawesi.

Introduction

Skin cancer is one of the public health problems that continues to show an increase globally. The latest projections show that the incidence of melanoma could increase significantly by 2040 if there are no more effective preventive interventions, with an estimated number of new cases reaching more than half a million per year (Arnold et al., 2022). The overall burden of skin and subcutaneous tissue diseases is also increasing in various regions of the world, including tropical countries such as Indonesia, which has high levels of ultraviolet exposure throughout the year (Yakupu et al., 2023). This situation underscores the need for analytical and educational approaches that can describe the patterns of skin cancer risk in a more comprehensive and easily understandable manner for the public.

Mathematical modeling has long been the foundation for understanding the dynamics of disease spread in populations. The classic book by Anderson RM & May RM (1991) emphasizes that mathematical models play an important role in describing the basic mechanisms of disease spread, predicting epidemiological patterns, and assessing the effectiveness of intervention programs. With the development of modern technology, mathematical models are now often combined with advanced computational methods. One important study by (Esteva A et al., 2017) shows that deep learning algorithms are capable of classifying skin cancer with accuracy equivalent to that of medical specialists. This finding shows that the integration of epidemiological theory and computation can provide stronger visual representations and risk predictions. In addition, a global study by (Karimkhani et al., 2017) highlights the importance

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of data mapping to understand the geographical distribution of melanoma.

At the same time, data visualization has become an important element in modern health communication. A study by (Schulze et al., 2023) emphasizes that visual dashboards and interactive graphics can improve public understanding of health issues when designed to be clear and informative. In the context of skin cancer education, data-driven visualization makes it possible to convey complex epidemiological information in a way that is easier for the general public to understand.

Based on this background, this study aims to develop a mathematical model simulation related to skin cancer dynamics and present the results through easy-to-understand data visualization. This approach is expected to help provide a clearer picture of skin cancer risk and support more effective public education. By integrating classical epidemiological theory and modern data visualization, this study seeks to bridge the gap between technical information and the public's need for simpler yet accurate understanding.

Research Method

Model

This study uses a mathematical model developed to calculate the spread of skin cancer (PAPER). This model consists of a system of differential equations that describe the interaction between the $Susceptible\ (S)$ population, which represents the vulnerable class, the $Exposed\ (E)$ population, which represents the symptomatic class, the $Infected\ (I)$ population, which represents the infected class, and the $Recovered\ (R)$ population, which represents the recovered class. Then, to form the model, several assumptions are required, as follows :

1. The population is closed (no immigration or emigration)
2. There is one disease spreading in the population
3. The birth rate and natural death rate are considered equal
4. There is an incubation period in the formation of skin cancer
5. Individuals in the latent phase (E) do not transmit the disease
6. Infected individuals (I) can be cured through treatment

Based on assumptions and relationships between parameters, the following differential system is obtained:

$$\begin{aligned}\frac{dS}{dt} &= \mu_1 N - \mu_2 S - \beta S \\ \frac{dE}{dt} &= \beta S - \mu_2 E - \gamma E \\ \frac{dI}{dt} &= \gamma E - \mu_2 I - \varepsilon I \\ \frac{dR}{dt} &= \varepsilon I - \mu_2 R \end{aligned} \quad (1)$$

Where $N(t) = S(t) + E(t) + I(t) + R(t)$ is the total population at time t.

Equation (1) can be simplified using scaling, by forming equation (1) into a proportion between the population in a class and the total population. To simplify, let us assume that:

$$s = \frac{S}{N}, e = \frac{E}{N}, i = \frac{I}{N}, r = \frac{R}{N}$$

So that equation (1) can be simplified to :

$$\begin{aligned}\frac{ds}{dt} &= \mu_1 - \mu_2 s - \beta s \\ \frac{de}{dt} &= \beta s - \mu_2 e - \gamma e \\ \frac{di}{dt} &= \gamma e - \mu_2 i - \varepsilon i \\ \frac{dr}{dt} &= \varepsilon i - \mu_2 r \end{aligned} \quad (2)$$

With $s(t) + e(t) + i(t) + r(t) = 1$

Explanation:

N : Total Population

S : Number of individuals susceptible to skin cancer

E : Number of individuals experiencing symptoms but not yet infected

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 : Rate of individuals becoming latent due to exposure to ultraviolet rays
γ : Rate of individuals infected with skin cancer
ε : Rate of recovery for each infected individual due to treatment

Equilibrium point

The equilibrium point is obtained by setting the time derivative equal to zero, namely:

$$\frac{ds}{dt} = 0, \frac{de}{dt} = 0, \frac{di}{dt} = 0, \frac{dr}{dt} = 0$$

In general, there are two equilibrium points: disease-free and disease-infected.

a. Cancer-free

Assuming that $i = 0$ and $e = 0$, which means that no individuals are infected or undergoing treatment. Then we obtain:

Equation (1)

$$\frac{ds}{dt} = \mu_1 - \mu_2 s - \beta s = 0$$

$$\frac{ds}{dt} = \mu_1 - s(\mu_2 + \beta)$$

$$s = \frac{\mu_1}{\mu_2 + \beta}$$

Equation (4)

$$\frac{dr}{dt} = \varepsilon i - \mu_2 r = 0$$

$$r = \frac{0}{-\mu_2}$$

$$r = 0$$

So the disease-free equilibrium point is:

$$E_0 = (s_0, e_0, i_0, r_0) = \left(\frac{\mu_1}{\mu_2 + \beta}, 0,0,0\right)$$

his shows that if the disease disappears completely, then no one will be exposed or sick (the value is 0), and the population will only consist of a small number of susceptible and healthy people.

b. Cancer Infection

he endemic equilibrium point only exists if the disease is present in the population $i > 0$ in other words, the condition for endemic existence. If $s_1, i_1, t_1, r_1 \neq 0$, then we obtain:

Equation (1)

$$\frac{ds}{dt} = \mu_1 - \mu_2 s - \beta s = 0$$

$$\frac{ds}{dt} = \mu_1 - s(\mu_2 + \beta)$$

$$s_1 = \frac{\mu_1}{\mu_2 + \beta}$$

Equation (2)

$$\frac{de}{dt} = \beta s - (\mu_2 + \gamma)e = 0$$
$$e_1 = \frac{\beta s}{\mu_2 + \gamma}$$

From equation (1) , we obtain

$$s_1 = \frac{\mu_1}{\mu_2 + \beta}$$

Subtitute into e_1, then

$$e_1 = \frac{\beta\left(\frac{\mu_1}{(\mu_2+\beta)}\right)}{\mu_2 + \gamma}$$

$$e_1 = \frac{\frac{\mu_1\beta}{\mu_2+\beta}}{\mu_2 + \gamma}$$

$$e_1 = \frac{\mu_1\beta}{\mu_2 + \beta} \cdot \frac{1}{\mu_2 + \gamma}$$

$$e_1 = \frac{\mu_1\beta}{\mu_2 + \beta} \cdot \frac{1}{\mu_2 + \gamma}$$

$$e_1 = \frac{\beta\mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)}$$

Equation (3)

$$\frac{di}{dt} = \gamma e - \mu_2 i - \epsilon i = 0$$

$$-\mu_2 i - \varepsilon = -i(\mu_2 + \varepsilon)$$

$$\gamma e - i(\mu_2 + \varepsilon) = 0$$

$$\gamma e = i(\mu_2 + \varepsilon)$$

$$i_1 = \frac{\gamma e}{\mu_2 + \varepsilon}$$

From Equation (2), we obtain

$$e_1 = \frac{\beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)}$$

Subtitute into e, then

$$i_1 = \frac{\gamma \left(\frac{\beta \mu_1}{(\mu_2+\beta)(\mu_2+\gamma)}\right)}{\mu_2 + \varepsilon}$$

$$i_1 = \frac{\frac{\gamma \beta \mu_1}{(\mu_2+\beta)(\mu_2+\gamma)}}{\mu_2 + \varepsilon}$$

$$i_1 = \frac{\gamma \beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)} \cdot \frac{1}{\mu_2 + \varepsilon}$$

$$i_1 = \frac{\gamma \beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)(\mu_2 + \varepsilon)}$$

Equation (4)

$$\frac{dr}{dt} = \varepsilon\, i - \mu_2 r = 0$$

$$r_1 = \frac{\varepsilon i}{\mu_2}$$

From equation (3), we obtain

$$i_1 = \frac{\gamma \beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)(\mu_2 + \epsilon)}$$

Subtitute into r, then

$$r_1 = \frac{\varepsilon \left(\frac{\gamma \beta \mu_1}{(\mu_2+\beta)(\mu_2+\gamma)(\mu_2+\varepsilon)}\right)}{\mu_2}$$

$$r_1 = \frac{\frac{\varepsilon \gamma \beta \mu_1}{(\mu_2+\beta)(\mu_2+\gamma)(\mu_2+\varepsilon)}}{\mu_2}$$

$$r_1 = \frac{\varepsilon \gamma \beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)(\mu_2 + \varepsilon)} \cdot \frac{1}{\mu_2}$$

$$r_1 = \frac{\varepsilon \gamma \beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)(\mu_2 + \varepsilon)\mu_2}$$

So the equilibrium point for cancer infection is:

$$E_1 = (s_1, e_1, i_1, r_1)$$

$$E_1 = \left(\frac{\mu_1}{\mu_2 + \beta}, \frac{\beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)}, \frac{\gamma \beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)(\mu_2 + \varepsilon)}, \frac{\varepsilon \gamma \beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)(\mu_2 + \varepsilon)\mu_2}\right)$$

In situations like this, the disease will remain. There is no zero point in the balance between the number of vulnerable, symptomatic, infected, and recovered individuals.

Existence

a. Cancer-Free

It is known that the cancer-free equilibrium point is obtained

$$E_0 = (s_0, e_0, i_0, r_0) = \left(\frac{\mu_1}{\mu_2 + \beta}, 0,0,0\right)$$

Since $\mu_1 > 0$ and $\mu_2 + \beta > 0$, the value of s_0 will always be positive ($s_0 > 0$). Thus, the cancer-free equilibrium point E_0 will always exist in the population.

b. Contract Cancer

It is known that the equilibrium point of cancer infection is obtained

$$E_1 = \left(\frac{\mu_1}{\mu_2 + \beta}, \frac{\beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)}, \frac{\gamma \beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)(\mu_2 + \varepsilon)}, \frac{\varepsilon \gamma \beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)(\mu_2 + \varepsilon)\mu_2}\right)$$

Since $\mu_1 > 0, \mu_2 > 0, \gamma > 0$, we obtain

$$i_1 = \frac{\gamma \beta \mu_1}{(\mu_2 + \beta)(\mu_2 + \gamma)(\mu_2 + \varepsilon)}$$

I tis said to exist i fit satisfies the condition $\beta > 0$. If it satisfies this condition, then the equilibrium point E_1 will exist.

Equilibrium point stability

To determine the stability of the disease-free equilibrium point, the Jacobian matrix of the differential equation system is formed as follows:

$$f_1 = \frac{ds}{dt} = \mu_1 - \mu_2 s - \beta s$$

$$f_2 = \frac{de}{dt} = \beta s - \mu_2 e - \gamma e$$

$$f_3 = \frac{di}{dt} = \gamma e - \mu_2 i - \varepsilon i$$

$$f_4 = \frac{dr}{dt} = \varepsilon i - \mu_2 r$$

Then the Jacobian Matrix is as follows:

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial s} & \frac{\partial f_1}{\partial e} & \frac{\partial f_1}{\partial i} & \frac{\partial f_1}{\partial r} \\ \frac{\partial f_2}{\partial s} & \frac{\partial f_2}{\partial e} & \frac{\partial f_2}{\partial i} & \frac{\partial f_2}{\partial r} \\ \frac{\partial f_3}{\partial s} & \frac{\partial f_3}{\partial e} & \frac{\partial f_3}{\partial i} & \frac{\partial f_3}{\partial r} \\ \frac{\partial f_4}{\partial s} & \frac{\partial f_4}{\partial e} & \frac{\partial f_4}{\partial i} & \frac{\partial f_4}{\partial r} \end{bmatrix}$$

This yields the equation from the partial derivative calculation results.

Equation (1)

$$\frac{\partial f_1}{\partial s} = -\mu_2 - \beta$$

$$\frac{\partial f_1}{\partial e} = 0$$

$$\frac{\partial f_1}{\partial i} = 0$$

$$\frac{\partial f_1}{\partial r} = 0$$

Equation (2)

$$\frac{\partial f_2}{\partial s} = \beta$$

$$\frac{\partial f_2}{\partial e} = -\mu_2 - \gamma$$

$$\frac{\partial f_2}{\partial i} = 0$$

$$\frac{\partial f_2}{\partial r} = 0$$

Equation (3)

$$\frac{\partial f_3}{\partial s} = 0$$

$$\frac{\partial f_3}{\partial e} = \gamma$$

$$\frac{\partial f_3}{\partial i} = -\mu_2 - \varepsilon$$

$$\frac{\partial f_3}{\partial r} = 0$$

Equation (4)

$$\frac{\partial f_4}{\partial s} = 0$$

$$\frac{\partial f_4}{\partial e} = 0$$

$$\frac{\partial f_4}{\partial i} = \varepsilon$$

$$\frac{\partial f_4}{\partial r} = -\mu_2$$

a. Cancer-free

$$J\left(\frac{\mu_1}{\mu_2 + \beta}, 0,0,0\right) = \begin{bmatrix} -\mu_2 - \beta & 0 & 0 & 0 \\ \beta & -\mu_2 - \gamma & 0 & 0 \\ 0 & \gamma & -\mu_2 - \varepsilon & 0 \\ 0 & 0 & \varepsilon & -\mu_2 \end{bmatrix}$$

To determine the stability value E_0, the eigenvalues of the matrix will be sought with

$$J\left(\frac{\mu_1}{\mu_2 + \beta}, 0,0,0\right) - \lambda I = 0$$

Where λ is the eigenvalue and I is the identity matrix.

$$\begin{bmatrix} -\mu_2 - \beta & 0 & 0 & 0 \\ \beta & -\mu_2 - \gamma & 0 & 0 \\ 0 & \gamma & -\mu_2 - \varepsilon & 0 \\ 0 & 0 & \varepsilon & -\mu_2 \end{bmatrix} - \lambda \begin{bmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{bmatrix} = 0$$

$$\begin{bmatrix} (-\mu_2 - \beta) - \lambda & 0 & 0 & 0 \\ \beta & (-\mu_2 - \gamma) - \lambda & 0 & 0 \\ 0 & \gamma & (-\mu_2 - \varepsilon) - \lambda & 0 \\ 0 & 0 & \varepsilon & (-\mu_2) - \lambda \end{bmatrix} = 0$$

Then calculate the determinant of the matrix, which is equal to 0.

$$det\begin{bmatrix}(-\mu_2-\beta)-\lambda & 0 & 0 & 0\\ \beta & (-\mu_2-\gamma)-\lambda & 0 & 0\\ 0 & \gamma & (-\mu_2-\varepsilon)-\lambda & 0\\ 0 & 0 & \varepsilon & (-\mu_2)-\lambda\end{bmatrix}=0$$

I Remember that when finding the determinant of an upper or lower triangular matrix, the result is the same as the product of its diagonal elements.

$$(-\mu_2-\beta-\lambda)\cdot(-\mu_2-\gamma-\lambda)\cdot(-\mu_2-\varepsilon-\lambda)\cdot(-\mu_2-\lambda)=0$$

The eigenvalues $\lambda_1,\lambda_2,\lambda_3,\lambda_4$ are obtained with $\lambda_1=-\mu_2-\beta,\lambda_2=-\mu_2-\gamma,\lambda_3=-\mu_2-\varepsilon,\lambda_4=-\mu_2$. Since $\mu_2,\beta,\gamma,\varepsilon$ are positive values and the eigenvalues $\lambda_1,\lambda_2,\lambda_3,\lambda_4$ are negative values, the cancer-free equilibrium point E_0 is stable.

b. Cancer Infection

Next, determine the type of endemic equilibrium stability in the same way as determining the type of disease-free equilibrium stability.

$$J(s_1,e_1,i_1,r_1)=\begin{bmatrix}-\mu_2-\beta & 0 & 0 & 0\\ \beta & -\mu_2-\gamma & 0 & 0\\ 0 & \gamma & -\mu_2-\varepsilon & 0\\ 0 & 0 & \varepsilon & -\mu_2\end{bmatrix}=0$$

It can be seen that the Jacobian matrix is constant. All elements in it only contain parameters $(\beta,\gamma,\mu_2,\varepsilon)$ without any variabels (s,e,i,r). As a result, the Jacobian matrix that describes E_1 is equivalent to the Jacobian matrix that describes E_0.

Since the matrices are equivalent, the stability values and eigenvalues produced will also be the same as the results of the cancer-free calculation E_0.

$$\det(J(s_1,e_1,i_1,r_1)-\lambda I)=0$$

$$det\begin{bmatrix}(-\mu_2-\beta)-\lambda & 0 & 0 & 0\\ \beta & (-\mu_2-\gamma)-\lambda & 0 & 0\\ 0 & \gamma & (-\mu_2-\varepsilon)-\lambda & 0\\ 0 & 0 & \varepsilon & (-\mu_2)-\lambda\end{bmatrix}=0$$

$$(-\mu_2-\beta-\lambda)\cdot(-\mu_2-\gamma-\lambda)\cdot(-\mu_2-\varepsilon-\lambda)\cdot(-\mu_2-\lambda)=0$$

The eigenvalues $\lambda_1,\lambda_2,\lambda_3,\lambda_4$ are obtained with $\lambda_1=-\mu_2-\beta,\lambda_2=-\mu_2-\gamma,\lambda_3=-\mu_2-\varepsilon,\lambda_4=-\mu_2$. Since $\mu_2,\beta,\gamma,\varepsilon,$ are positive values and the eigenvalues $\lambda_1,\lambda_2,\lambda_3,\lambda_4$ are negative values, the equilibrium point E_1 affected by cancer is stable.

Parameter and Data Estimation

This study used data related to skin cancer cases in South Sulawesi Province from 2018 to 2019 without considering the gender or age of the patients. The data included the number of individuals who were susceptible, symptomatic, infected, and recovered from skin cancer in accordance with the SEIR mathematical model (**Table**).

Table 1. Sample data on skin cancer cases in South Sulawesi Province

Symbol	Value	Source
N	8819500	South Sulawesi Central Statistics Agency
S	8818990	Wahidin Sudirohusodo Hospital
E	193	Wahidin Sudirohusodo Hospital
I	188	Wahidin Sudirohusodo Hospital
R	129	Wahidin Sudirohusodo Hospital

The parameters obtained and used in this study along with their values (**Table**).

Table 2. SEIR Model Parameters in Skin Cancer.

Symbol	Parameter Value 1	Parameter Value 2	Parameter Value 3
μ_1	0.083	0.083	0.083
μ_2	0.083	0.083	0.083
β	0.000022	0.000022	0.000022
γ	0.97	0.97	0.97
ε	0.1	0.5	0.9

The steps to be taken in this study are:

1. Steps to create an SEIR model for skin cancer in South Sulawesi province
   a. Collect information about skin cancer in South Sulawesi province.
   b. Determining assumptions and defining parameters used in the SEIR model.
   c. Formulating the SEIR model for skin cancer based on the constraints, assumptions, and parameters that have been created..
2. Steps for analyzing the SEIR model for skin cancer in South Sulawesi province
   a. Determining the equilibrium point of the model based on the equations that have been obtained.
   b. Determine the existence based on the equilibrium point.

c. Analyze the stability of the equilibrium point..

3. Steps for simulating the SEIR model for skin cancer in South Sulawesi province using Matlab 2007 software
   a. Determine the initial values obtained from data on skin cancer patients in South Sulawesi province.
   b. Input the model into Matlab 2007 software.
   c. Input the parameter values used in the model.
   d. Interpret the simulation results.
   a. Draw conclusions from the SEIR model simulation results.

Result And Discussion

Equilibrium Point Analysis

a. Cancer-free

Given a cancer-free equilibrium point

$$E_0 = (s_0, e_0, i_0, r_0) = \left(\frac{\mu_1}{\mu_2 + \beta}, 0,0,0\right)$$

with

$$\mu_1 = 0{,}83, \mu_2 = 0{,}083, \beta = 0{,}000022$$

result

$$s_0 = \frac{0.83}{0.083 + 0.000022} \approx 9.99735$$

So the disease-free equilibrium point is

$$E_0 = (9.99735, e_0, i_0, r_0)$$

This shows that in a disease-free state, almost the entire population is in the susceptible class, while the number of symptomatic, infected, and recovered individuals is zero. This condition describes an ideal situation where the disease does not circulate within the population.

b. Cancer Infection

Given the equilibrium point of cancer infection

$$E_1 = \left(\frac{\beta\mu_1}{(\mu_2+\beta)(\mu_2+\gamma)}, \frac{\gamma\beta\mu_1}{(\mu_2+\beta)(\mu_2+\gamma)(\mu_2+\varepsilon)}, \frac{\varepsilon\gamma\beta\mu_1}{(\mu_2+\beta)(\mu_2+\gamma)(\mu_2+\varepsilon)\mu_2}\right)$$

with

$$i_1 = \frac{\gamma\beta\mu_1}{(\mu_2+\beta)(\mu_2+\gamma)(\mu_2+\varepsilon)}$$

for $\varepsilon = 0.1$

$$E_1 = \frac{0.97 \cdot 0.000022 \cdot 0.83}{(0.83 + 0.000022)(0.83 + 0{,}97)(0.83 + 0.1)} \approx 1.27476 \times 10^{-5}$$

for $\varepsilon = 0.5$

$$E_1 = \frac{0.97 \cdot 0.000022 \cdot 0.83}{(0.83 + 0.000022)(0.83 + 0{,}97)(0.83 + 0.5)} \approx 8.91372 \times 10^{-6}$$

for $\varepsilon = 0.9$

$$E_1 = \frac{0.97 \cdot 0.000022 \cdot 0.83}{(0.83 + 0.000022)(0.83 + 0{,}97)(0.83 + 0.9)} \approx 6.85274 \times 10^{-6}$$

The calculation results show that the value of E_1 decreases as the value of ε increases. This illustrates that the higher the recovery rate (ε), the lower the number of individuals in the infected state (i).

Equilibrium Point Existence Analysis

Using the parameter values (**Tabel**), the following can be obtained

a. Cancer-free

$$\mu_1 = 0.083$$

$$\mu_2 + \beta = 0.083 + 0.000022 = 0.083022$$

Since $s_0 > 0$, there will always be cancer-free equilibrium points in the population.

b. Cancer Infection

The condition for the existence of E_1 is satisfied because the value of $\beta = 0.000022 > 0$. This shows numerically that cancer infection can biologically occur in the modeled population.

Based on the analysis results, it can be concluded that both modeled equilibrium points exist. Since the parameters μ_1 and $\mu_2 + \beta$ have

positive values, the existence of E_0 is fulfilled. In addition, the main condition, $\beta > 0$ is also fulfilled, as validated by the parameter data used, namely $\beta = 0.000022$.

Equilibrium Point Stability Analysis

Stability analysis is performed using the eigenvalues of the Jacobian Matrix at E_0 and E_1. Since both Jacobian Matrices have constant properties, the following eigenvalues are obtained through calculation:

$$\lambda_1 = -\mu_2 - \beta = -0.083 - 0.000022 = -0.083022$$

$$\lambda_2 = -\mu_2 - \gamma = -0.083 - 0.97 = -1.053$$

$$\lambda_4 = -\mu_2 = -0.083$$

at λ_3 because it depends on different values of ε , we obtain

for $\varepsilon = 0.1$

$$\lambda_3 = -\mu_2 - \varepsilon = -0.083 - 0.1 = -0.183$$

for $\varepsilon = 0.5$

$$\lambda_3 = -\mu_2 - \varepsilon = -0.083 - 0.5 = -0.583$$

for $\varepsilon = 0.9$

$$\lambda_3 = -\mu_2 - \varepsilon = -0.083 - 0.9 = -0.983$$

Additionally, stability analysis indicates that both equilibrium points are asymptotically stable. This conclusion is based on numerical calculations of the constant eigenvalues of the Jacobian matrix. For all parameter scenarios, the resulting eigenvalues are negative.

These results indicate that the model predicts that skin cancer will not disappear from the population. At the cancer-infected point (E_1), the system will converge towards a stable condition compared to the disease-free point (E_0) as long as exposure to ultraviolet rays (β) remains.

Matlab Simulation

In this section, the SEIR model is simulated using three variations of the parameter value ε, namely low ε ($\varepsilon = 0.1$), medium ε ($\varepsilon = 0.5$), and high ε ($\varepsilon = 0.9$).

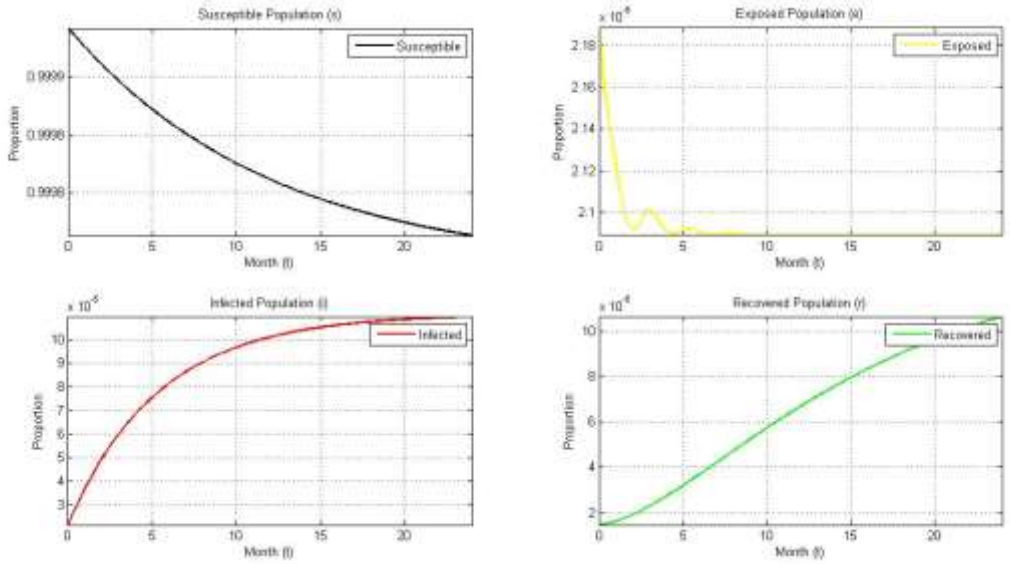

Figure 1. Parameter simulation 1

In (Figure 1), which illustrates population dynamics with a low ε value, it can be seen that the infected curve (I) ises at the beginning of the period and remains fairly high before slowly declining. The high number of infected individuals in this scenario is due to the low recovery rate,

meaning that only a few individuals recover. The recovery curve (R) also increases very slowly, indicating that treatment is not yet effective enough in reducing the number of cases. Meanwhile, the susceptible (S) and exposed (E) curves experience relatively small changes but still show an upward trend in exposure due to the high number of infections.

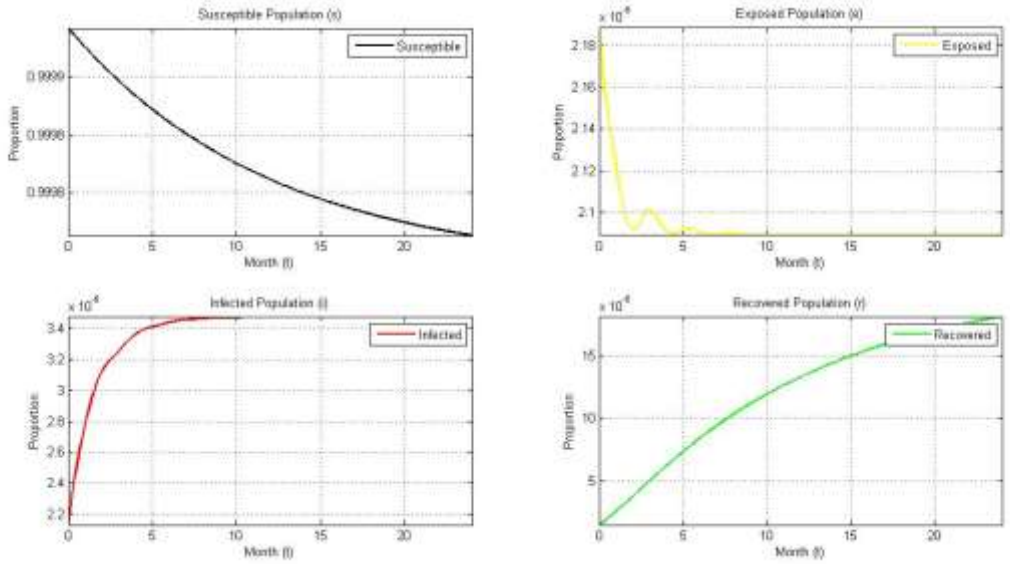

Figure 2. Parameter Simulation 2

In (Figure 2), which is a simulation with a moderate ε value, the infected curve (S) begins to show a faster decline than in (Figure 1). This indicates that an increase in the ε value makes treatment more effective, thereby increasing the number of individuals who recover and suppressing the growth of new cases. The recovery curve (R) appears to increase more significantly than in the first scenario, while the exposed curve (E) experiences more controlled fluctuations. Thus, the rate of disease spread begins to be suppressed even though infection (I) has not completely disappeared from the population.

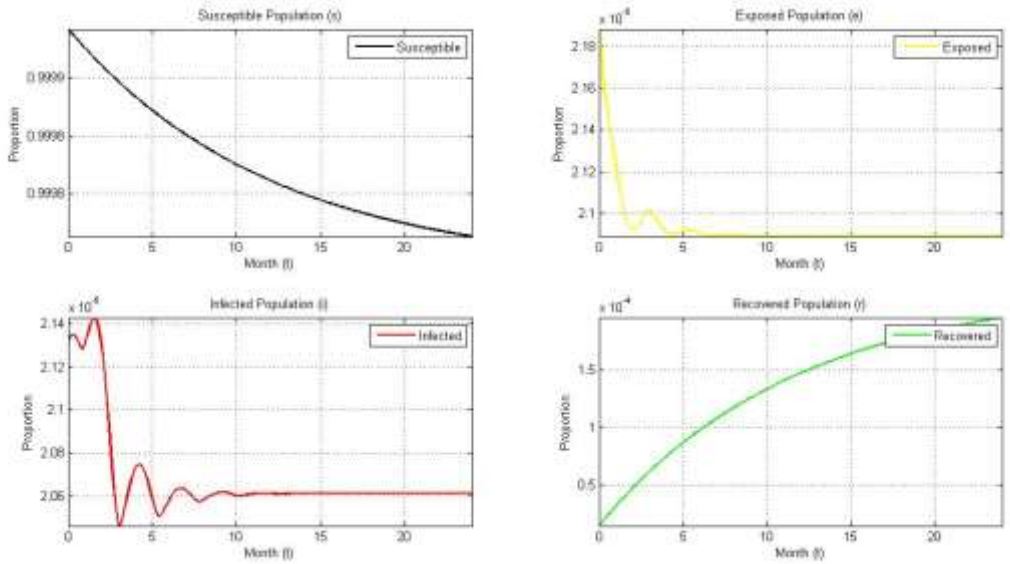

Figure 3. Parameter Simulation 3

Next, in (Figure 3), which is a simulation with a high ε value, it can be seen that the infected curve (I) decreases very rapidly to near zero in a shorter period of time. The recovery curve (R) increases sharply, indicating that most infected individuals recover within a short period of time. The exposed (E) and susceptible (S) curves tend to be stable, indicating that the disease is no longer able to maintain its transmission. Overall, the three graphs show that the higher the ε value, the faster the disease is suppressed and the greater the proportion of individuals who recover.

Based on the three simulation graphs, it can be seen that the parameter (ε) has a dominant influence on the dynamics of disease spread. In (Figure 1 (low ε)), the number of infected individuals remains high because the recovery rate cannot keep up with the infection rate. This condition makes the disease difficult to control and cases persist for a long time. This is different from (Figure 2 (medium ε)), where an increase in the value of ε begins to have a real impact on the decline in cases. The number of infected individuals decreases more quickly and the number of recovered individuals increases significantly, indicating that the treatment has been quite effective even though it has not been able to completely eliminate the infection.

In (Figure 3 (high ε)), the system moves towards a disease-free condition. This is shown by the infected curve approaching zero and the recovered curve continuing to increase steadily. In this scenario, the disease is no longer able to maintain its chain of transmission, so the number of cases decreases dramatically. Thus, the simulation results reinforce that increasing the effectiveness of treatment, represented by the value of ε, is a key factor in controlling the spread of disease and pushing the system towards a stable, disease-free state.

Batik Motifs

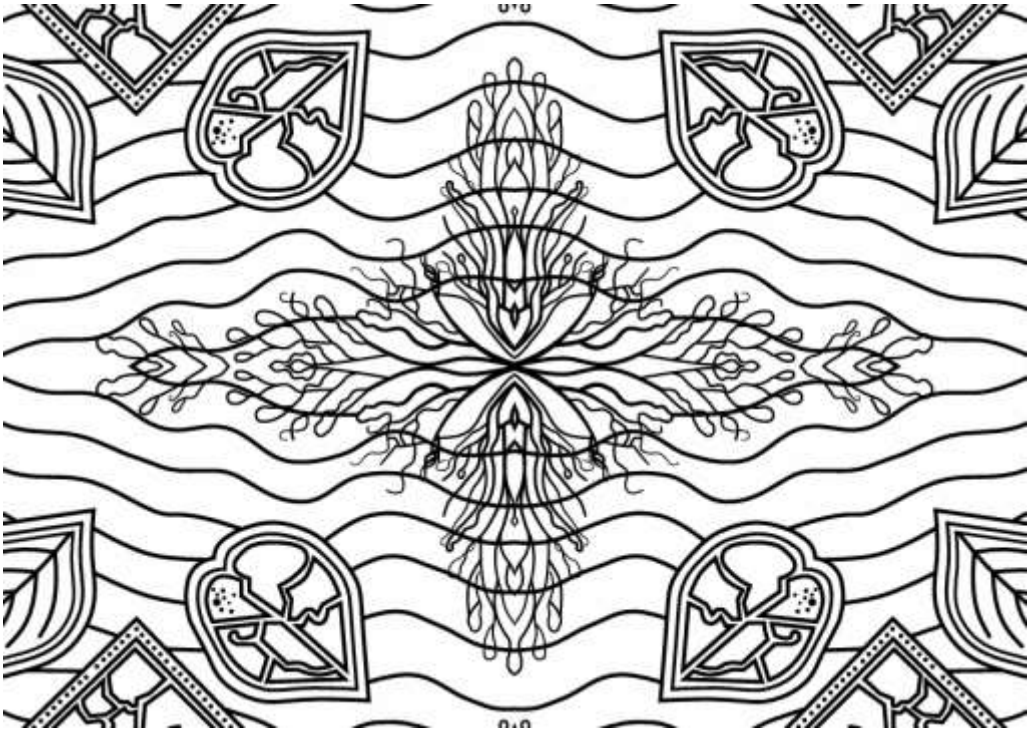

Figure 4. Batik Motifs

a. Philosophy

This batik motif represents the balance between nature, life, and science. Each shape symbolizes the cycle of human life, facing illness and striving to survive. The biological and social systems of humans adapt to environmental threats such as exposure to ultraviolet rays, and the circular and wave patterns reflect the dynamics of life that are

constantly moving and changing. All illnesses are not the end; they are part of the cycle towards a new balance, like leaves and branches at the end. As a result, this batik becomes a symbol of balance, optimism, and wisdom in facing life's trials through an understanding of religion and science.

b. Biology

As shown by the SEIR model for skin cancer, this motif biologically reflects the structure and processes of human skin when exposed to ultraviolet light. The wave pattern in the background shows the diffusion of ultraviolet energy penetrating the skin layers, while the crack-like and leaf vein-like patterns show cellular changes caused by DNA mutations. The circular pattern with a core inside shows the structure of cells and the phase of uncontrolled division. The brown and orange patterns show inflammation occurring in the epidermal layer due to exposure to ultraviolet rays. According to the SEIR class classification (susceptible, exposed, infected, and recovered), this design illustrates the biological processes underlying skin cancer from the susceptible phase to the recovery phase.

c. Mathematical

From a mathematical perspective, this batik shows the balance of a dynamic system in the SEIR model, with each motif showing the relationship between the variables of the differential system. The repeating geometric shapes show the stability of the equilibrium point and the periodicity of population changes for each class. The layered and interconnected patterns show the relationship between the parameters β, γ, and ε that affect the rate of recovery and transmission. The continuous solutions of various systems are shown in smooth background waves, which indicate the progression of time in the skin cancer patient population. The shape and color of this batik are harmonious, indicating a stable system. As a result, this batik motif is more than just a work of art. This batik is a visual representation of the numerical balance between disease, healing, and the continuity of life.

Conclusion

An analysis of the stability of the SEIR model for skin cancer in South Sulawesi Province shows that both equilibrium points, namely the disease-free point and the infected point, are asymptotically stable based on the negative eigenvalues of the Jacobian matrix. These results indicate that when the parameters are set, the disease dynamics tend to return to stability.

Using numerical simulations with three variations in the recovery rate (R), it was found that higher values indicate that the number of infected people decreases significantly and the recovered population increases. Conversely, lower values indicate that the number of cases tends to remain high. Therefore, the recovery rate is a major factor in controlling the spread of skin cancer and determining the achievement of disease-free conditions.

One example of how epidemiological data can be used as an educational tool in cultural media is the use of model results and data visualization in the creation of the "Sulur Harapan" batik motif. This motif conveys health messages and aesthetic value while representing the spread of skin cancer according to the SEIR model.

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