

EXPLORING THE FUTURE OF HEALTH THROUGH THE SELR MATHEMATICAL MODEL WITH TIME DELAY ON THE RISK OF DIABETES AMONG MATHEMATICS STUDENTS OF FMIPA UNM DUE TO UNHEALTHY LIFESTYLES

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Abstrak. Penelitian ini bertujuan untuk membangun model SELR dengan waktu tunda pada kasus diabetes, menganalisis model tersebut, dan melakukan simulasi untuk memprediksi tingkat kejadian diabetes. Mengingat meningkatnya prevalensi diabetes di kalangan mahasiswa, penelitian ini sangat penting untuk mengidentifikasi faktor-faktor yang mempengaruhi penyebaran penyakit ini dan untuk memberikan solusi yang efektif dalam pengendaliannya. Penelitian ini merupakan gabungan antara kajian teori dan aplikasi. Analisis model SELR dengan waktu tunda difokuskan pada kasus diabetes, sedangkan simulasi dilakukan menggunakan Software Maple. Proses penelitian yang dilakukan meliputi pengumpulan data mengenai tingkat kejadian diabetes di kalangan mahasiswa aktif FMIPA UNM; membangunan model matematis SELR dengan waktu tunda yang direpresentasikan sebagai sistem persamaan diferensial; analisis titik kesetimbangan untuk mengidentifikasi titik kesetimbangan bebas dan endemik; menghitun nilai bilangan reproduksi dasar (R₀) untuk menentukan potensi penyebaran penyakit; dan simulasi model untuk memprediksi dampak dari intervensi solusi terhadap penyebaran diabetes. Populasi penelitian adalah mahasiswa aktif FMIPA UNM, dengan jumlah sampel sebanyak 1.000 mahasiswa yang diperoleh menggunakan teknik Slovin. Penelitian ini menghasilkan model matematika SELR dengan waktu tunda untuk kasus diabetes yang direpresentasikan sebagai sistem persamaan diferensial. Analisis model menunjukkan adanya titik kesetimbangan bebas dari kasus diabetes serta titik kesetimbangan endemik yang stabil. Selain itu, hasil penelitian ini menemukan nilai bilangan reproduksi dasar (Ro) untuk kasus tanpa solusi sebesar 25,97333855, yang berarti satu individu dapat mempengaruhi 25-26 orang di lingkungan FMIPA UNM. Namun, jika solusi diterapkan, nilai Ro menurun menjadi 0,7502918529, menunjukkan tidak terjadi penyebaran secara psikologis, di mana setiap individu tidak mempengaruhi individu lainnya.

Kata Kunci: Diabetes, Model SELR, Titik Kesetimbangan, Bilangan Reproduksi Dasar

Abstract. This research aims to develop a delayed SELR model for diabetes, analyze the model, and conduct simulations to predict the incidence rate of diabetes. Given the increasing prevalence of diabetes among students, this study is crucial for identifying factors that influence the spread of this disease and providing effective for its control. This research combines





theoretical studies and applications. The analysis of the delayed model focuses on diabetes, while simulations are conducted using Maple software. The research process includes: collecting data on the incidence of diabetes among active students at FMIPA UNM; constructing a mathematical model of the delayed SELR represented as a system of differential equations; analyzing equilibrium points to identify free and endemic equilibrium points; calculating the basic reproduction number (R_0) to determine for disease spread; and simulating the model to predict the impact of solution interventions on the spread of diabetes. The study population consists of students at FMIPA UNM, with a sample size of 1,000 students obtained using the Slovin technique. This research produces a mathematical model of the delayed SELR for diabetes cases represented as a system of differential equations. The model analysis the existence of free equilibrium points for diabetes and stable endemic equilibrium points. Furthermore, the findings indicate that the basic reproduction number (R_0) without solutions is 25.97333855, meaning one individual can influence 25-26 others in the FMIPA UNM environment. However, when solutions are applied, the R_0 value decreases to 0.7502918529, indicating no psychological spread occurs, where each individual does not influence others.

Keywords: Diabetes, SELR Model, Equilibrium Point, Basic Reproduction Number

A. INTRODUCTION

Diabetes Mellitus (DM) poses a global challenge that threatens individuals and communities alike. One of the key factors contributing to the onset of DM is an unhealthy lifestyle. An irregular lifestyle, marked by poor dietary choices and inadequate physical activity, is at the root of the increasing incidence of DM. The situation in Indonesia is becoming increasingly alarming. According to data from the Health Research and Development Agency (Balitbangkes), the prevalence of DM in Indonesia is rising. In 2018, approximately 10.9 million individuals suffered from DM, and this number is projected to continue growing (Hsu et al., 2021).

Numerous studies have highlighted the multifaceted nature of DM. A recent study conducted in Taiwan in 2021 identified that excessive intake of foods high in sugar and fat, along with a lack of physical activity and rising obesity rates, creates a perilous combination that drives the increase in DM cases (Kumar et al., 2021). In the United States, researchers have utilized mathematical models to predict the progression of DM, taking into account risk factors such as obesity, insulin resistance, and smoking. They developed computer simulations aimed at identifying effective prevention strategies (Lau et al., 2021). Similarly, in India, mathematical models have been employed to forecast future DM cases by considering dietary habits, smoking, and physical inactivity. The simulation results indicated that implementing appropriate preventive measures could significantly reduce the number of DM cases (Hsu et al., 2021; Kumar et al., 2021; Lau et al., 2021).

A thorough understanding of these factors is essential. Therefore, this study will focus on undergraduate students in the Faculty of Mathematics and Natural Sciences. By utilizing data from interviews, the study aims to analyze the impact of unhealthy lifestyles on future generations and develop improvement strategies through mathematical modeling. Drawing on various leading research efforts, this study seeks to offer better solutions to address the potential rise of DM resulting from poor lifestyle choices. With an enhanced understanding of the DM issue, the current situation in Indonesia, and its preventive measures, we can make significant strides in improving overall public health (Abdy et al., 2021; Annas et al., 2020).

B. Method





This research is a hypothetical and applicative exploration, with the aim of testing the analysis and simulation of the behavior of potential diabetes generations caused by unhealthy lifestyles, focusing on a case study of Mathematics students at the Faculty of Mathematics and Natural Sciences (FMIPA) of Makassar State University (UNM). This research uses a Mathematical Modeling approach and was conducted in the Makassar City area, South Sulawesi. The data used in this study covers the population of South Sulawesi Province, which is around 80,965 individuals. The research process involves a series of important steps. First, the formation of a time-delayed SELR model to model the spread of potential diabetes generations due to unhealthy lifestyles, with a case study of Mathematics students at FMIPA UNM. This step includes measuring assumptions, identifying variables, and determining relevant parameters for the time-delayed SELR model.

Next, the time-delay SELR model was analyzed by determining the equilibrium point, evaluating the type of stability based on the eigenvalue, and calculating the basic reproduction number (R_0) . In this context, a high basic reproduction number (R_0) value indicates a higher rate of spread, while a lower value indicates the opposite (Annas, et al., 2020). The final step involves simulating the time-delay SELR model to describe the potential spread of diabetes generation due to unhealthy lifestyles, using Maple software, in the Makassar City area, South Sulawesi Province. This study is expected to provide important contributions in understanding the impact of lifestyle on health, as well as providing valuable insights in diabetes prevention efforts among Mathematics students at UNM and the general public.

C. RESULT AND DISCUSSION

1 Formation of SELR Model of Diabetes Generation Delay Time

In this study, the population in this model is divided into three classes: Suspectible Class (S), which consists of individuals who are susceptible to becoming candidates for the generation of diabetes due to unhealthy lifestyles with a total of 176 samples; Exposed Class (E), which represents individuals who are influenced by the environment of other individuals to become candidates for the generation of diabetes due to unhealthy lifestyles but are not yet included in that group with a total of 96 samples; Lifestyle Class (L), which represents individuals who are included in the group of candidates for the generation of diabetes due to unhealthy lifestyles with a total of 320 samples; and Recover Class (R), which is free from the group of candidates for the generation of diabetes due to unhealthy lifestyles with a total of 408 samples, so that the total sample in this study is 1000 obtained from the sampling process using the Slovin formula for the number of students at the Faculty of Mathematics and Natural Sciences. Population changes in the problem of social cases of student interest with the SELR time delay model can be interpreted in Figure 1, while the definition of variables and parameters of the SELR time delay model are presented in Table 1.

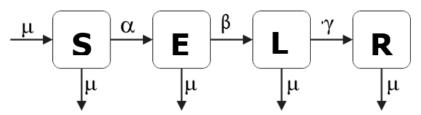


Figure 1. Schematic of SELR Model of Delay Time for Future Generation of Diabetes Due to Unhealthy Lifestyle.





Table 1. SELR Model Parameters for Delay Time of Potential Generation of Diabetes Due to Unhealthy Lifestyle.

Variable /	Definition	
Parameter	Definition	
S(t)	The number of population that is susceptible to entering the group of potential diabetes generations due to unhealthy lifestyles.	
E(t)	The population is starting to be affected by the group of individuals who are potential generations of diabetes due to unhealthy lifestyles but are not yet included in the group of potential generations of diabetes due to unhealthy lifestyles.	
L(t)	The number of population included in the group of potential diabetes generations due to unhealthy lifestyles.	
R(t)	The number of population that is free from the group of potential generations of diabetes due to unhealthy lifestyles.	
N	Total population	
α	The population rate that has begun to be affected by the group of potential generations of diabetes due to unhealthy lifestyles, but has not yet become part of the group of potential generations of diabetes due to unhealthy lifestyles.	
β	The population growth rate that has habits that are considered as potential generations of diabetes due to unhealthy lifestyles.	
γ	The population rate that has carried out or experienced healthy habits or lifestyles, especially as a potential generation of diabetes due to unhealthy lifestyles.	
μ	The population rate that enters as new students or transfer students or leaves campus as graduates or Drops Out	

N = S + E + L + R, where N is the total sample.

2 Data Analysis

Previously, the data collection process has been carried out by distributing questionnaires or surveys online and offline. After the data is collected, the analysis process is carried out so that it becomes quantitative as follows:

Table 1. Results of Data Analysis related to SELR Model Parameters for Diabetes Generation Delay Time .

Parameter	Data	Source
α	0.45444	
$oldsymbol{eta}$	0.5840	
γ	0.5721	Online and Offline Questionnaire
μ	0.17	

3 Equilibrium Point Analysis of the SELR Time Delay Model

Based on Figure 1, the mathematical model of the SELR time delay is obtained . This can be seen in Equations (1) to (4):

$$\frac{dS}{dt} = \mu - \alpha SL - \mu S \tag{1}$$

$$\frac{dE}{dt} = \alpha S - \beta E - \mu E \tag{2}$$

$$\frac{dL}{dt} = \beta E - \gamma L(\tau - t) - \mu L \tag{3}$$

$$\frac{dR}{dt} = \gamma L - \mu R \tag{4}$$





Determining the Equilibrium Point

Equations (1) to (4) have equilibrium points which are solutions to $\frac{dS}{dt}$, $\frac{dE}{dt}$, $\frac{dE}{dt}$, $\frac{dR}{dt}$ = (0,0,0,0). If the right side of the equation is equated to zero, then equations (5) to (8) are obtained, namely:

$$\frac{dS}{dt} = \mu - \alpha SL - \mu S = 0 \tag{5}$$

obtained, namely:
$$\frac{dS}{dt} = \mu - \alpha SL - \mu S = 0 \tag{5}$$

$$\frac{dE}{dt} = \alpha S - \beta E - \mu E = 0 \tag{6}$$

$$\frac{dL}{dt} = \beta E - \gamma L(\tau - t) - \mu L = 0 \tag{7}$$

$$\frac{dR}{dt} = \gamma L - \mu R = 0 \tag{8}$$

$$\frac{d\tilde{L}}{dt} = \beta E - \gamma L(\tau - t) - \mu L = 0 \tag{7}$$

$$\frac{dR}{dt} = \gamma L - \mu R = 0 \tag{8}$$

The equilibrium point in this time delay SELR model is formed by two equilibrium point conditions, namely the free equilibrium point and the equilibrium point.

4.1 4.1 Free Equilibrium Point

Free can be assumed that there is no population. The free equilibrium point occurs if L =0. In this situation, equations (5) to (8) are used to represent the equilibrium as follows.

Trait 1

time delay free equilibrium point of the SELR model is given as:

$$(S, E, L, R) = (1, 0, 0, 0)$$

So, based on the free equilibrium point for the SELR model, the time delay after the parameter values in table 1 are substituted is (S,E,L,R) = (1,0,0,0).

Proof:

By making L = 0 equation (7) subtitusi ke persamaan (5,6,8) we obtain equations (9) to (12):

$$L = 0 (9)$$

$$\mu - \alpha SL - \mu S = 0$$

$$S = \frac{\mu}{\mu} = 1 \tag{10}$$

$$\alpha SL - \beta E - \mu E = 0$$

$$\alpha SL - \beta E - \mu E = 0$$

$$E = \frac{\alpha SL}{\beta + \mu} = \frac{0}{\beta + \mu} = 0$$
(11)

$$\nu L(\tau - t) - \mu R = 0$$

$$\gamma L(\tau - t) - \mu R = 0
R = \frac{\gamma L(\tau - t)}{\mu} = \frac{0}{\mu} = 0$$
(12)

So, we get an equilibrium point which is symbolized by

$$(S, E; L; R) = \left(\frac{\mu}{\mu} = 1; \ 0; \ 0; 0\right)$$
 (13)

4.2 **Non-Free Equilibrium Point**

Determining the non-free equilibrium point is obtained by making the left side of equations (1.1) to (1.3) have a value of zero, then looking for a solution in the form of the values of the variables S*, E*, L*, R*.

Trait 2

The endemic equilibrium point of the time-delay SELR model is given as:





$$(S^*, E^*, L^*, R^*) = \begin{pmatrix} \frac{\left(\beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^2\right)}{(\alpha\beta)}, \\ \frac{\mu\left(\frac{\beta E}{(\gamma(\tau-t) + \mu}\right)\left(-\alpha\beta + \beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^2\right)}{(\beta(\beta + \mu)\alpha)}, \\ \frac{\mu\left(-\alpha\beta + \beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^2\right)}{\left(\alpha(\beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^2\right)}, \\ \frac{\gamma\left(-\alpha\beta + \beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^2\right)}{(\alpha(\beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^2))} \end{pmatrix}$$

Proof:

Making the simple algebraic operation equation from equations (2 & 3) to be zero, we obtain

$$L1 = \frac{\beta E}{\gamma(\tau - t) + \mu} \tag{14}$$

$$L2 = \frac{\beta E + \mu E}{\alpha S}$$

$$\frac{\beta E}{(\gamma(\tau - t) + \mu)} = \frac{\beta E + \mu E}{\alpha S}$$

$$(\gamma(\tau - t) + \mu)(\beta + \mu)(E) = \alpha S(\beta)(E)$$

$$\frac{(\gamma(\tau - t) + \mu)(\beta + \mu)(E)}{E} = \alpha S(\beta)$$

$$(\gamma(\tau - t) + \mu)(\beta + \mu) = \alpha S(\beta)$$

$$S = \frac{(\gamma(\tau - t) + \mu)(\beta + \mu)}{\alpha(\beta)}$$

$$S = \frac{(\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\alpha \beta)}$$
Then the value of $s^* = \frac{(\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\alpha \beta)}$
(16)

with equations (2 & 3) equated to zero, we obtain equation (17). Substitute S* then,

$$E = \frac{\alpha SI}{\beta + \mu} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) \frac{(\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\alpha \beta)}}{\beta + \mu} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \beta + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \beta + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \beta + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \beta + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \beta + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \beta + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \beta + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \beta + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \beta + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \beta + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \beta + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \beta \gamma(\tau - t) + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \gamma(\tau - t) + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \beta \mu + \gamma(\tau - t)\mu + \mu^{2})}{(\beta \gamma(\tau - t) + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \mu^{2})}{(\beta \gamma(\tau - t) + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \mu^{2})}{(\beta \gamma(\tau - t) + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \mu^{2})}{(\beta \gamma(\tau - t) + \mu)(\alpha \beta)} = \frac{\alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \mu^{2})}{(\beta \gamma(\tau - t) + \mu)(\alpha \beta)} = \frac{\alpha \alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \mu^{2})}{(\beta \gamma(\tau - t) + \mu)(\alpha \beta)} = \frac{\alpha \alpha \left(\frac{\beta E}{(\gamma(\tau - t) + \mu}\right) (\beta \gamma(\tau - t) + \mu^{2})}{(\beta \gamma(\tau - t) + \mu)(\alpha \beta)} = \frac{\alpha \alpha \alpha \alpha \beta}{(\beta \gamma(\tau - t) + \mu)(\alpha \beta)} = \frac{\alpha \alpha \alpha \alpha \beta}{(\beta \gamma(\tau - t) + \mu)(\alpha \beta)} = \frac{\alpha \alpha \alpha \alpha \beta}{(\beta \gamma(\tau - t) + \mu)(\alpha \beta)} = \frac{\alpha \alpha \alpha \alpha \beta}{$$

Then the value of
$$E^* = \frac{\mu(-\alpha\beta + \beta\gamma(\tau - t) + \beta\mu + \gamma(\tau - t)\mu + \mu^2)}{(\beta(\beta + \mu)\alpha)}$$
 (18) with equation (3) equated to zero, then equation (19) is obtained.

Substitute E* then,

$$L = \frac{\beta\left(\frac{\mu(-\alpha\beta + \beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^{2})}{(\beta(\beta + \mu)\alpha)}\right)}{\gamma(\tau-t) + \mu} = \frac{\mu(-\alpha\beta + \beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^{2})}{(\alpha(\beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^{2}))}$$
(19)

Then the value of
$$L^* = \frac{\mu(-\alpha\beta + \beta\gamma(\tau - t) + \beta\mu + \gamma(\tau - t)\mu + \mu^2)}{(\alpha(\beta\gamma(\tau - t) + \beta\mu + \gamma(\tau - t)\mu + \mu^2))}$$
 (20)

with equation (4) equated to zero, then equation (21) is obtained. Substitute L* then,





$$R = \frac{\gamma(\tau - t) \left(\frac{\mu(-\alpha\beta + \beta\gamma(\tau - t) + \beta\mu + \gamma(\tau - t)\mu + \mu^2)}{\left(\alpha(\beta\gamma(\tau - t) + \beta\mu + \gamma(\tau - t)\mu + \mu^2)\right)} \right)}{\mu} = \frac{\mu(-\alpha\beta + \beta\gamma(\tau - t) + \beta\mu + \gamma(\tau - t)\mu + \mu^2)}{\left(\alpha(\beta\gamma(\tau - t) + \beta\mu + \gamma(\tau - t)\mu + \mu^2)\right)}$$
(21)

Then the value of
$$R^* = \frac{\gamma(\tau - t)(-\alpha\beta + \beta\gamma(\tau - t) + \beta\mu + \gamma(\tau - t)\mu + \mu^2)}{(\alpha(\beta\gamma(\tau - t) + \beta\mu + \gamma(\tau - t)\mu + \mu^2))}$$
 (22)

So, we obtain the non-free equilibrium points which are symbolized by (S*, E*, L*, R*) as follows:

$$(S^*, E^*, L^*, R^*) = \begin{pmatrix} \frac{\left(\beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^2\right)}{(\alpha\beta)}, \\ \frac{\mu\left(\frac{\beta E}{(\gamma(\tau-t) + \mu}\right)\left(-\alpha\beta + \beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^2\right)}{(\beta(\beta + \mu)\alpha)}, \\ \frac{\mu\left(-\alpha\beta + \beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^2\right)}{\left(\alpha(\beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^2\right)}, \\ \frac{\gamma\left(-\alpha\beta + \beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^2\right)}{\left(\alpha(\beta\gamma(\tau-t) + \beta\mu + \gamma(\tau-t)\mu + \mu^2\right)} \end{pmatrix}$$

And then, the equilibrium point for the social bullying case after the parameter values in table 1. Substituted is $(S^*, E^*, L^*, R^*) = (2.1084, 0.2499, 0.1967, 0.6618)$.

4.3 **Basic Reproduction Number**

The basic reproduction number (R_0) can be determined using the next generation matrix method. This matrix is formed by considering the positive and negative components of the transmission rate from the time delay SELR model. The formula for calculating the basic reproduction number can be found in equation (23):

$$R = F' \cdot (V')^{-1} \tag{23}$$

Based on the Equation

$$\frac{dE}{dt} = \alpha SL - \beta E - \mu E = 0$$

(24)

$$\frac{dL}{dt} = \beta E - \gamma L - \mu L = 0$$

(25)

So that it is obtained

$$F = \begin{bmatrix} \alpha SL \\ \mathbf{0} \end{bmatrix}, F' = \begin{bmatrix} \mathbf{0} & \alpha S \\ \mathbf{0} & \mathbf{0} \end{bmatrix}$$
 (26)

$$V = \begin{bmatrix} \beta E + \mu E \\ \gamma(\tau - t)L + \mu L + \beta E \end{bmatrix}, V_{h'} = \begin{bmatrix} \beta + \mu & 0 \\ \beta & \gamma(\tau - t) + \mu \end{bmatrix}$$
Then we get the inverse of the matrix of equation (27), namely

$$(V')^{-1} = \begin{bmatrix} \frac{1}{\beta + \mu} & \mathbf{0} \\ \frac{\beta}{(\beta + \mu)(\gamma(\tau - t) + \mu)} & \frac{1}{\gamma(\tau - t) + \mu} \end{bmatrix}$$
(28)

Next, the eigenvalues of the R matrix will be determined, based on Equation (28).





$$R = \begin{bmatrix} \mathbf{0} & \alpha S \\ \mathbf{0} & \mathbf{0} \end{bmatrix} \begin{bmatrix} \frac{1}{\beta + \mu} & \mathbf{0} \\ \frac{\beta}{(\beta + \mu)(\gamma(\tau - t) + \mu)} & \frac{1}{\gamma(\tau - t) + \mu} \end{bmatrix}$$

$$R = \begin{bmatrix} \frac{\alpha S \beta}{(\beta + \mu)(\gamma(\tau - t) + \mu)} & \frac{\alpha S}{\gamma + \mu} \\ \mathbf{0} & \mathbf{0} \end{bmatrix}$$
(29)

After obtaining the matrix R In equation (29), the eigenvalues will then be searched for using the formula $det(\lambda I - R) = 0$, where I is the identity matrix. The basic reproduction number will be determined based on the largest eigenvalues (λ).

$$|\lambda I - R| = \left| \begin{pmatrix} \lambda \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix} - \begin{bmatrix} \frac{\alpha S \beta}{(\beta + \mu)(\gamma(\tau - t) + \mu)} & \frac{\alpha S}{\gamma(\tau - t) + \mu} \end{bmatrix} \right| = 0$$
(30)

So by using the matrix addition operation above, two lambda values are obtained which are eigenvalues based on equation (30), namely

$$\lambda_{1,2} = \left[\frac{\alpha S \beta}{(\beta + \mu)(\gamma(\tau - t) + \mu)}, 0 \right]$$

So the lambda value above is the largest eigenvalue, namely

$$\lambda = \left[\frac{\alpha S \beta}{(\beta + \mu)(\gamma(\tau - t) + \mu)} \right]$$

with the largest lambda value compared to λ_2 the assumption of parameter rates and the presence of a population in the model. So based on the largest eigenvalue substituted for the free equilibrium point value, the basic reproduction number is obtained:

$$R_0 = \frac{\alpha\mu\beta}{(\beta+\mu)(\gamma(\tau-t)+\mu)\mu} \tag{31}$$

Based on the equation above, the basic reproduction number for cases wit out a solution will produce $R_0n = 25.97333855$ which means in social cases where one individual can influence 25-26 people in the FMIPA UNM environment, but on the other hand if the case is given a solution then it will produce $R_0s = 0.7502918529$ which means that there is no psychological spread, where each individual does not influence other individuals.

4.4 Stability of the SELR Time Delay Model

Based on equations (1) - (4), the Jacobian matrix (J) can be formed as follows::

$$J = \begin{bmatrix} -\alpha L - \mu & 0 & -\alpha s & 0\\ \alpha L & -\beta - \mu & \alpha s & 0\\ 0 & \beta & -\gamma(\tau - t) - \mu & 0\\ 0 & 0 & \gamma(\tau - t) & -\mu \end{bmatrix}$$
(32)

Theorem





The equilibrium point in the SELR model is considered stable if the basic reproduction number (R_0) is less than or equal to 1 $(R_0 \le 1)$, and is considered unstable if R_0 is greater than 1 $(R_0 > 1)$.

Proof:

The equilibrium points are inserted into the J matrix in equation (32).,

$$J = \begin{bmatrix} -\alpha L - \mu & 0 & -\alpha s & 0 \\ \alpha L & -\beta - \mu & \alpha s & 0 \\ 0 & \beta & -\gamma(\tau - t) - \mu & 0 \\ 0 & 0 & \gamma(\tau - t) & -\mu \end{bmatrix}$$

Then, the eigenvalues of the matrix in equation (32) are determined, with the following description:

$$|\lambda I - J| = \mathbf{0}$$
• $|\lambda I - J| = \left| \begin{pmatrix} \lambda \begin{bmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{bmatrix} - \begin{bmatrix} -\alpha L - \mu & 0 & -\alpha s & 0 \\ \alpha L & -\beta - \mu & \alpha s & 0 \\ 0 & \beta & -\gamma(\tau - t) - \mu & 0 \\ 0 & 0 & \gamma(\tau - t) & -\mu \end{bmatrix} \right| = 0$

$$|\lambda I - J| = \begin{bmatrix} -\alpha L - \mu - \lambda & 0 & -\alpha s & 0\\ \alpha L & -\beta - \mu - \lambda & \alpha s & 0\\ 0 & \beta & -\gamma(\tau - t) - \mu - \lambda & 0\\ 0 & 0 & \gamma(\tau - t) & -\mu - \lambda \end{bmatrix} = 0$$
(33)

Next, the determinant will be calculated, resulting in:

$$\alpha\beta\gamma(\tau - t)L\lambda + \alpha\beta\gamma(\tau - t)L\mu + \alpha\beta L\lambda^{2} + 2\alpha\beta L\gamma(\tau - t)\mu + \alpha\beta L\mu^{2} - \alpha\beta\lambda^{2}s - 2\alpha\beta\lambda\mu s - \alpha\beta\mu^{2}s + \alpha\gamma(\tau - t)L\lambda^{2} + 2\alpha\gamma(\tau - t)L\lambda\mu + \alpha\gamma(\tau - t)L\mu^{2} + \alpha L\lambda^{3} + 3\alpha L\lambda^{2}\mu + 3\alpha L\lambda\mu^{2} + \alpha L\mu^{3} + \beta\gamma(\tau - t)\lambda^{2} + 2\beta\gamma(\tau - t)\lambda\mu + \beta\gamma(\tau - t)\mu^{2} + \beta\lambda^{3} + 3\beta\lambda^{2}\mu + 3\beta\lambda\mu^{2} + \beta\mu^{3} + \gamma\lambda^{3} + 3\gamma(\tau - t)\lambda^{2}\mu + 3\gamma(\tau - t)\lambda\mu^{2} + \gamma(\tau - t)\mu^{3} + \lambda^{4} + 4\lambda^{3}\mu + 6\lambda^{2}\mu^{2} + 4\lambda\mu^{3} + \mu^{4}$$
(34)

According to Descartes' rule of signs, equation (34) will have only negative roots if all terms are positive. Therefore, the equilibrium point for bullying behavior is deemed stable when $R_0 \le 1$ and unstable when $R_0 > 1$.

5 Eigenvalues

Based on equation (34), the eigenvalues obtained from the values (λ) obtained at the previous equilibrium point are real and negative numbers. Referring to the stability properties, the type of stability at this equilibrium point is asymptotically stable. So, the stability of the SELR time delay Model of Diabetes Generation is (S, E, L, R) = (-0.1700000000, -0.1700000000, -0.99830522640, -0.41089477360) and (S*, E*, L*, R*) = $(-1.3 \times 10^{-22}, -4.174249888 \times 10^{-11}, 9.260254038 \times 10^{-22}, -0.17$). Therefore, the type of stability at both equilibrium points is asymptotic stability.

6 Results of the SELR Model of Delay Time for the Future Generation of Diabetes Due to Unhealthy Lifestyles.

In this case, a simulation will be carried out based on the data that has been successfully collected as follows and shown in the graphs in Figure 2 and Figure 3. SELR mathematical model of time delay can be seen in Figures 2 & 3 as follows.





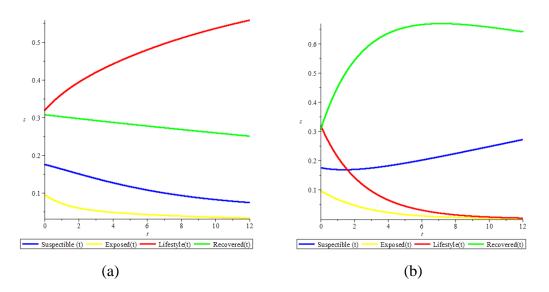


Fig. 2. Plot of SELR model with solution (a) and without solution (b) for potential generation of diabetes due to unhealthy lifestyle.

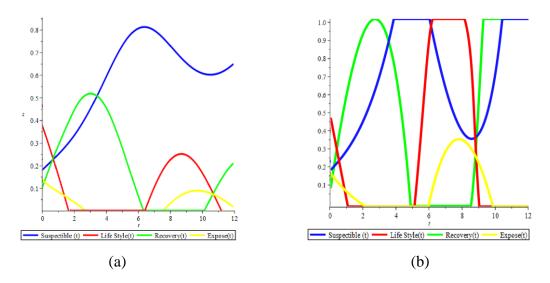


Fig. 3. Plot of the time-delay SELR model with solution (a) and without solution (b) on the potential generation of diabetes due to unhealthy lifestyle.

In Figure 2. Part (a) it can be seen that the number of samples that are likely to become candidates for the generation of diabetes due to unhealthy lifestyles (blue line), has decreased from an observation of 0.18 to 0.09 in 12 months. Samples of individuals who are influenced by other individuals and their environment (yellow line) also decreased from 0.1 to almost 0 in 12 months. Samples that have been included in the group of candidates for the generation of diabetes due to unhealthy lifestyles experienced (red line) a very significant increase from 0.31 to 0.56 in 12 months. And finally for samples that are free from candidates for the generation of diabetes due to unhealthy lifestyles experienced (green line) a decrease of 0.32 to 0.25 if we are not given a solution in 12 months.

In Figure 2. Part (a) it can be seen that the number of samples that are likely to become candidates for the generation of diabetes due to unhealthy lifestyles (blue line), has decreased from an observation of 0.18 to 0.09 in 12 months. Samples of individuals who are influenced by other individuals and their environment (yellow line) also decreased from 0.1 to almost 0 in





12 months. Samples that have been included in the group of candidates for the generation of diabetes due to unhealthy lifestyles experienced (red line) a very significant increase from 0.31 to 0.56 in 12 months. And finally for samples that are free from candidates for the generation of diabetes due to unhealthy lifestyles experienced (green line) a decrease of 0.32 to 0.25 if we are not given a solution in 12 months.

In Figure 2. Part (b) it can be seen that the number of samples that are likely to become candidates for the generation of diabetes due to unhealthy lifestyles (blue line), increased from the observation point of 0.23 to point 0.31 in 12 months. Samples of individuals who are affected by and their environment become candidates for the generation of diabetes due to unhealthy lifestyles but are not included in the group of candidates for the generation of diabetes due to unhealthy lifestyles (yellow line) decreased from 0.19 to a point of almost 0 in 12 months. This also happened to individuals who were included in the candidate generation of diabetes due to unhealthy lifestyles (red line) at the beginning at point 0.31 experiencing a decrease in 12 months, to almost reaching point 0 in the 12th month. Furthermore, individuals experienced a very significant increase (green line) until the 7th month with a peak point of 0.67 with therapy solutions, counseling guidance, health seminars, etc., although individuals after the 7th month experienced a decrease until the 12th month of approximately 5%.

In Figure 3. Part (a) it can be seen that the number of samples that are likely to become candidates for the generation of diabetes due to unhealthy lifestyles (blue line), experienced a gradual increase from observation 0.18 to 0.84 in 6 months, then decreased in 4 months and increased again until the 12th month. Samples of individuals who were influenced by friends and their environment also experienced (yellow line) a decrease from 0.1 to point 0 in 3 months and increased again in the 6th month and decreased again to zero 1 and a half months later. Samples that had been included in the group of candidates for the generation of diabetes due to unhealthy lifestyles experienced (red line) a very significant decrease in 1 month from 0.36 to 0 in and increased again in the 7th month to point 0.25 in the 9th month and decreased again to 1. And finally, for samples that are free from potential diabetes generations due to unhealthy lifestyles (green line), there was a very significant increase from 0.36 to 0.52 in 3 months and fell back to 0 in the following 3 months and rose again in the 10th to 12th months.

In Figure 3. Part (b) it can be seen that the number of samples that are likely to become candidates for the generation of diabetes due to unhealthy lifestyles (blue line), has increased from the observation point 0.23 to point 1 in 12 months. Samples of individuals who are affected by and their environment become candidates for the generation of diabetes due to unhealthy lifestyles but are not included in the group of candidates for the generation of diabetes due to unhealthy lifestyles decreased (yellow line) from 0.19 to a point of almost 0 in 12 months. This also happened to individuals who were included in the candidate generation of diabetes due to unhealthy lifestyles (red line) at the beginning at point 0.31 experiencing a decrease in 12 months, to almost reaching point 0 in the 12th month. Furthermore, individuals experienced (green line) a very significant increase until the 7th month with a peak point of 1 with therapy solutions, counseling guidance, health seminars, etc., although individuals after the 7th month experienced a decrease until the 12th month.

In general, the results of the simulation of the SELR mathematical model of time delay in the FMIPA UNM environment can be seen in the basic reproduction number or R_0 which is a number that can explain the potential for spread in a population. Where the basic reproduction number will be obtained for cases without a solution will produce $R_0 n = 25.97333855$ which means in social cases where one individual can influence 25-26 people in the FMIPA UNM environment, but on the contrary if the case is given a solution it will produce $R_0 s = 0.7502918529$ which means that there is no psychological spread, where each individual does not affect other individuals.





7 Discussion

In this study, we conducted an analysis and simulation of a mathematical model aimed at assessing the potential for future generations to develop diabetes due to unhealthy lifestyle habits, with a focus on Mathematics students at FMIPA UNM. The simulation results reveal significant changes in behavior and risk levels for potential future diabetes cases over a 12-month period.

Figure 2, depicting the results without solutions, shows a decrease in the proportion of samples at risk of diabetes due to unhealthy lifestyles from 18% to 9% over the period. This indicates a growing awareness among FMIPA UNM Mathematics students about the importance of maintaining a healthy lifestyle. The proportion of samples influenced by peers and environment also significantly dropped from 10% to nearly 0% in 12 months, reflecting the positive impact of a supportive environment for healthy living.

However, a notable increase occurred among samples already in the high-risk category, with rates rising from 31% to 56% over 12 months. This highlights the urgent need for further preventive measures and interventions for this group. Lastly, the proportion of individuals free from diabetes risk decreased from 32% to 25% over the same period in the absence of any solutions.

When solutions were applied, as shown in Figure 3, the proportion of samples potentially becoming future diabetes cases due to unhealthy lifestyles increased from 23% to 31% over 12 months. This indicates challenges in shifting to healthier lifestyles among Mathematics students. Nonetheless, a supportive environment had a positive impact, with individuals influenced by their surroundings but not in the risk group experiencing a significant drop from 19% to nearly 0%. Furthermore, individuals initially in the risk group saw a substantial increase to 67% by month 7 with the implementation of solutions such as therapy, counseling, and health seminars. Although there was a decline to 62% by month 12, this still demonstrates the positive effect of these interventions.

In comparison to previous studies, this research distinguishes itself with its approach and focus. Previous studies on habituating healthy behavior in children (Anisa et al., 2021) and clean living behavior in river basins and peatlands (Ulfa et al., 2022) generally employed qualitative methods, focusing more on literature reviews and direct observations without simulations or mathematical predictions. Research on factors affecting diabetic foot ulcers (Rahmawati, 2022) and the relationship between family social support and emotional stress in diabetes patients (Baktiar et al., 2022) also did not involve mathematical simulations but relied on statistical and empirical analyses.

This study, however, utilizes an advanced quantitative approach by applying mathematical modeling to predict and simulate the potential for diabetes due to unhealthy lifestyles. Unlike previous studies that examined general disease spread (Abdy et al., 2021; Annas et al., 2020; Bahtiar & Ariyanti, 2022; Side et al., 2016; Nurhaeda et al., 2021; Maryam et al., 2021; Anwar et al., 2021; Asri et al., 2021; Side & Noorani, 2013; Singhal, 2020; Soewono & Supriatna, 2020; Yang et al., 2020; Rangkuti et al., 2015; Rachman et al., 2019; Derouich et al., 2003; Etbaigha et al., 2018; Side & Noorani, 2012; Sanusi et al., 2021), this study provides a novel contribution by focusing specifically on diabetes risk among college students, offering deeper insights and more targeted solutions.

Thus, the simulation results from the SELR delayed time model at FMIPA UNM demonstrate that changes in individual behavior have a significant impact on the spread of potential diabetes cases due to unhealthy lifestyles. The basic reproduction number, R_0 , reflects the potential for spread within the population. The results indicate that the introduction of solutions can reduce R_0 from 25.97 to 0.75, implying no psychological spread. Previously, each individual could influence 25-26 others psychologically to become potential diabetes cases due to unhealthy lifestyles, but with the solutions in place, individuals no longer impact others. This





underscores the importance of a comprehensive prevention approach, including education, lifestyle changes, environmental support, and interventions such as therapy and counseling, to address diabetes risk in potential future generations caused by unhealthy lifestyles at FMIPA UNM.

D. Conclusion

In this study, a four-dimensional mathematical model, the SELR DELAY TIME model, was developed. The model identifies two equilibrium points one stable and one unstable along with their asymptotic stability and basic reproduction number (R_0). For the case without solutions, the basic reproduction number is $R_0 = 25.97333855$, indicating that, in a social context, one individual can influence 25-26 others within the FMIPA UNM environment. Conversely, when solutions are applied, the basic reproduction number is $R_0 = 0.7502918529$, signifying that psychological spread does not occur, as each individual no longer influences others.

The model also highlights that psychological therapy, counseling, and seminars play a crucial role in mitigating the negative impacts experienced by individuals involved in potential diabetes generation. This study emphasizes the role of psychological therapy as an intervention method that can help alleviate psychological behaviors among individuals at risk of diabetes. The solutions proposed assist individuals in identifying the causes of their behavior and developing strategies to manage emotions and conflicts in healthier ways.

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