

## INTEGRATING HOUSING ENVIRONMENTAL FACTORS INTO THE SEIR MODEL FOR PULMONARY TUBERCULOSIS TRANSMISSION: A CASE STUDY IN BANJAR, INDONESIA

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### ABSTRACT

Pulmonary Tuberculosis (TB) remains one of the serious public health issues in Indonesia, including in Banjar Regency. The transmission of TB is not only influenced by biological and behavioral factors but also highly depends on the characteristics of the living environment. This study aims to analyze the influence of physical environmental factors of housing on the incidence of pulmonary TB, and to integrate the analysis results into a modified SEIR model. The research was conducted using a cross-sectional observational approach involving 73 respondents from the working areas of Puskesmas Martapura 1 and Martapura 2. Data were collected through direct observation and interviews, and analyzed using binary logistic regression to identify significant variables. The significant variables were subsequently integrated into the transmission rate parameters in the SEIR model. The results show that ventilation area and room temperature have a significant impact on the incidence of pulmonary TB. Empirical findings show that the probability of pulmonary TB incidence is highest (86.68%) when both ventilation and temperature are below standard, and lowest (26.23%) when both meet the standards. Partial compliance still results in a high probability of incidence (around 60%). The SEIR model simulation with environmental scenarios shows that living conditions that do not meet ventilation area and temperature standards result in more aggressive TB transmission. Conversely, living conditions that meet both standards significantly reduce the number of infected individuals and increase the recovery rate. This research emphasizes the importance of environment-based interventions in a comprehensive TB control strategy.



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## 1. INTRODUCTION

Tuberculosis (TB) is an infectious disease caused by the *Mycobacterium tuberculosis* bacteria, a type of bacteria that enters the body through the respiratory system. This bacterium is rod-shaped and acid-fast, which is why it is often referred to as Acid-Fast Bacillus [1]. TB is a contagious disease that is mostly spread through the air. Most of the *Mycobacterium tuberculosis* bacteria that cause TB attack the lungs and are referred to as pulmonary TB. The *Mycobacterium tuberculosis* bacteria can also affect other organs, referred to as extrapulmonary TB [2].

According to the Global TB Report 2023 by the World Health Organization (WHO), tuberculosis (TB) remains a major health issue worldwide and was the second leading cause of death after COVID-19 in 2022 [3]. Every year, more than 10 million people are infected with TB, with an untreated mortality rate reaching 50%. About 87% of all TB cases worldwide were reported to have occurred in 30 countries, with the greatest TB incidence in 2022. With about 10% of all cases worldwide, Indonesia comes in second only to India. According to gender, men account for 55% of TB cases, followed by women (33%), and children (12%) [4]. Only around 8% of TB cases in Indonesia are extrapulmonary, with pulmonary TB accounting for 92% of cases [5]. One of the areas in Indonesia with a comparatively high incidence rate of tuberculosis is Banjar Regency, South Kalimantan, where cases have significantly increased. Based on data from the South Kalimantan Provincial Health Office, the number of TB cases in Banjar Regency increased from 387 cases in 2017 to 1,035 cases in 2023 [6]. To better understand the drivers behind these alarming statistics, it is essential to examine TB transmission within the framework of the epidemiologic triangle.

Based on the concept of the epidemiologic triangle, TB depends not only on the agent (bacteria) and host (susceptible humans) but also on the environment that supports the spread of the disease [7], [8]. *Mycobacterium tuberculosis* bacteria have been found to survive and be present in a variety of natural as well as artificial environments, possibly after being contaminated by human sources, as evidenced by experimental and epidemiological studies [9]. Furthermore, a study conducted by Verma et al. showed that TB DNA can survive and be detected on various surfaces in prison cells inhabited by active TB patients, especially on clothing, bedding, bathrooms, and room surfaces (such as floors, walls, windows, and ceilings) [10]. This indicates that the living environment may be a risk factor in the spread of *Mycobacterium tuberculosis*. Therefore, TB control strategies cannot rely solely on clinical aspects such as diagnosis and treatment but must also include environment-based interventions that target the physical conditions of the dwelling [11]. Based on this, the environment plays an important role in the disease transmission cycle, so comprehensive control efforts or strategies are needed. These efforts include improved sanitation, waste management, use of air ventilation, and implementation of policies that integrate human, animal, and environmental health so as not to create conditions that favor bacterial growth [12].

The cleanliness of one's living environment is very important in order to support efforts to control the spread of TB. Research by Heriyani et al. suggests that residential density, air temperature, ventilation, and natural lighting have a significant relationship with the incidence of pulmonary TB [13]. Some of the main factors that increase the risk of TB transmission include poor ventilation, which leads to the accumulation of *Mycobacterium tuberculosis* bacteria in the air and increases the likelihood of inhalation by healthy individuals. Homes with minimal air circulation, rarely opened windows, or inadequate ventilation systems contribute to the spread of these bacteria. In addition, high occupancy density accelerates transmission from one individual to another, especially when many people live in a small space with limited ventilation. Zhang and Zhang concluded that temperature and wind speed have a positive effect on the incidence of TB [14]. Furthermore, research by Xu et al. explained that average temperature and relative humidity are factors that affect the TB epidemic, so that it can be a practical reference for improving the TB warning system [15]. Research Ghadimi-Moghadam conducted a study to investigate how environmental and climatic factors, such as temperature, rainfall, humidity, altitude, slope, and land cover, affect the incidence of TB in southwestern Iran [16]. Later, research by Zhang et al. emphasized that the environment plays a role in the spread of TB, so a One Health approach is essential in understanding, detecting, and controlling *Mycobacterium tuberculosis* infection [12].

In epidemiological studies, mathematical models such as SEIR (Susceptible-Exposed-Infected-Recovered) have been widely used to understand the spread of infectious diseases, including tuberculosis (TB). Numerous studies have developed SEIR models to comprehend TB transmission dynamics and evaluate the effectiveness of control interventions. For instance, Kar and Mondal analyzed the SEITE model by considering exogenous reinfection and backward transcritical bifurcation in TB transmission dynamics [17].

Furthermore, Athithan and Ghosh developed an SEIR model incorporating case detection and treatment [18], while Zhang and Feng investigated the global stability of the SEIT model by including isolation and incomplete treatment [19]. Ullah et al. used TB data from Khyber Pakhtunkhwa, Pakistan, to model SLITR dynamics and estimate parameters based on epidemiological data [20]. Other studies by Nkamba et al. and Mengistu and Witbooi developed the SVELI model to assess the impact of vaccination and contact rates on TB transmission, highlighting that inadequate vaccine coverage could exacerbate transmission [21], [22]. More recently, Liana et al. developed a deterministic mathematical model of TB transmission that integrates malnutrition as a factor in disease dynamics [23], and James et al. extended the SEIR model of TB disease spread by considering imperfect vaccination and treatment failure in TB dynamics [24]. Environment-based epidemiological modeling approaches that include global stability and bifurcation analysis have incorporated environmental pollution into the models, as explored by Mettle et al. and Cai et al. [25], [26]. In addition, Li and Wang developed a model that considers TB transmission through contaminated environments and reinfection scenarios [27]. Meanwhile, Ullah et al. constructed a model emphasizing public awareness and treatment compliance [28]. Simulation results from their study indicated that enhanced awareness and environment-based interventions can significantly reduce TB infection rates. These models demonstrate that understanding the interaction between epidemiological, environmental, and behavioral factors is essential for designing more effective TB control strategies.

The previously described epidemiological models generally rely on biological parameters, such as transmission rate, incubation period, and duration of infection, obtained from literature or previous studies. These approaches have not directly considered the environmental conditions where people with pulmonary TB live. In reality, pulmonary TB transmission is not only determined by the interaction between healthy and infected individuals, but is also strongly influenced by the characteristics of the living environment, such as ventilation, occupancy density, natural lighting, humidity, and indoor temperature. Therefore, this study was conducted with the aim of developing an SEIR model that links environmental factors based on real data collected from respondents' homes. The model uses a logistic regression approach to integrate environmental factors with TB transmission rates in the SEIR model, resulting in a model that more realistically represents the spread of pulmonary TB disease.

Thus, this research not only enhances understanding of the role of physical environments in pulmonary TB transmission but also introduces a more contextual and applicable modeling approach. The findings of this study are expected to serve as a scientific basis for formulating environment-based interventions, particularly in regions with high TB burdens, to reduce TB incidence more effectively and sustainably.

## 2. RESEARCH METHODS

This study is an observational study with a cross-sectional design, in which risk factors and outcome variables were observed simultaneously at a specific point in time. Banjar Regency, Indonesia, has a relatively high prevalence of pulmonary TB, making it a suitable location for investigating the relationship between physical housing parameters and the incidence of pulmonary TB in the working areas of Martapura 1 and Martapura 2 primary Health Centers (Puskesmas). A total of 73 respondents participated in this study, with no missing data. The sample size of 73 was calculated using a standard formula for finite population sample estimation, based on the proportion of pulmonary TB cases in 2023 (26.83%), a total population of 2,135 individuals, and a 95% confidence level. The sampling method used was stratified proportional random sampling, with respondents distributed proportionally according to the number of TB cases in Martapura 1 and Martapura 2 Health Centers. Information on health problems or pulmonary TB history was obtained through interviews and records from local Health Centers, while environmental factors, specifically the physical parameters of housing, were directly observed through home visits to the respondents' residences. Ethics Approval: This study received ethical approval from the Health Research Ethics Committee of the DPD PPNI Banjarbaru City, Indonesia, with approval number 083/EC/KEPK-DPDPPNI/IX/2024.

Dependent variable: Pulmonary TB ( $Y$ ). Independent variable:

1. Environmental Factors, which include physical parameters of the residential housing environment: Ventilation area( $X_1$ ), Lighting ( $X_2$ ), Temperature ( $X_3$ ), Humidity ( $X_4$ ), and Occupancy density ( $X_5$ ). These environmental factor categories are based on the standards outlined in the Indonesian Ministry of Health Regulation No. 2 of 2023 [29].

2. Host Factors: Age ( $X_6$ ), Sex ( $X_7$ ), Education level ( $X_8$ ), Employment status ( $X_9$ ), Smoking status ( $X_{10}$ ), and the Smoking status of family members living in the same household as the respondent ( $X_{11}$ )

The analysis of factors influencing the incidence of pulmonary TB was conducted in three stages: univariate, bivariate, and multivariate analysis. Univariate analysis presents the distribution of research data based on the characteristics of respondents and the conditions of their household environment. Bivariate analysis was conducted using the Chi-square test to assess whether independent variables (namely, gender, age, education, occupation, respondent's smoking status, household members' smoking status, ventilation area, lighting, temperature, humidity, and housing density) have a statistically significant relationship with the incidence of pulmonary TB. The selection of initial variables for multivariate analysis includes all variables, and variables with a significance level of less than 0.25 in the Chi-square test are included in the binary logistic regression model [30], [31]. Binary logistic regression aims to predict the probability of a certain event occurring (pulmonary TB incidence) based on the independent variables included in the model. This multivariate analysis involves the Omnibus test, the Hosmer and Lemeshow goodness-of-fit test, and multicollinearity diagnostics.

A binary logistic regression model is a regression model used to model the relationship between one binary dependent variable and one or more independent variables. The binary dependent variable has only two possible values, such as '1' (success) or '0' (failure). Differentiating the binary logistic regression model from the linear regression model is that the dependent variable is dichotomous or consists of two categories, so the dependent variable is qualitative data [32]. The binary logistic regression model follows the Bernoulli distribution because it has two possibilities. The binary logistic regression model is used to predict the probability of the occurrence of the dependent variable based on the independent variables. The basic formula of the logistic regression model is as follows [33]:

$$\text{logit}(\pi) = \ln\left(\frac{\pi}{1-\pi}\right) = \mathbf{B}^T \mathbf{x} = b_0 + b_1 x_1 + b_2 x_2 + \dots + b_p x_p. \quad (1)$$

Where:

- $x_i$  : The  $i$ -th independent variable, ( $i = 1, 2, \dots, p$ )
- $b_0$  : Intercept (constant)
- $b_i$  : Binary logistic regression coefficient for the independent variable  $x_i$
- $\frac{\pi}{1-\pi}$  : Odds ratio, which is the ratio between the probability of occurrence and non-occurrence of the event

Following the statistical analysis, the probability of pulmonary TB incidence is obtained as a function of the contributing independent variables. These results represent the combined influence of all relevant factors. Based on this, the authors integrate the statistical findings into an epidemiological transmission model. In the classical SEIR model, the transmission rate, denoted as  $\beta$ , represents the contact rate between susceptible and infectious individuals only. In contrast, the modified SEIR model adopted in this study, as proposed by Das et al. [34], incorporates the influence of environmental factors into the transmission dynamics.

$$\begin{aligned} \frac{dS}{dt} &= \mu N - \mu S - \frac{\beta SI}{N}, \\ \frac{dE}{dt} &= \frac{\beta SI}{N} - (\mu + \sigma)E, \\ \frac{dI}{dt} &= \sigma E - (\mu + \gamma)I, \\ \frac{dR}{dt} &= \gamma I - \mu R. \end{aligned} \quad (2)$$

The model divides the total population  $N$ , into four compartments:  $S(t)$  (susceptible),  $E(t)$  (exposed),  $I(t)$  (infectious), and  $R(t)$  (recovered). The parameter  $\mu$  represents the natural birth and death rate,  $\beta$  is the transmission rate,  $\sigma$  is the progression rate from exposed to infectious, and  $\gamma$  is the recovery rate. The model accounts for demographic dynamics and assumes that only individuals in  $I$  contribute to disease transmission.

However, in this case,  $\beta$  will be modified into a function  $\beta_{\text{eff}}$  (the effective transmission rate that incorporates the influence of environmental factors related to housing conditions). Subsequently, simulations will be performed to solve the model based on combinations of these environmental factors, using the fourth-order Runge-Kutta method.

Due to the unavailability of detailed local population data, the subpopulation sizes used in the simulation were chosen for illustrative purposes only. The main objective of the simulation was to demonstrate the influence of environmental factors on TB transmission dynamics, rather than to produce precise epidemiological forecasts.

### 3. RESULTS AND DISCUSSION

#### 3.1 Univariate Analysis

The distribution of research data based on the characteristics of respondents and the environmental conditions of their households in Banjar Regency, Indonesia, is presented in **Table 1**.

**Table 1. Analysis of the Distribution of Research Data**

Variables	Total	Percentace
<b>Ventilation Area (<math>X_1</math>):</b>		
Does Not Meet Standard (< 10% of floor area)	41	56.16%
Meets Standard ( $\geq 10\%$ of floor area)	32	43.84%
<b>Lighting (<math>X_2</math>):</b>		
Does Not Meet Standard (< 60 lux)	37	50.68%
Meets Standard ( $\geq 60$ lux)	36	49.32%
<b>Temperature (<math>X_3</math>):</b>		
Does Not Meet Standard ( $< 18^{\circ}\text{C}$ and $> 30^{\circ}\text{C}$ )	40	54.79%
Meets Standard ( $18^{\circ}\text{C}$ - $30^{\circ}\text{C}$ )	33	45.21%
<b>Humidity(<math>X_4</math>):</b>		
Does Not Meet Standard (<40% and $> 60\%$ )	63	86.30%
Meets Standard (40% - 60%)	10	13.70%
<b>Occupancy Density(<math>X_5</math>):</b>		
Does Not Meet Standard ( $< 9 \text{ m}^2$ )	14	19.18%
Meets Standard ( $\geq 9 \text{ m}^2$ )	59	80.82%
<b>Sex(<math>X_6</math>):</b>		
Female	42	57.53%
Male	31	42.47%
<b>Age(<math>X_7</math>):</b>		
Non-productive (<15 years or $>64$ years)	17	23.29%
Productive (15–64 years)	56	76.71%
<b>Education Level (<math>X_8</math>):</b>		
Basic ( $\leq$ Junior High School)	48	65.75%
Higher ( $\geq$ Senior High School)	25	34.25%
<b>Employment Status (<math>X_9</math>):</b>		
Unemployed	30	41.10%
Employed	43	58.90%
<b>Smoking Status (<math>X_{10}</math>):</b>		
Non-smoker	56	76.71%
Smoker	17	23.29%
<b>Smoking status of family members living in the same household as the respondent (<math>X_{11}</math>):</b>		
None	28	38.36%
Present	45	61.64%
<b>Total</b>	<b>73</b>	<b>100%</b>

### 3.2 Bivariate Analysis

Based on the distribution of respondent characteristics and household environmental conditions presented in **Table 1**, a bivariate analysis was then conducted to examine the association between each variable and pulmonary TB incidence, as shown in **Table 2**.

**Table 2. Bivariate Analysis of Environmental and Host Factors on Pulmonary TB Cases (Chi-Square Test)**

Variable	Pulmonary TB				Total (n = 73)	p-value
	Not		Yes			
	n <sub>1</sub>	%	n <sub>2</sub>	%	n <sub>1</sub> + n <sub>2</sub>	%
<b>Ventilation Area (X<sub>1</sub>):</b>						
Does Not Meet Standard (< 10% of floor area)	10	24.39	31	75.61	41	100
Meets Standard ( $\geq 10\%$ of floor area)	18	56.25	14	43.75	32	100
						<b>0.005*</b>
<b>Lighting (X<sub>2</sub>):</b>						
Does Not Meet Standard (< 60 lux)	14	37.84	23	62.16	37	100
Meets Standard ( $\geq 60$ lux)	14	38.89	22	61.11	36	100
						0.926
<b>Temperature (X<sub>3</sub>):</b>						
Does Not Meet Standard ( $<18^{\circ}\text{C}$ and $>30^{\circ}\text{C}$ )	10	25.00	30	75.00	31	100
Meets Standard ( $18^{\circ}\text{C}$ - $30^{\circ}\text{C}$ )	18	54.55	15	45.45	42	100
						<b>0.010*</b>
<b>Humidity (X<sub>4</sub>):</b>						
Does Not Meet Standard (<40% and > 60%)	25	39.68	38	60.32	63	100
Meets Standard (40% - 60%)	3	30.00	7	70.00	10	100
						0.559
<b>Occupancy Density (X<sub>5</sub>):</b>						
Does Not Meet Standard ( $< 9 \text{ m}^2$ )	6	42.86	8	57.14	14	100
Meets Standard ( $\geq 9 \text{ m}^2$ )	22	37.29	37	62.71	59	100
						0.700
<b>Sex (X<sub>6</sub>):</b>						
Female	21	50.00	21	50.00	42	100
Male	7	22.58	24	77.42	31	100
						<b>0.017*</b>
<b>Age (X<sub>7</sub>):</b>						
Non-Productive	5	29.41	12	70.59	17	100
Productive	23	41.07	33	58.93	56	100
						0.387
<b>Education Level (X<sub>8</sub>):</b>						
Basic ( $\leq$ Junior High School)	17	35.42	31	64.58	48	100
Advanced ( $\geq$ Senior High School)	11	44.00	14	56.00	25	100
						0.474
<b>Employment Status (X<sub>9</sub>):</b>						
Unemployed	10	33.33	20	66.67	30	100
Employed	18	41.86	25	58.14	43	100
						0.461
<b>Smoking Status (X<sub>10</sub>):</b>						
Non-smoker	25	44.64	31	55.36	56	100
Smoker	3	17.65	14	82.35	17	100
						<b>0.045*</b>
<b>Smoking status of family members living in the same household as the respondent (X<sub>11</sub>):</b>						
None	12	42.86	16	57.14	28	100
Present	16	35.56	29	64.44	45	100
						0.533

\*Significance at  $p < 0.05$

Based on the results of the bivariate analysis in **Table 2**, several factors were found to have a significant association with the incidence of pulmonary TB, namely ventilation area ( $p = 0.005$ ), room temperature ( $p = 0.010$ ), sex ( $p = 0.017$ ), and the respondent's smoking status ( $p = 0.045$ ). Respondents living in houses with substandard ventilation, room temperatures outside the 18–30°C range, as well as male and smoking respondents, showed a higher proportion of pulmonary TB cases. Other environmental factors, such as lighting, humidity, and occupancy density, as well as host factors, such as age, education, employment

status, and the smoking habits of family members living in the same household, were not significantly associated with pulmonary TB incidence ( $p > 0.05$ ).

### 3.3 Multivariate Analysis

Multivariate analysis was conducted using a binary logistic regression model to examine the simultaneous relationship between all independent variables and the dependent variable. This study aims to analyze the influence of environmental factors and host factors on the likelihood of pulmonary TB cases. The initial selection of variables based on [Table 2](#), with a significance level of less than 0.25 includes two variables from the environmental factor ( $X_1$  and  $X_3$ ), and two variables from the host factor ( $X_6$  and  $X_{10}$ ).

Based on the results of the Omnibus Tests of Model Coefficients, the  $p$ -value = 0.002 ( $< 0.05$ ) means that the logistic regression model as a whole is significant in predicting the occurrence of pulmonary TB. Next, the results of the Hosmer and Lemeshow test yielded a  $p$ -value of 0.904 ( $> 0.05$ ), meaning the model has a good fit or is consistent with the data. This means the model fits the data (there is no significant difference between the observed and predicted values).

Correlation between the four significant variables was carried out and the result is presented in [Table 3](#).

**Table 3. Correlation Matrix of Independent Variables Correlation Coefficient**

Correlation Between	Correlation Coefficient
Ventilation Area and Temperature	0.286
Ventilation Area and Sex	0.211
Ventilation Area and Smoking Status	-0.142
Temperature and Sex	0.408
Temperature and Smoking Status	-0.281
Sex and Smoking Status	0.547

As presented in [Table 3](#), pairwise correlation values between independent variables were all below the threshold of  $|0.8|$ , indicating the absence of multicollinearity. Therefore, the variables (Ventilation area, Temperature, Sex, and Smoking status) can be appropriately included together in the subsequent regression analysis.

**Table 4. Binary Logistic Regression Test – Phase I**

Variable	B	S.E. (Standar Error)	Wald	Sign	Exp (B)	95% C.I. for Exp (B)	
						Lower	Upper
Ventilation Area (1)	-1.450	0.568	6.509	0.011	0.235	0.077	0.715
Temperature (1)	-1.387	0.558	6.179	0.013	0.250	0.084	0.746
Sex (1)	0.122	0.572	0.046	0.831	1.130	0.369	3.464
Smoking Status (1)	1.187	0.907	1.714	0.190	3.278	0.554	19.388
Constant	1.617	0.599	7.299	0.007	5.038		

Phase I of the binary logistic regression analysis is presented in [Table 4](#). It is evident that out of the four independent variables examined, two variables were statistically significant in influencing the incidence of pulmonary TB, namely ventilation area and temperature. The variable ventilation area had a significance value of 0.011 with an Exp(B) of 0.235, indicating that better ventilation is associated with a 76.5% reduction in the odds of developing pulmonary TB. Similarly, the temperature variable showed a significance value of 0.013 with an Exp(B) of 0.250, meaning that higher temperatures were associated with a 75% reduction in the risk of TB occurrence.

On the other hand, the sex variable ( $p = 0.831$ ) and smoking status ( $p = 0.190$ ) did not demonstrate a statistically significant effect on disease incidence. Although smoking status was not statistically significant ( $p = 0.190$ ), the odds ratio (Exp(B) = 3.278) suggests a potential increase in risk. Moreover, the wide 95% confidence interval (0.554–19.388) reflects a considerable degree of uncertainty in the estimate. Therefore, smoking status was further evaluated in subsequent analysis as a potential confounding variable affecting the relationship between physical environmental factors (such as ventilation and temperature) and pulmonary TB.

An analysis of changes in logistic regression coefficients was conducted to evaluate the potential influence of confounding variables. The formula used to calculate the percentage change is as follows:

$$\text{Percentage change} = \left| \frac{B_{\text{without confounders}} - B_{\text{with confounders}}}{B_{\text{without confounders}}} \right| \times 100\%. \quad (3)$$

A variable is considered a confounder if the coefficient of the main variable changes by  $\geq 10\%$  after including the suspected confounding variable in the model [35]. In this case, smoking status was evaluated as a potential confounder in the relationship between ventilation area, temperature, and pulmonary TB. The percentage change in the B coefficient for ventilation area was 3.26%, and for temperature it was 0.14%, based on the formula presented in Eq. (3). Both values are below the 10% threshold commonly used to identify confounding effects. Therefore, it can be concluded that smoking status does not substantively influence the estimated relationship between the main variables (ventilation area and temperature) and pulmonary TB, and does not indicate the characteristics of a confounder in this model.

**Table 5. Binary Logistic Regression Test – Phase II**

Variabel	B	S.E. (Standard Error)	Wald	Sign	Exp (B)	95% C.I. for Exp (B)	
						Lower	Upper
Ventilation Area (1)	-1.501	0.547	7.520	0.006	0.223	0.076	0.652
Temperature (1)	-1.406	0.547	6.595	0.010	0.245	0.084	0.717
Constant	1.873	0.508	13.579	0.000	6.506		

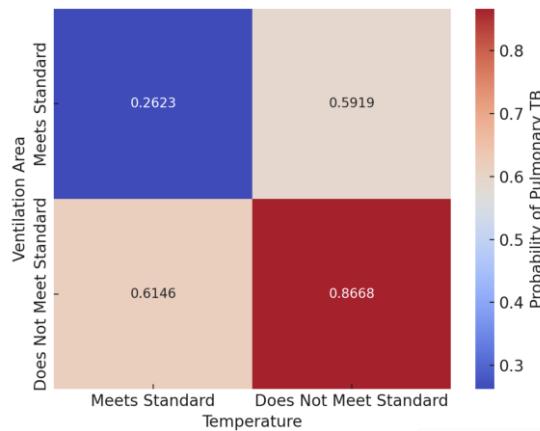
The binary logistic regression model based on Eq. (1) and the significant variables in Table 5 is expressed in the following equation:

$$\ln \left( \frac{\pi}{1 - \pi} \right) = 1.873 - 1.501X_1 - 1.406X_3. \quad (4)$$

Based on Eq. (4), the predicted probability ( $\pi$ ) of an individual having pulmonary TB, according to the categorical variables of ventilation area and temperature, is expressed as follows:

$$\pi = \frac{1}{1 + e^{-(1.873 - 1.501X_1 - 1.406X_3)}} \quad (5)$$

A heatmap visualization is presented to illustrate the probability of pulmonary TB incidence based on combinations of ventilation area and temperature conditions using Eq. (5).



**Figure 1. Pulmonary TB Probability ( $\pi$ ) by Ventilation Area ( $X_1$ ) and Temperature ( $X_3$ ) Conditions**

A heatmap visualization of the probability of pulmonary TB incidence based on the combination of ventilation area and temperature conditions is presented in Fig. 1. The results indicate that the highest probability of TB incidence (0.8668) occurs when both environmental factors (ventilation area and temperature) do not meet health standards. In contrast, the lowest probability (0.2623) is observed when both factors are within recommended standards. Intermediate probabilities are found when only one factor meets the standard: 0.6146 when only temperature is adequate, and 0.5919 when only ventilation is adequate. This pattern highlights the synergistic effect of ventilation and temperature on TB transmission risk, emphasizing that combined improvements in both environmental conditions are more effective in reducing the likelihood of pulmonary TB compared to improvements in either factor alone.

### 3.4 Modified SEIR Model

The SEIR model described in Eq. (2) defines the transmission rate  $\beta$  as the contact rate between susceptible and infectious individuals. In this case, however,  $\beta$  is modified into a function  $\beta_{\text{eff}}$ , which represents an effective transmission rate that incorporates environmental influences. The environmental factors considered in Eq. (4) include ventilation area ( $X_1$ ) and temperature ( $X_3$ ). Taking these factors into account, the modified transmission rate is defined as follows:

$$\beta_{\text{eff}} = c \cdot \pi(X_1, X_3)$$

Where:

$c$  : Represents the baseline transmission rate, i.e., the rate of transmission in the absence of environmental factors. It reflects direct contact between susceptible and infectious individuals and serves as the baseline that is subsequently modified by environmental influences.

$\pi$  : A probability function dependent on the environmental factors under investigation (as described in Eq. (5)).

By incorporating  $\beta_{\text{eff}}$  into Eq. (2), the modified SEIR model is expressed as follows:

$$\begin{aligned} \frac{dS}{dt} &= \mu N - \beta_{\text{eff}} \frac{I}{N} S - \mu S, \\ \frac{dE}{dt} &= \beta_{\text{eff}} \frac{I}{N} S - (\sigma + \mu) E, \\ \frac{dI}{dt} &= \sigma E - (\gamma + \delta + \mu) I, \\ \frac{dR}{dt} &= \gamma I - \mu R, \end{aligned} \quad (6)$$

where  $\delta$  represents the mortality rate due to pulmonary tuberculosis.

#### 3.4.1 Equilibrium Point and Basic Reproduction Number

The equilibrium points of the model are obtained by solving Eq. (6) simultaneously, by substituting all time derivatives to zero  $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$  [36], [37]. This procedure yields two types of equilibrium points: the disease-free equilibrium (DFE), which represents a state where no infection exists in the population, and the endemic equilibrium (EE), which reflects a condition in which the infection persists in the population at a certain level. These equilibrium points are denoted respectively by  $T_1 = (S^*, E^*, I^*, R^*) = (N, 0, 0, 0)$  and  $T_2 = (\hat{S}, \hat{E}, \hat{I}, \hat{R})$ , where  $\hat{S} = \frac{N(\sigma+\mu)(\gamma+\delta+\mu)}{\beta_{\text{eff}} \sigma}$ ,  $\hat{E} = \frac{(\gamma+\delta+\mu)}{\sigma} \hat{I}$ ,  $\hat{I} = \frac{\mu(N-\hat{S})N}{\beta_{\text{eff}} \hat{S}}$ ,  $\hat{R} = \frac{\gamma}{\mu} \hat{I}$ .

One of the key measures commonly used in mathematical epidemiology is the basic reproduction number, denoted as  $R_0$ . This value represents the average number of new cases generated by a single infected individual in a fully susceptible population. Therefore, determining the value of  $R_0$  is essential for analyzing disease dynamics and evaluating control strategies. In this SEIR model,  $R_0$  is calculated using the Next Generation Matrix method [38], [39]. For this,  $\mathbf{x} = (E, I)^T$ . From Eq. (6), we can write

$$\frac{d\mathbf{x}}{dt} = \begin{pmatrix} \frac{dE}{dt} \\ \frac{dI}{dt} \end{pmatrix} = \mathbf{F} - \mathbf{V}, \text{ with } \mathbf{F} = \begin{pmatrix} \mathcal{F}_1 \\ \mathcal{F}_2 \end{pmatrix} = \begin{pmatrix} \beta_{\text{eff}} \frac{I}{N} S \\ 0 \end{pmatrix} \text{ and } \mathbf{V} = \begin{pmatrix} \mathcal{V}_1 \\ \mathcal{V}_2 \end{pmatrix} = \begin{pmatrix} (\sigma + \mu)E \\ -\sigma E + (\gamma + \delta + \mu)I \end{pmatrix}$$

By considering the Jacobian of  $\mathbf{F}$  and  $\mathbf{V}$  at DFE ( $T_1$ ), we get

$$\mathbf{F} = \begin{pmatrix} \frac{\partial \mathcal{F}_1}{\partial E} & \frac{\partial \mathcal{F}_1}{\partial I} \\ \frac{\partial \mathcal{F}_2}{\partial E} & \frac{\partial \mathcal{F}_2}{\partial I} \end{pmatrix} = \begin{pmatrix} 0 & \beta_{\text{eff}} \\ 0 & 0 \end{pmatrix} \text{ and } \mathbf{V} = \begin{pmatrix} \frac{\partial \mathcal{V}_1}{\partial A} & \frac{\partial \mathcal{V}_1}{\partial B} \\ \frac{\partial \mathcal{V}_2}{\partial A} & \frac{\partial \mathcal{V}_2}{\partial B} \end{pmatrix} = \begin{pmatrix} (\sigma + \mu) & 0 \\ -\sigma & (\gamma + \delta + \mu) \end{pmatrix}$$

It is to be noted that the threshold parameter  $R_0$  is the dominant eigenvalue of  $\mathbf{G} = \mathbf{F}\mathbf{V}^{-1} = \begin{pmatrix} \beta_{\text{eff}} \sigma & \beta_{\text{eff}} \\ \frac{(\sigma+\mu)(\gamma+\delta+\mu)}{N} & (\gamma+\delta+\mu) \end{pmatrix}$  that is  $R_0 = \rho(\mathbf{G})$ , which gives

$$R_0 = \frac{\beta_{\text{eff}} \sigma}{(\sigma + \mu)(\gamma + \delta + \mu)}. \quad (7)$$

Subsequently, the endemic equilibrium (EE) can be expressed as follows.

$$T_2 = (\hat{S}, \hat{E}, \hat{I}, \hat{R}) = \left( \frac{N}{R_0}, \frac{\mu N (\gamma + \delta + \mu) (R_0 - 1)}{\beta_{\text{eff}} \sigma}, \frac{\mu N (R_0 - 1)}{\beta_{\text{eff}}}, \frac{N \gamma (R_0 - 1)}{\beta_{\text{eff}}} \right).$$

### 3.4.2 Stability Analysis of the Equilibrium Points

Using the linearization method, the local behavior of Eq. (6) around the equilibrium is investigated. The Jacobian matrix is obtained as follows:

$$J = \begin{pmatrix} -\beta_{\text{eff}} \frac{I}{N} - \mu & 0 & -\beta_{\text{eff}} \frac{S}{N} & 0 \\ \beta_{\text{eff}} \frac{I}{N} & -(\sigma + \mu) & \beta_{\text{eff}} \frac{S}{N} & 0 \\ 0 & \sigma & -(\gamma + \delta + \mu) & 0 \\ 0 & 0 & \gamma & -\mu \end{pmatrix}$$

#### Stability of Disease Free Equilibrium (DFE)

**Theorem 1.** *The disease-free equilibrium  $T_1$  for System in Eq. (6) is locally asymptotically stable for  $R_0 < 1$  and unstable for  $R_0 > 1$ .*

**Proof.** The Jacobian matrix is evaluated at the DFE  $T_1 = (N, 0, 0, 0)$  as follows

$$J_{T_1} = \begin{pmatrix} -\mu & 0 & -\beta_{\text{eff}} & 0 \\ 0 & -(\sigma + \mu) & \beta_{\text{eff}} & 0 \\ 0 & \sigma & -(\gamma + \delta + \mu) & 0 \\ 0 & 0 & \gamma & -\mu \end{pmatrix}$$

The characteristic equation corresponding to  $J_{T_1}$  is given as

$$\begin{aligned} |J_{T_1} - \lambda I| &= 0 \\ \begin{vmatrix} -\mu - \lambda & 0 & -\beta_{\text{eff}} & 0 \\ 0 & -(\sigma + \mu) - \lambda & \beta_{\text{eff}} & 0 \\ 0 & \sigma & -(\gamma + \delta + \mu) - \lambda & 0 \\ 0 & 0 & \gamma & -\mu - \lambda \end{vmatrix} &= 0 \\ (-\mu - \lambda)^2 [(-(\sigma + \mu) - \lambda)(-(\gamma + \delta + \mu) - \lambda) - \beta_{\text{eff}} \sigma] &= 0 \end{aligned}$$

This implies that  $\lambda_{1,2} = -\mu$  and the other two eigenvalues correspond to the roots of the following polynomial equation

$$a_0 \lambda^2 + a_1 \lambda + a_2 = 0 \quad (8)$$

Where:

$$a_0 = 1$$

$$a_1 = (\sigma + \mu) + (\gamma + \delta + \mu) = \sigma + \gamma + \delta + 2\mu > 0$$

$$a_2 = (\sigma + \mu)(\gamma + \delta + \mu) - \beta_{\text{eff}} \sigma = (\sigma + \mu)(\gamma + \delta + \mu)(1 - R_0) > 0, \text{ if } R_0 < 1$$

From Eq. (8), the following Routh-Hurwitz matrix [40] can be constructed

$$H = \begin{pmatrix} a_1 & 0 \\ a_0 & a_2 \end{pmatrix} = \begin{pmatrix} \sigma + \gamma + \delta + 2\mu & 0 \\ 1 & (\sigma + \mu)(\gamma + \delta + \mu)(1 - R_0) \end{pmatrix}.$$

Based on the Routh-Hurwitz criterion,  $\Delta_1 = a_1 > 0$  and  $\Delta_2 = a_1 a_2 > 0$  then all roots of Eq. (8) have negative real parts. Therefore, the DFE  $T_1$  is locally asymptotically stable when  $R_0 < 1$ , and becomes unstable once  $R_0 > 1$ . ■

### Stability of Endemic Equilibrium (EE)

**Theorem 2.** The endemic equilibrium  $T_2$  for System in Eq. (6) is locally asymptotically stable for  $R_0 > 1$  and unstable for  $R_0 < 1$ .

**Proof.** The Jacobian matrix is evaluated at the EE  $T_2 = (\hat{S}, \hat{E}, \hat{I}, \hat{R})$  as follows

$$J_{T_2} = \begin{pmatrix} -\beta_{\text{eff}} \frac{\hat{I}}{N} - \mu & 0 & -\beta_{\text{eff}} \frac{\hat{S}}{N} & 0 \\ \beta_{\text{eff}} \frac{\hat{I}}{N} & -(\sigma + \mu) & \beta_{\text{eff}} \frac{\hat{S}}{N} & 0 \\ 0 & \sigma & -(\gamma + \delta + \mu) & 0 \\ 0 & 0 & \gamma & -\mu \end{pmatrix}$$

The characteristic equation corresponding to  $J_{T_2}$  is given as

$$\begin{vmatrix} J_{T_2} - \lambda I & \\ \begin{array}{cccc} -\beta_{\text{eff}} \frac{\hat{I}}{N} - \mu - \lambda & 0 & -\beta_{\text{eff}} \frac{\hat{S}}{N} & 0 \\ \beta_{\text{eff}} \frac{\hat{I}}{N} & -(\sigma + \mu) - \lambda & \beta_{\text{eff}} \frac{\hat{S}}{N} & 0 \\ 0 & \sigma & -(\gamma + \delta + \mu) - \lambda & 0 \\ 0 & 0 & \gamma & -\mu - \lambda \end{array} \end{vmatrix} = 0$$

$$(-\mu - \lambda)[a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3] = 0$$

This implies that  $\lambda_1 = -\mu$  and the other eigenvalues correspond to the roots of the following polynomial equation

$$a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0. \quad (9)$$

Where:

$$a_0 = 1 > 0$$

$$a_1 = \left( \beta_{\text{eff}} \frac{\hat{I}}{N} + \mu \right) + (\sigma + \mu) + (\gamma + \delta + \mu) = \mu(R_0 - 1) + 3\mu + \sigma + \gamma + \delta > 0, \text{ if } R_0 > 1$$

$$a_2 = (\mu(R_0 - 1) + \mu)((\sigma + \mu) + (\gamma + \delta + \mu)) > 0, \text{ if } R_0 > 1$$

$$a_3 = (\mu(R_0 - 1))(\sigma + \mu)(\gamma + \delta + \mu) > 0, \text{ if } R_0 > 1$$

From Eq. (9), the following Routh-Hurwitz matrix [40] can be constructed

$$H = \begin{pmatrix} a_1 & a_3 & a_5 \\ a_0 & a_2 & a_4 \\ 0 & a_1 & a_3 \end{pmatrix} = \begin{pmatrix} a_1 & a_3 & 0 \\ 1 & a_2 & 0 \\ 0 & a_1 & a_3 \end{pmatrix}$$

Based on the Routh-Hurwitz criterion, because  $\Delta_1 = a_1 > 0$ ,  $\Delta_2 = a_1a_2 - a_3 > 0$  and  $\Delta_3 = a_3(\Delta_2) > 0$ , then all roots of Eq. (9) have negative real parts. Therefore, the EE  $T_2$  is locally asymptotically stable when  $R_0 > 1$ , and becomes unstable once  $R_0 < 1$ . ■

### 3.5 Numerical Simulation

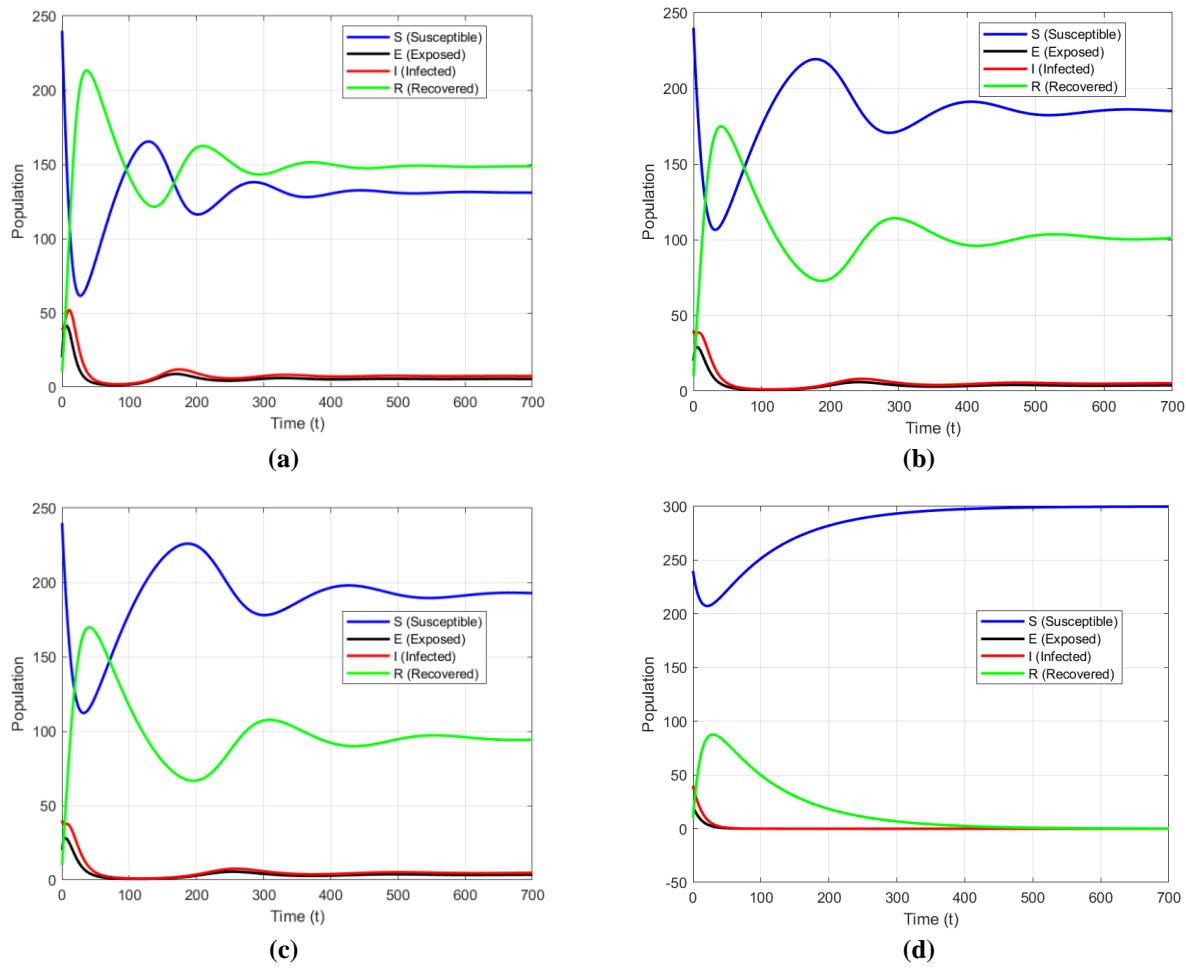
Numerical simulations are conducted using the RK4 method with parameter and initial values listed in Table 6.

**Table 6.** The Parameter Values and Initial Condition of the Model

Parameters and Initial Condition	Values	Source
$\pi$	0.8668; 0.5919; 0.6146; 0.2623	Based on the results of the multivariate analysis in Fig. 1.
$c$	0.6	assumed

Parameters and Initial Condition	Values	Source
$\delta$	0.01	assumed
$\gamma$	0.2	assumed
$\mu$	$\frac{1}{68.26} \approx 0.01$	Based on life expectancy in Banjar Regency in 2024 [41]
$\sigma$	0.3	assumed
$S(0)$	230	assumed
$E(0)$	40	assumed
$I(0)$	20	assumed
$R(0)$	10	assumed

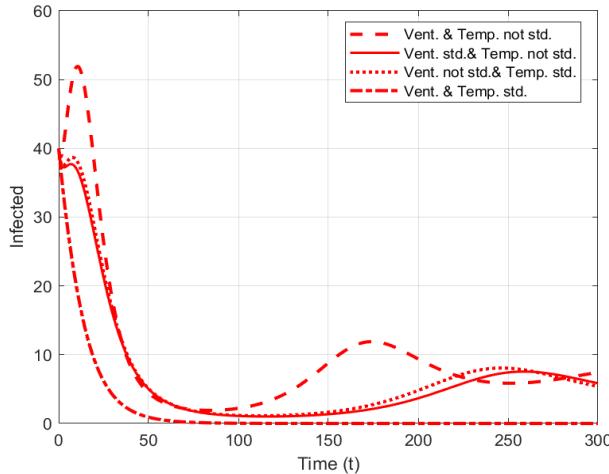
As an illustration, the simulation results of the SEIR model solution (Eq. (6) and Table 6) are presented in four figures, each representing a combination of ventilation area and temperature. These are used to illustrate the population dynamics of  $S$  (Susceptible),  $E$  (Exposed),  $I$  (Infectious), and  $R$  (Recovered).



**Figure 2. Simulation of the SEIR Model of Pulmonary TB Transmission Influenced by Environmental Factors:** (a) Ventilation Area Does Not Meet The Standard, Temperature Does Not Meet The Standard,  $R_0 > 1$ , (b) Ventilation Area Meets The Standard, Temperature Does Not Meet The Standard,  $R_0 > 1$ , (c) Ventilation Area Does Not Meet The Standard, Temperature Meets The Standard,  $R_0 > 1$ , and (d) Ventilation Area Meets The Standard, Temperature Meets The Standard,  $R_0 < 1$

The simulation results in Fig. 2 show a comparison of the SEIR model dynamics for four environmental scenarios based on ventilation and room temperature feasibility. The first three scenarios (a), (b), and (c), which each represent a combination of non-optimal ventilation and temperature conditions, all result in the value of the basic reproduction number  $R_0 > 1$ , so the system does not go to the DFE point. This is reflected by the number of infected individuals  $I(t)$  remaining above zero in the long run, indicating local stability at the endemic point. Scenario (a), where ventilation and temperature both do not meet the standard, shows the highest infection rate. When only one of the environmental factors is improved, both ventilation (b) and

temperature (c) decrease in infection, but not enough to stabilise the system at the DFE. In contrast, in scenario (d), where ventilation and temperature both meet the standard, the value of  $R_0$  becomes less than 1, and infection decreases dramatically until it disappears completely, indicating that the DFE point becomes asymptotically stable. This finding is in line with the stability theory of dynamical systems, where DFE stability is only achieved when  $R_0 < 1$ , and confirms that simultaneous environmental interventions are much more effective in controlling pulmonary TB transmission.



**Figure 3. Comparison of Four Scenarios Based on Transmission Rate Variations**

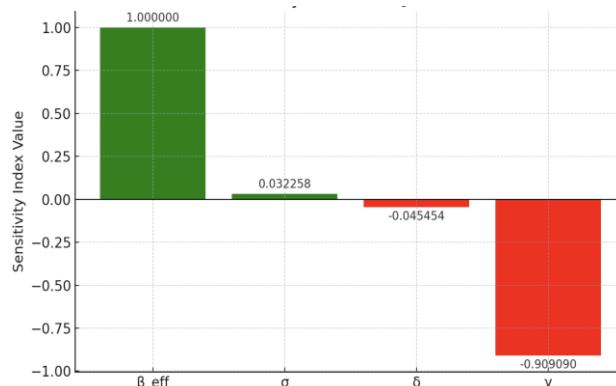
A comparison of the number of infected individuals in the four scenarios based on variations in ventilation and room temperature compliance is shown in Fig. 3. The curves show that the scenario with ventilation and temperature that meet the standards results in the fastest decline in infection towards zero, reflecting a condition with  $R_0 < 1$ . In contrast, scenarios with environmental conditions that do not meet the standards show a slow decline and repeated fluctuations in infection, indicating stability at the endemic point ( $R_0 > 1$ ). This confirms that simultaneous environmental interventions are instrumental in reducing the transmission rate and steering the system towards an EE point.

### 3.6 Sensitivity Analysis

This section presents a sensitivity analysis to identify parameters that affect the basic reproduction number (Eq. (7)). The analysis process is carried out by calculating the sensitivity index value of each parameter that forms the basic reproduction number. The normalised sensitivity index [42], [43] of the basic reproduction number is defined as follows:

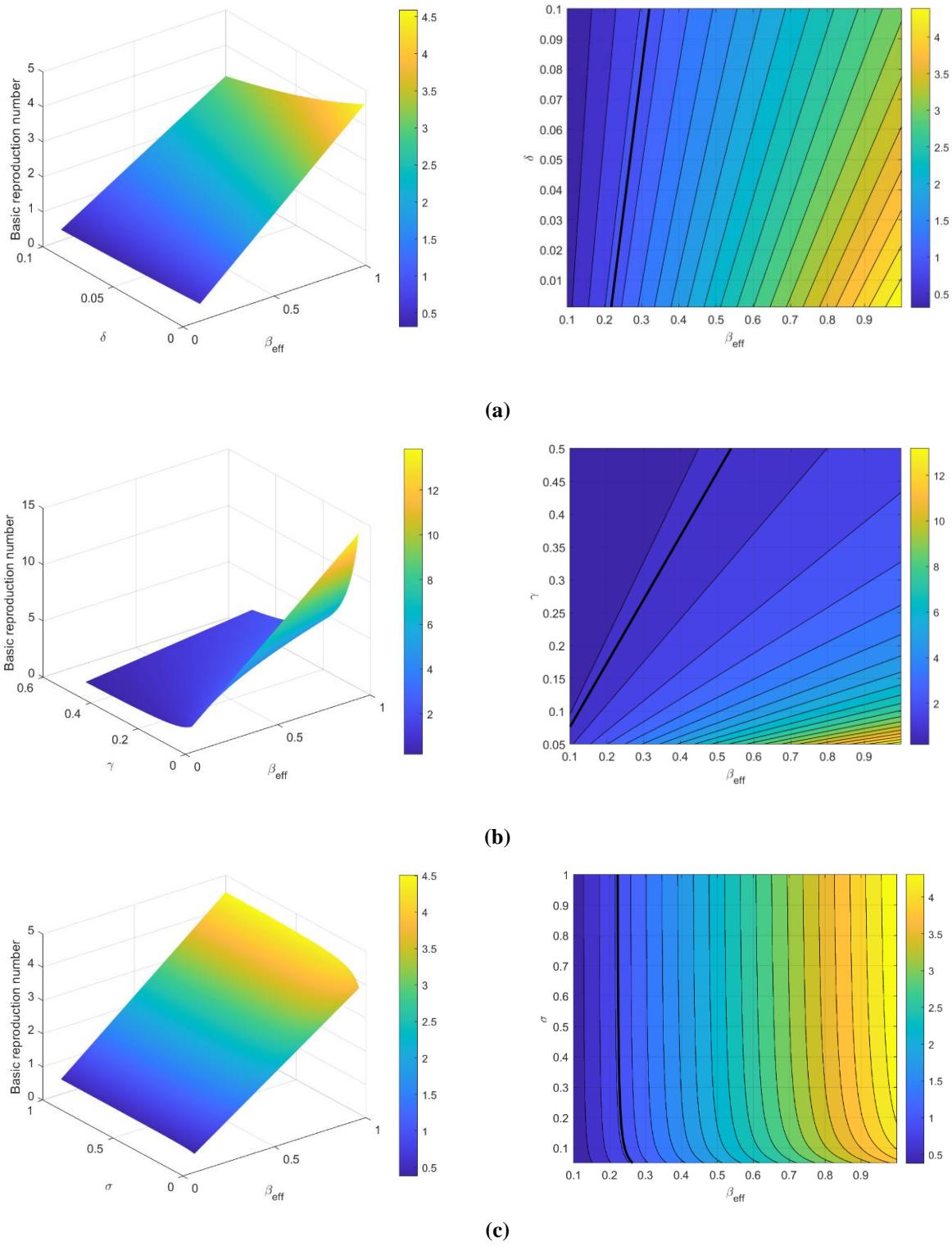
$$\Gamma_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}.$$

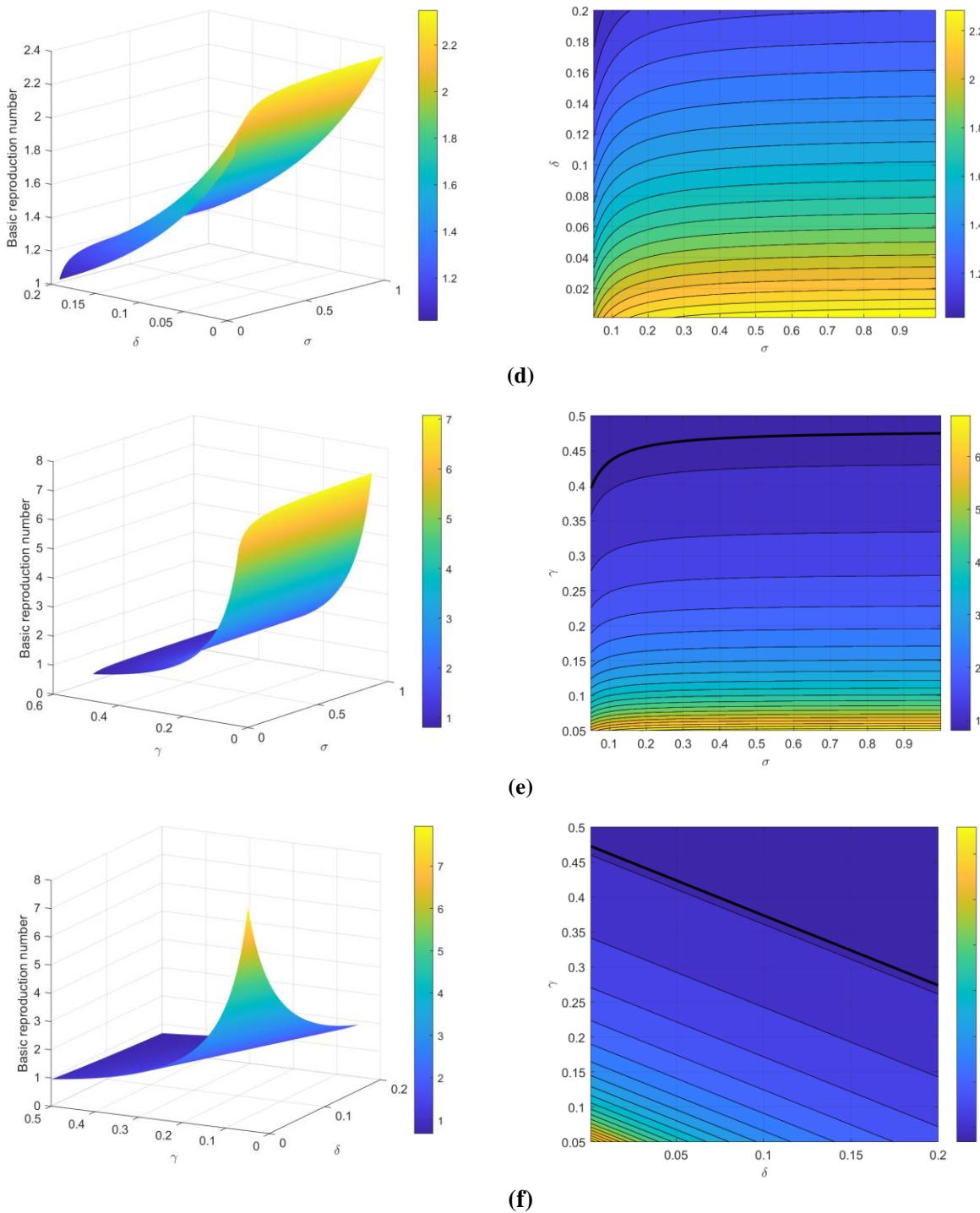
The sensitivity index values of the basic reproduction number, based on the parameter values presented in Table 6, are as follows:



**Figure 4. Sensitivity Index of  $R_0$  with Respect to Model Parameters**

A bar chart of the normalized sensitivity index values of the basic reproduction number with respect to four key model parameters is shown in Fig. 4. The analysis reveals that  $\beta_{\text{eff}}$  has the highest positive sensitivity index (1.000000), indicating that small changes in this parameter have a direct and proportional effect on  $R_0$ . In contrast,  $\gamma$  shows the most significant negative influence (-0.909090), suggesting that increasing the recovery rate substantially reduces  $R_0$ . The parameters  $\sigma$  and  $\delta$  contribute less, with sensitivity indices of 0.032258 and -0.045454, respectively, implying a relatively minor impact. This chart emphasizes that control strategies aiming to reduce  $\beta_{\text{eff}}$  and  $\gamma$  enhance are the most effective in suppressing disease transmission.





**Figure 5.** Simulation of the Surface and Contour Plots of the Basic Reproduction Number as a Function of Various Combinations of Model Parameters: (a)  $R_0$  with respect to  $\beta_{\text{eff}}$  and  $\delta$ , (b)  $R_0$  with Respect to  $\beta_{\text{eff}}$  and  $\gamma$ , (c)  $R_0$  with Respect to  $\beta_{\text{eff}}$  and  $\sigma$ , (d)  $R_0$  with Respect to  $\delta$  and  $\sigma$ , (e)  $R_0$  with Respect to  $\gamma$  and  $\sigma$ , (f)  $R_0$  with Respect to  $\gamma$  and  $\delta$

The surface and contour plots of the basic reproduction number as a function of various combinations of model parameters is illustrated in Fig. 5. Subfigures (a)–(f) demonstrate how changes in two parameters at a time affect  $R_0$ . In general,  $R_0$  increases with larger values of the effective contact rate  $\beta_{\text{eff}}$  and decreases with higher values of the recovery rate  $\gamma$  or disease-induced death rate  $\delta$ . The steepest gradients appear when  $\gamma$  or  $\delta$  are small, showing a nonlinear and sensitive response in these regions. The interaction between  $\sigma$  and the other parameters also contributes to modulating the threshold value of  $R_0$ , confirming that both the exposure rate and removal mechanisms play a key role in disease control. These visualizations highlight the most influential parameters in reducing transmission, particularly through increasing  $\gamma$  or reducing  $\beta_{\text{eff}}$ .

#### 4. CONCLUSION

This study confirms that the physical residential environment, particularly the ventilation area and room temperature, is an important component in understanding the dynamics of pulmonary tuberculosis transmission in Banjar District, Indonesia. By integrating the logistic regression approach and the SEIR model, this study makes a significant contribution to the development of a more realistic and contextualised epidemiological model, in accordance with local environmental conditions. Based on the simulation results, the most effective strategy to reduce the number of pulmonary TB patients is to reduce the effective transmission rate or increase the treatment rate. The effective transmission rate in this study was analysed through four scenarios based on residential environmental conditions. These findings highlight the importance of policy transformation in spatial planning and housing design that supports environmental health. Neighbourhood-based interventions are not only complementary but also a strategic foundation that must be fully integrated into sustainable pulmonary TB prevention and control efforts.

#### Author Contributions

Yuni Yulida: Conceptualization, Methodology, Investigation, Writing Original Draft, Data Curation, Formal Analysis. Eko Suhartono: Conceptualization, Supervision, Writing Original Draft, Writing Review, and Editing. Dewi Anggraini: Methodology, Visualization, Writing Review, and Editing. Syamsul Arifin: Methodology, Formal Analysis, Writing Review, and Editing. All authors discussed the results and contributed to the final manuscript.

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#### Declarations

The authors declare no competing interests.

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