Zero : Jurnal Sains, Matematika, dan Terapan
E-ISSN : 2580-5754; P-ISSN : 2580-569X
Volume 10, Number 1, 2026
DOI: 10.30829/zero.v10i1.28823
Page: 379-391

Transmission Dynamics Model of Scabies in Islamic Boarding Schools

[1] David Prasetyo
Mathematics, Diponegoro University, Semarang, Indonesia

[2] Sunarsih
Mathematics, Diponegoro University, Semarang, Indonesia

[3] Sutrisno
Mathematics, Diponegoro University, Semarang, Indonesia

Article info

Article history:

Accepted 15 May, 2026

Keywords:

Basic reproduction number;
Mathematical epidemiology;
Mathematical model;
Scabies transmission dynamics;
Stability analysis.

ABSTRACT

Scabies continues to pose a major public health challenge in densely populated congregate settings such as Islamic boarding schools (*pesantren*) in Indonesia. This study develops a stage structured S-I_E-I_L-R compartmental model (Susceptible, Early-stage Infected, Late-stage Infected, Recovered) that explicitly accounts for differences in mite burden, infectivity, and recovery rates between early and late-stage infections. Using qualitative analysis of the nonlinear differential equations, the basic reproduction number (R_0) was derived via the Next Generation Matrix method, and stability analysis was performed. Results show that the diseasefree equilibrium is locally and globally asymptotically stable when $R_0 < 1$. Under baseline parameters, $R_0 \approx 3.99$, with late-stage infections accounting for approximately 88.3% of total transmission. Due to the model's assumptions of a closed population and permanent immunity, no biologically meaningful endemic equilibrium exists; once the infection runs its course, the disease eventually dies out as susceptibles are exhausted. Sensitivity analysis highlights that reducing the late-stage transmission rate (β_2) and increasing the early-stage recovery rate (γ_1) are the most effective intervention targets. This model provides a rigorous theoretical framework to guide evidencebased scabies control strategies in high-density residential institutions.

Corresponding Author:

David Prasetyo
Mathematics, Diponegoro University
Email: davidp101288@gmail.com

1. INTRODUCTION

Scabies remains a major global public health problem. The World Health Organization estimates an annual incidence of 200–300 million cases worldwide [1]. This parasitic skin disease, caused by the mite Sarcoptes scabiei var. hominis, causes intense itching and skin lesions. It also damages the skin barrier and increases the risk of secondary bacterial infections, which may lead to serious complications such as acute glomerulonephritis and rheumatic heart disease [2]. In 2017, the WHO classified scabies as a Neglected Tropical Disease, prompting greater efforts to develop effective control strategies.

Islamic boarding schools (*pesantren*) in Indonesia represent typical high-risk environments. Students live together, share sleeping areas, and have frequent skin to skin contact. Limited access to clean water and poor hygiene practices further increase transmission risk [3]. Recent studies report high prevalence rates in Indonesian pesantren, ranging from 25% to over 70% [4]. For example, Wahdini et al. (2025) found a prevalence of 43.8% among students in a traditional urban Islamic boarding school in Jakarta, with poor bedding cleanliness as the only significant risk factor (odds ratio = 2.009) [5]. Similar high rates were documented in Jember Regency (46.07% and 25.81%) [6] and other pesantren where sharing personal items strongly predicted greater disease severity (odds ratio = 9.06) [7].

Mathematical compartmental models are a standard tool for analyzing infectious disease dynamics and designing interventions [8]. Most existing models treat the infected population as a single homogeneous compartment. They do not distinguish between early-stage and late-stage infections, despite clear differences in mite burden, infectivity, detectability, and recovery time. Late-stage infections, particularly crusted scabies, involve high mite loads and act as superspreaders [9]. Recovery from late-stage disease is also slower and more difficult [10]. In high-density residential settings where individuals maintain a relatively fixed number of close contacts, frequency-dependent transmission is more appropriate than density dependent transmission.

No previous modeling study has specifically addressed stage structured scabies transmission in the Indonesian pesantren context. While recent models have incorporated temporal delays [11], stochastic variability [12], and agent based heterogeneity [13], none explicitly partition the infected population into early-stage and late-stage compartments with differential transmission rates, nor focus on the high-density, closed population setting of Islamic boarding schools. This study fills that gap by developing a novel S-I_E-I_L-R compartmental model. The infected population is divided into early-stage (I_E) and late-stage (I_L) compartments with differential transmission rates (β_1 for I_E and β_2 for IL, where $\beta_2 > \beta_1$), variable recovery rates (γ_1 and γ_2), and a progression rate from early to late stage (α). The study's four objectives are: (1) formulate and parameterize a deterministic stage-structured model for high-density residential institutions; (2) derive R_0 and establish stability conditions for the diseasefree equilibrium; (3) perform sensitivity analysis to identify key transmission parameters; and (4) translate theoretical results into practical, stage specific intervention strategies.

2. RESEARCH METHODS

2.1 Study Design and Scope

This is a purely theoretical mathematical modeling study. No primary field data were collected and no calibration to observed outbreak data was performed. The model is a theoretical framework inspired by the epidemiological context of pesantren rather than a data driven reconstruction of any specific outbreak. Assumptions of a closed population, frequency dependent transmission, and permanent immunity are idealizations that permit rigorous analytical treatment. Future work should incorporate field data, demographic turnover, and stochastic effects to enhance realism and predictive validity.

2.2 Model Structure and Assumptions

The population is partitioned into four mutually exclusive compartments: Susceptible (*S*), Early-stage Infected (I_E), Late-stage Infected (I_L), and Recovered (R). Susceptible individuals acquire infection through frequency dependent contact at rates β_1 (from I_E) and β_2 (from I_L), where $\beta_2 > \beta_1$, reflecting higher mite burden in late-stage disease. Early-stage individuals either progress to late-stage at rate α or recover directly at rate γ_1. Late-stage individuals recover at rate γ_2. Recovery confers permanent immunity, making R an absorbing compartment. The population is closed (no births, deaths, or migration), and environmental transmission is excluded, so total population N is constant throughout ($dN/dt = 0$).

An important structural implication is that no biologically meaningful endemic equilibrium exists under these assumptions. Setting all derivatives to zero with $I_E > 0$ forces $S^* = 0$, which is epidemiologically trivial. Therefore, the primary analytical focus is on the diseasefree equilibrium and R_0, which governs whether the disease can invade a fully susceptible population.

2.3 Parameterization

All parameter values are drawn from published epidemiological and clinical scabies literature. Table 1 summarizes each parameter with its biological meaning, baseline value, unit, sensitivity range, and source.

Table 1. Model Parameter Values, Ranges, and Sources

Param.	Description	Baseline	Unit	Range	Justification	Source
β_1	Transmission rate, early-stage (I_E)	0.10	day^{-1}	0.05–0.20	Low mite burden; moderate infectivity during incubation	[14]
β_2	Transmission rate, late-stage (I_L), incl. crusted cases	0.35	day^{-1}	0.20–0.50	High mite burden; crusted scabies superspreader potential	[14]; [9]

Param.	Description	Baseline	Unit	Range	Justification	Source
α	Progression rate from I_E to I_L	0.071	day^{-1}	0.05–0.10	Early stage 2 weeks; delayed treatment extends duration	[14]; [15]
γ_1	Recovery rate, early-stage (I_E) to R	0.143	day^{-1}	0.10–0.20	Prompt treatment resolves early scabies in 7 days	[16]; [2]
γ_2	Recovery rate, late-stage (I_L) to R	0.033	day^{-1}	0.02–0.05	Late/crusted scabies requires 1 month treatment	[16]; [1]

3. RESULT AND ANALYSIS

This section presents the analytical results of the S-I_E-I_L-R model: (1) model formulation and fundamental mathematical properties; (2) disease-free equilibrium and R_0 derivation via the Next Generation Matrix method; (3) local and global stability of the DFE; (4) discussion of the endemic equilibrium under this model's structural constraints; and (5) sensitivity analysis of R_0. All results are purely analytical.

3.1 Model Formulation and Fundamental Mathematical Properties

This research will discuss an S-I_E-I_L-R epidemiological model with the following compartments: individuals who are initially susceptible (S), individuals who are infected and begin to show symptoms, characterized by itching and rashes, categorized as Early-stage infection (I_E), individuals who are infected and develop pus and severe sores due to scratching, categorized as Late-stage Infection (I_L), and individuals in the recovery phase or recovered (R). Thus, the total population is:

$$N(t) = S(t) + I_E(t) + I_L(t) + R(t) \tag{1}$$

The parameters used are: β_1 represents the effective contact rate for transmission from I_E, β_2 represents the effective contact rate for transmission from I_E, α represents the progression rate from I_E to I_L, γ_1 represents the recovery rate from I_E directly to R, and γ_2 represents the recovery rate from I_L to R.

The assumptions used are: The population is closed, meaning there is no migration into or out of the population, scabies transmission only occurs from student to student, and environmental factors are disregarded.

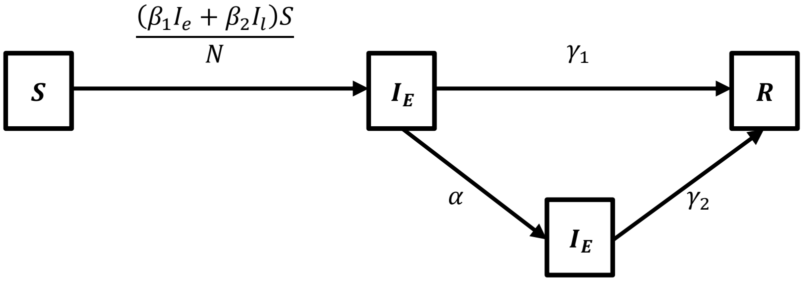

Figure 1. Diagram of compartmen models S-I_E-I_L-R

Table 2. Description of The Parameters Used in The Model

Parameters	Description
S(t)	Susceptible individuals
I_E(t)	Early-stage infected individuals
I_L(t)	Late-stage infected individuals
R(t)	Recovered individuals
β_1	Effective contact rate for transmission from I_E
β_2	Effective contact rate for transmission from I_L
A	Progression rate from I_E to I_L
$\gamma 1$	Recovery rate from I_E directly to R
$\gamma 2$	Recovery rate from I_L to R

Assume the total population $N = S + I_E + I_L + R$. The system of ordinary differential equations (ODEs) describing the model is:

$$\begin{aligned} \frac{dS}{dt} &= -\frac{(\beta_1 I_E+\beta_1 I_L)}{N}S, \\ \frac{dI_E}{dt} &= \frac{(\beta_1 I_E+\beta_1 I_L)}{N}S - \alpha I_E - \gamma_1 I_E, \\ \frac{dI_L}{dt} &= \alpha I_E - \gamma_2 I_L, \\ \frac{dR}{dt} &= \gamma_1 I_E + \gamma_2 I_L, \end{aligned} \tag{2}$$

Nonlinear systems of differential equations have a fundamental domain $S(0) > 0, I_E(0) > 0, I_E(0) > 0$, and *R(0)>0.*

$$N(t) = S(t) + I_E(t) + I_L(t) + R(t) \tag{3}$$

Assume $s = \frac{S}{N}$, $i_e = \frac{I_E}{N}$, $i_l = \frac{I_L}{N}$ and $r = \frac{R}{N}$ each representing the fraction of the susceptible, early-stage infection, late-stage infection, and recovered classes in the population. Thus, we obtain equation (3),

$$s(t) + i_e(t) + i_l(t) + r(t) = 1 \tag{4}$$

Equation (2) is simplified to become:

$$\begin{aligned} \frac{ds}{dt} &= -(\beta_1 i_e + \beta_1 i_l)s, \\ \frac{di_e}{dt} &= (\beta_1 i_e + \beta_1 i_l)s - \alpha i_e - \gamma_1 i_e, \\ \frac{di_l}{dt} &= \alpha i_e - \gamma_2 i_l, \\ \frac{dr}{dt} &= \gamma_1 i_e + \gamma_2 i_l \end{aligned} \tag{5}$$

With the main constraint

$$s(0) = s_0 > 0, i_e(0) = i_{e_0} > 0, i_l(0) = i_{l_0} > 0, r(0) = r_0 > 0.$$

3.2 Positivity and Boundedness of Solutions

Theorem 3.1 (Positivity). *If the initial conditions satisfy* $S(0) > 0,\ I_E(0) > 0,\ I_L(0) \geq 0,$ *and* $R(0) \geq 0,$ *then the solutions* $S(t), I_E(t), I_L(t),$ *and* $R(t)$ *of the system (1) remain non negative for all time* $t > 0$.

Proof. We prove the positivity for each compartment by demonstrating that the rate of change becomes non negative as the compartment value approaches zero, thus preventing it from crossing into negative values.

Suspectible Compartment (*S*):

The dynamics of S are given by:

$$\frac{dS}{dt} = -\frac{\beta_1 I_E+\beta_2 I_L}{N}S.$$

This can be viewed as a first order linear differential equation:

$$\frac{dS}{dt} + \lambda(t)S = 0, \text{where } \lambda(t) = \frac{\beta_1 I_E+\beta_2 I_L}{N} \geq 0.$$

The solution is obtained using the integrating factor method. The integrating factor is $\mu(t) = \exp\left(\int_0^t \lambda(\tau)\ d\tau\right)$. Multiplying through:

$$\frac{d}{dt}(S \cdot \mu(t)) = 0.$$

Integrating both sides with respect to t:

$$S(t) \cdot \mu(t) = S(0).$$

Therefore,

$$S(t) = S(0)\exp\left(-\int_0^t \lambda(\tau)\ d\tau\right). \tag{6}$$

Since $S(0) > 0$ and the exponential function is always positive, $S(t) > 0$ for all $t > 0$.

Early-Stage Infected Compartment (I_E):

The dynamics are:

$$\frac{dI_E}{dt} = \frac{\beta_1 I_E+\beta_2 I_L}{N}S - (\alpha + \gamma_1)I_E.$$

This is a non homogeneous linear differential equation. We rewrite it as:

$$\frac{dI_E}{dt} + (\alpha + \gamma_1)I_E = \frac{\beta_1 I_E + \beta_2 I_L}{N} S.$$

The integrating factor is $\mu_E(t) = \exp\left((\alpha + \gamma_1)t\right)$. Thus,

$$\frac{d}{dt}(I_E \cdot \mu_E(t)) = \frac{\beta_1 I_E + \beta_2 I_L}{N} S \cdot \mu_E(t).$$

Integrating from 0 to t:

$$I_E(t)\mu_E(t) - I_E(0) = \int_0^t \frac{\beta_1 I_E(\tau) + \beta_2 I_L(\tau)}{N} S(\tau)\mu_E(\tau)\, d\tau.$$

Therefore,

$$I_E(t) = I_E(0)\exp\left(-(\alpha + \gamma_1)t\right) + \exp\left(-(\alpha + \gamma_1)t\right) \int_0^t \exp\left((\alpha\gamma_1)\tau\right) \frac{\beta_1 I_E(\tau) + \beta_2 I_L(\tau)}{N} S(\tau)\, d\tau. \quad (7)$$

The first term is positive given $I_E(0) > 0$. The integrand $\frac{\beta_1 I_E + \beta_2 I_L}{N} S$ is non negative for all τ (as $S(\tau) > 0$ from above, and $I_E, I_L \geq 0$ by the nature of the population). Therefore, the entire second term is non negative. Hence, $I_E(t) \geq 0$ for all $t > 0$.

Late-Stage Infected Compartment (I_L):

The dynamics are:

$$\frac{dI_L}{dt} = \alpha I_E - \gamma_2 I_L.$$

This is also a non homogeneous linear equation:

$$\frac{dI_L}{dt} + \gamma_2 I_L = \alpha I_E.$$

The integrating factor s $\mu_L(t) = \exp(\gamma_2 t)$. Then,

$$\frac{d}{dt}(I_L \cdot \mu_L(t)) = \alpha I_E \mu_L(t).$$

Integrating:

$$I_L(t)\mu_L(t) - I_L(0) = \int_0^t \alpha I_E(\tau)\mu_L(\tau)\, d\tau.$$

So,

$$I_L(t) = I_L(0)\exp(-\gamma_2 t) + \exp(-\gamma_2 t) \int_0^t \alpha I_E(\tau)\exp(\gamma_2 \tau)\, d\tau. \quad (8)$$

Given $I_L(0) \geq 0$ and $I_E(\tau) \geq 0$, it follows that $I_L(t) \geq 0$ for all $t > 0$.

Recovered Compartment (R):

The dynamics are:

$$\frac{dR}{dt} = \gamma_1 I_E + \gamma_2 I_L.$$

This is a direct integral:

$$R(t) = R(0) + \int_0^t (\gamma_1 I_E(\tau) + \gamma_2 I_L(\tau))\, d\tau. \quad (9)$$

Since $R(0) \geq 0$ and the integrand is non negative, $R(t) \geq 0$ for all $t > 0$.

Having shown that each compartment remains non negative, we conclude that the system preserves the positivity of solutions.

Theorem 3.2 (Boundedness of Solutions). *The feasible region* $\Omega = \{(S, I_E, I_L, R) \in \mathbb{R}_+^4 : S + I_E + I_L + R = N\}$ *is positively invariant for the system (1). That is, every solution with initial conditions in* Ω *remains in* Ω *for all* $t > 0$.

Proof. To prove boundedness, we consider the time derivative of the total population N:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI_E}{dt} + \frac{dI_L}{dt} + \frac{dR}{dt}.$$

Substituting the expressions from the system (2):

$$\frac{dN}{dt} = \left[-\frac{\beta_1 I_E + \beta_2 I_L}{N} S\right] + \left[\frac{\beta_1 I_E + \beta_2 I_L}{N} S - (\alpha + \gamma_1)I_E\right] + [\alpha I_E - \gamma_2 I_L] + [\gamma_1 I_E + \gamma_2 I_L]$$
$$= 0.$$

This shows that the total population N is constant. Since N is constant and all state variables are non negative (from Theorem 3.1), it follows that:

$$0 \leq S(t), I_E(t), I_L(t), R(t) \leq N \text{ for all } t \geq 0.$$

Therefore, all solutions are bounded and remain within the biologically feasible region Ω.

3.3 Equilibrium Analysis and The Basic Reproduction Number (R_0)

A fundamental question in epidemiology is the longterm behavior of a disease: will it fade out or persist? This is determined by analyzing the equilibrium points of the system and a key threshold parameter known as the basic reproduction number, R_0.

3.3.1 Disease Free Equilibrium (DFE)

The DiseaseFree Equilibrium (DFE) is the state where the infection is entirely absent from the population. To find it, we set the time derivatives to zero and the infected compartments to zero:

$$\frac{dS}{dt} = \frac{dI_E}{dt} = \frac{dI_L}{dt} = \frac{dR}{dt} = 0, \text{with } I_E = 0, I_L = 0.$$

From $\frac{dI_E}{dt} = 0$ and $I_E = I_L = 0$, the equation is satisfied. From $\frac{dS}{dt} = 0$, we get $0 = 0$. The recovered compartment, in the absence of infection, will also be zero. Thus, the entire population remains susceptible. The DFE is:

$$E_0 = (S^*, I_E^*, I_L^*, R^*) = (N, 0,0,0).$$

In the normalized form, the DFE is $(s^*, i_e^*, i_l^*, r^*) = (1,0,0,0)$.

3.3.2 Remark on the Endemic Equilibrium

A biologically meaningful endemic equilibriumone in which susceptible, infected, and recovered individuals coexist at constant positive levels indefinitelydoes not exist under the current model assumptions. This is a direct structural consequence of two features: (i) the population is closed with no demographic renewal (births or immigration), and (ii) recovery confers permanent immunity. Together, these imply that the recovered compartment R grows monotonically until all infections cease. Formally, setting all derivatives of system (2) to zero with $I_E > 0$ forces $S^* = 0$, leaving no susceptible individuals to sustain transmission. Consequently, no non trivial endemic equilibrium exists in the full model, and the diseasefree equilibrium $E_0 = (N, 0, 0, 0)$ is the only equilibrium of the complete system.

This is a known and mathematically expected property of SIR type models with closed populations and permanent immunity: the system describes a single epidemic wave that eventually exhausts susceptibles, after which the infection dies out. The quantity R_0 governs whether the disease can initially invade ($R_0 > 1$) and determines the scale of the epidemic wave, not its persistence. The analysis in Sections 3.3 through 3.5 accordingly focuses on the diseasefree equilibrium, the invasion threshold, and sensitivity analysis of R_0 which are the epidemiologically meaningful quantities in this model. If the model were extended to include demographic turnover (births, deaths) or waning immunity, a genuine endemic equilibrium with $S^* > 0$ could emerge, and its stability would then be an appropriate subject of analysis.

3.4 Derivation of the Basic Reproduction Number (R_0)

The basic reproduction number, R_0, is defined as the average number of secondary infections produced by one typical infected individual in a completely susceptible population. We compute R_0 using the Next Generation Matrix (NGM) method, a standardized approach in mathematical epidemiology.

The NGM method involves dentifying the *nfected compartments* (here, I_E and I_L), and linearizing the system around the DFE. We define two matrices, F and V, where:

F is the matrix of new infection terms.

V is the matrix of transition terms between and out of the infected compartments.

The next generation matrix is $K = FV^{-1}$, and R_0 is the spectral radius (the largest eigenvalue) of K.

For our system, the infected compartments are I_E and I_L. The equations for these compartments from system (2) are:

$$\begin{aligned} \frac{dI_E}{dt} &= \frac{\beta_1 I_E + \beta_2 I_L}{N} S - (\alpha + \gamma_1) I_E, \\ \frac{dI_L}{dt} &= \alpha I_E - \gamma_2 I_L. \end{aligned}$$

At the DFE, $S = N$. We separate the terms that correspond to new nfections $(\mathcal{F})$ from other transitions $(\mathcal{V})$.

$$\frac{dI_E}{dt} = \underbrace{\beta_1 I_E + \beta_2 I_L}_{\mathcal{F}_1} + \underbrace{0}_{\mathcal{V}_1^-} - \underbrace{(\alpha+\gamma_1) I_E}_{\mathcal{V}_1^+},$$

$$\frac{dI_L}{dt} = \underbrace{0}_{\mathcal{F}_2} + \underbrace{\alpha I_E}_{\mathcal{V}_2^-} - \underbrace{\gamma_2 I_L}_{\mathcal{V}_2^+}.$$

More precisely, we define:

$$\mathcal{F} = \begin{bmatrix} \beta_1 I_E + \beta_2 I_L \\ 0 \end{bmatrix}, \mathcal{V} = \begin{bmatrix} (\alpha+\gamma_1) I_E \\ -\alpha I_E + \gamma_2 I_L \end{bmatrix}.$$

The Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ with respect to the infected compartments (I_E, I_L), evaluated at the DFE, are:

$$F = \begin{bmatrix} \beta_1 & \beta_2 \\ 0 & 0 \end{bmatrix}, V = \begin{bmatrix} \alpha+\gamma_1 & 0 \\ -\alpha & \gamma_2 \end{bmatrix}.$$

The next generation matrix s $K = FV^{-1}$. First, we find V^{-1}:

$$V^{-1} = \frac{1}{(\alpha+\gamma_1)\gamma_2} \begin{bmatrix} \gamma_2 & 0 \\ \alpha & \alpha+\gamma_1 \end{bmatrix} = \begin{bmatrix} \frac{1}{\alpha+\gamma_1} & 0 \\ \frac{\alpha}{(\alpha+\gamma_1)\gamma_2} & \frac{1}{\gamma_2} \end{bmatrix}.$$

Now, compute $K = FV^{-1}$:

$$K = \begin{bmatrix} \beta_1 & \beta_2 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\alpha+\gamma_1} & 0 \\ \frac{\alpha}{(\alpha+\gamma_1)\gamma_2} & \frac{1}{\gamma_2} \end{bmatrix} = \begin{bmatrix} \frac{\beta_1}{\alpha+\gamma_1} + \frac{\beta_2 \alpha}{(\alpha+\gamma_1)\gamma_2} & \frac{\beta_2}{\gamma_2} \\ 0 & 0 \end{bmatrix}.$$

Simplifying the (1,1) element:

$$K_{11} = \frac{\beta_1 \gamma_2}{(\alpha+\gamma_1)\gamma_2} + \frac{\beta_2 \alpha}{(\alpha+\gamma_1)\gamma_2} = \frac{\beta_1\gamma_2 + \beta_2\alpha}{(\alpha+\gamma_1)\gamma_2}.$$

Thus,

$$K = \begin{bmatrix} \frac{\beta_1\gamma_2+\beta_2\alpha}{(\alpha+\gamma_1)\gamma_2} & \frac{\beta_2}{\gamma_2} \\ 0 & 0 \end{bmatrix}.$$

The eigenvalues of K are the solutions to $\det(K - \lambda I) = 0$:

$$\det \begin{bmatrix} \frac{\beta_1\gamma_2+\beta_2\alpha}{(\alpha+\gamma_1)\gamma_2} - \lambda & \frac{\beta_2}{\gamma_2} \\ 0 & -\lambda \end{bmatrix} = 0.$$

This gives:

$$\left(\frac{\beta_1\gamma_2+\beta_2\alpha}{(\alpha+\gamma_1)\gamma_2} - \lambda\right)(-\lambda) = 0.$$

The eigenvalues are $\lambda_1 = \frac{\beta_1\gamma_2+\beta_2\alpha}{(\alpha+\gamma_1)\gamma_2}$ and $\lambda_2 = 0$. The spectral radius, and hence the basic reproduction number, is:

$$R_0 = \frac{\beta_1\gamma_2+\beta_2\alpha}{(\alpha+\gamma_1)\gamma_2}.$$

The relative contribution of late-stage infections to R_0 can be quantified as $\frac{\beta_2\alpha}{(\alpha+\gamma_1)\gamma_2} / R_0 = \frac{\beta_2\alpha}{\beta_1\gamma_2+\beta_2\alpha}$. With the baseline parameter estimates from Table 1 ($\beta_2 = 0.35$, $\alpha = 0.071$, $\beta_1 = 0.10$, $\gamma_2 = 0.033$), this fraction evaluates to $\beta_2\alpha / (\beta_2\alpha + \beta_1\gamma_2) = (0.35 \times 0.071) / (0.35 \times 0.071 + 0.10 \times 0.033) = 0.02485 / 0.02815 \approx 0.883$, indicating that approximately 88.3% of all transmission is attributable to individuals in the late stage. This underscores their role as epidemic drivers and identifies them as the most effective targets for intervention.

3.5 Stability Analysis of Equilibrium Points

3.5.1 Local Stability of the Diseasefree Equilibrium

Theorem 3.3 *The diseasefree equilibrium* $E_0 = (N, 0,0,0)$ *is locally asymptotically stable if* $R_0 < 1$*, and unstable if* $R_0 > 1$.

Proof. We analyze the stability of the DFE by linearizing the system around it and examining the eigenvalues of the resulting Jacobian matrix. The Jacobian matrix J of the system (2) is given by:

$$J = \begin{bmatrix} -\frac{\beta_1 I_E + \beta_2 I_L}{N} & -\frac{\beta_1 S}{N} & -\frac{\beta_2 S}{N} & 0 \\ \frac{\beta_1 I_E + \beta_2 I_L}{N} & \frac{\beta_1 S}{N} - (\alpha + \gamma_1) & \frac{\beta_2 S}{N} & 0 \\ 0 & \alpha & -\gamma_2 & 0 \\ 0 & \gamma_1 & \gamma_2 & 0 \end{bmatrix}.$$

Evaluating this at the DFE $E_0 = (N, 0,0,0)$, where $S = N, I_E = 0, I_L = 0$, we get:

$$J(E_0) = \begin{bmatrix} 0 & -\beta_1 & -\beta_2 & 0 \\ 0 & \beta_1 - (\alpha + \gamma_1) & \beta_2 & 0 \\ 0 & \alpha & -\gamma_2 & 0 \\ 0 & \gamma_1 & \gamma_2 & 0 \end{bmatrix}.$$

The eigenvalues λ satisfy the characteristic equation $\det(J(E_0) - \lambda I) = 0$:

$$\det \begin{bmatrix} -\lambda & -\beta_1 & -\beta_2 & 0 \\ 0 & \beta_1 - (\alpha + \gamma_1) - \lambda & \beta_2 & 0 \\ 0 & \alpha & -\gamma_2 - \lambda & 0 \\ 0 & \gamma_1 & \gamma_2 & -\lambda \end{bmatrix} = 0.$$

This is a block lower triangular matrix. The eigenvalues are the roots of the determinants of the diagonal blocks. One block is $[-\lambda]$ (from the 4th row/column), giving $\lambda_1 = 0$. Another block is from the 1st row/column, but careful: the first row and column are not independent. A better approach is to note that the first and fourth equations are decoupled from the nfection dynamics at the DFE for the purpose of stability. The stability is determined by the infected subsystem (I_E, I_L). The Jacobian for the infected subsystem at DFE is:

$$J_{\text{inf}}(E_0) = \begin{bmatrix} \beta_1 - (\alpha + \gamma_1) & \beta_2 \\ \alpha & -\gamma_2 \end{bmatrix}.$$

The characteristic equation for this 2x2 matrix is:

$$\det \begin{bmatrix} \beta_1 - (\alpha + \gamma_1) - \lambda & \beta_2 \\ \alpha & -\gamma_2 - \lambda \end{bmatrix} = 0.$$

This expands to:

$$[\beta_1 - (\alpha + \gamma_1) - \lambda](-\gamma_2 - \lambda) - \beta_2 \alpha = 0.$$
$$-[\beta_1 - (\alpha + \gamma_1) - \lambda](\gamma_2 + \lambda) - \beta_2 \alpha = 0.$$

Multiply by -1:

$$[\beta_1 - (\alpha + \gamma_1) - \lambda](\gamma_2 + \lambda) + \beta_2 \alpha = 0.$$

Let's write it as:

$$(\lambda - (\beta_1 - (\alpha + \gamma_1)))(\lambda + \gamma_2) + \beta_2 \alpha = 0.$$

This is a quadratic: $\lambda^2 + a_1 \lambda + a_0 = 0$, where:

$$\begin{aligned} a_1 &= \gamma_2 + (\alpha + \gamma_1 - \beta_1), \\ a_0 &= (\alpha + \gamma_1 - \beta_1)\gamma_2 + \beta_2 \alpha = \gamma_2(\alpha + \gamma_1) - \beta_1 \gamma_2 + \beta_2 \alpha. \end{aligned}$$

Note that $a_0 = \gamma_2(\alpha + \gamma_1)(1 - \frac{\beta_1 \gamma_2 + \beta_2 \alpha}{\gamma_2(\alpha + \gamma_1)}) = \gamma_2(\alpha + \gamma_1)(1 - R_0)$.

By the Routh Hurwitz stability criterion for a quadratic, the roots have negative real parts if and only if $a_1 > 0$ and $a_0 > 0$.

- $a_0 > 0$ if and only if $1 - R_0 > 0$, i.e., $R_0 < 1$.
- $a_1 = \gamma_2 + (\alpha + \gamma_1 - \beta_1)$. For biologically realistic parameters, $\alpha + \gamma_1$ (the rate of leaving the early stage) is typically larger than the transmission rate β_1, so $a_1 > 0$.

Therefore, if $R_0 < 1$, both eigenvalues of the infected subsystem have negative real parts. The zero eigenvalues from the S and R directions correspond to neutral stability along the manifold of equilibria (since total N is constant), but the infection will die out. Thus, the DFE is locally asymptotically stable when $R_0 < 1$. f $R_0 > 1$, then $a_0 < 0$, implying at least one positive eigenvalue, and the DFE is unstable.

3.5.2 Global Stability of the Diseasefree Equilibrium

Local stability guarantees that the disease will die out only if the initial number of infected individuals is very small. Global stability ensures that regardless of the initial number of infected individuals, the disease will eventually vanish, provided $R_0 \leq 1$.

Theorem 3.4 *If* $R_0 \leq 1$*, the diseasefree equilibrium* E_0 *is globally asymptotically stable in the feasible region* Ω.
Proof. We prove this by constructing a Lyapunov function, a scalar function that measures the distance from the equilibrium. Consider the following Lyapunov function candidate:

$$L(I_E, I_L) = aI_E + bI_L,$$

where a and b are positive constants to be determined. This function is positive definite in the infected compartments and is zero at the DFE.

We compute the time derivative of L along the trajectories of the system:

$$\frac{dL}{dt} = a\frac{dI_E}{dt} + b\frac{dI_L}{dt}.$$

Substituting the equations for $\frac{dI_E}{dt}$ and $\frac{dI_L}{dt}$:

$$\frac{dL}{dt} = a(\frac{\beta_1 I_E + \beta_2 I_L}{N}S - (\alpha + \gamma_1)I_E) + b(\alpha I_E - \gamma_2 I_L).$$

Since $S \leq N$, we have $\frac{S}{N} \leq 1$. Thus,

$$\frac{dL}{dt} \leq a(\beta_1 I_E + \beta_2 I_L - (\alpha + \gamma_1)I_E) + b(\alpha I_E - \gamma_2 I_L).$$

Rearranging terms:

$$\frac{dL}{dt} \leq (a\beta_1 - a(\alpha + \gamma_1) + b\alpha)I_E + (a\beta_2 - b\gamma_2)I_L.$$

We now choose a and b to make the coefficients non positive. Let's choose b such that the coefficient of I_L s zero:

$$a\beta_2 - b\gamma_2 = 0 \Rightarrow b = \frac{a\beta_2}{\gamma_2}.$$

Substitute b into the coefficient of I_E:

$$\begin{aligned} &a\beta_1 - a(\alpha + \gamma_1) + (\frac{a\beta_2}{\gamma_2})\alpha \\ &= a(\beta_1 - (\alpha + \gamma_1) + \frac{\beta_2\alpha}{\gamma_2}) \\ &= a(\frac{\beta_1\gamma_2}{\gamma_2} - \frac{(\alpha+\gamma_1)\gamma_2}{\gamma_2} + \frac{\beta_2\alpha}{\gamma_2}) \\ &= \frac{a}{\gamma_2}(\beta_1\gamma_2 + \beta_2\alpha - (\alpha + \gamma_1)\gamma_2) \\ &= \frac{a}{\gamma_2}(\alpha + \gamma_1)\gamma_2(\frac{\beta_1\gamma_2 + \beta_2\alpha}{(\alpha+\gamma_1)\gamma_2} - 1) \\ &= a(\alpha + \gamma_1)(R_0 - 1). \end{aligned}$$

Therefore, with our choice of b, we have:

$$\frac{dL}{dt} \leq a(\alpha + \gamma_1)(R_0 - 1)I_E.$$

If $R_0 \leq 1$, then $\frac{dL}{dt} \leq 0$. Furthermore, $\frac{dL}{dt} = 0$ if and only if $I_E = 0$. When $I_E = 0$, from the equation for I_L, $\frac{dI_L}{dt} = -\gamma_2 I_L$, so $I_L \to 0$. By LaSalle's Invariance Principle, all solutions in Ω approach the largest invariant set where $I_E = I_L = 0$, which is the DFE. Hence, the DFE is globally asymptotically stable when $R_0 \leq 1$.

The Lyapunov function chosen in Theorem 3.4 is constructed as a weighted linear combination of the early and late-stage infected compartments, $V = a_1 I_E + a_2 I_L$. Biologically, this function represents a weighted measure of the total infection burden present in the population. When $R_0 < 1$, the time derivative of V is negative definite, indicating that the overall infection burden strictly decreases over time until it reaches zero, irrespective of the initial number of infected students. This behavior intuitively shows that the disease cannot maintain itself in the pesantren and will eventually die out naturally once R_0 is brought below unity.

The absence of a biologically meaningful endemic equilibrium in this model carries important epidemiological implications for scabies outbreaks in Islamic boarding schools. In a closed population with permanent immunity, the disease is mathematically self-terminating in the sense that it exhausts the supply of susceptibles — not in the sense of being benign or harmless. When $R_0 > 1$, the model predicts a large epidemic wave that severely affects the population before susceptibles are depleted. This distinction is epidemiologically critical: self-termination through susceptible exhaustion is not equivalent to a mild or controlled outbreak. Therefore, the only effective way to prevent or control a major outbreak is to drive R_0 below 1 through targeted interventions, particularly those focusing on late-stage cases.

3.5.3 Local Stability of the Diseasefree Equilibrium: Numerical Verification

Theorem 3.3 established analytically that the DFE is locally asymptotically stable when $R_0 < 1$. To verify this concretely, we evaluate the infected subsystem Jacobian $J_{inf}(E_0)$ at the Table 1 baseline values ($\beta_1 = 0.10, \beta_2 = 0.35, \alpha = 0.071, \gamma_1 = 0.143, \gamma_2 = 0.033$). At these values, $R_0 = (0.10 \times 0.033 + 0.35 \times 0.071) / (0.214 \times 0.033) = (0.0033 + 0.02485) / 0.007062 \approx 3.97 > 1$, so the DFE is unstable at baseline. To verify the stability criterion analytically for a hypothetical intervention scenario where $R_0 < 1$, suppose β_2 is reduced to 0.05 (aggressive treatment of late-stage cases). Then $R_0 = (0.10 \times 0.033 + 0.05 \times 0.071) / (0.214 \times 0.033) \approx 0.62 < 1$. The Routh-Hurwitz coefficients are $a_1 = \gamma_2 + (\alpha + \gamma_1 - \beta_1) = 0.033 + (0.214 - 0.10) = 0.147 > 0$; $a_0 = \gamma_2(\alpha + \gamma_1)(1 - R_0) = 0.033 \times 0.214 \times (1 - 0.62) = 0.00269 > 0$. Both Routh-Hurwitz conditions are satisfied, confirming local asymptotic stability of the DFE when $R_0 < 1$, consistent with Theorem 3.3.

3.6 Sensitivity Analysis of R_0

To identify the parameters that most strongly influence disease transmission, we perform a sensitivity analysis on the basic reproduction number R_0. We employ the normalized forward sensitivity index, as defined by Chitnis et al. [21]. For a differentiable variable u that depends on a parameter p, the normalized forward sensitivity index is given by

$$\Upsilon_u^p = \frac{\partial u}{\partial p} \cdot \frac{p}{u}.$$

This index measures the relative change in $u$resulting from a relative change in p. A positive value indicates that an increase in the parameter increases R_0, while a negative value indicates the opposite. The magnitude reflects the degree of influence. Applied to R_0 with respect to a parameter p, the normalized forward sensitivity index takes the form $\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}$.

Recall that

$$R_0 = \frac{\beta_1 \gamma_2 + \beta_2 \alpha}{(\alpha + \gamma_1)\gamma_2}.$$

Using the baseline parameter values ($\beta_1 = 0.10$, $\beta_2 = 0.35$, $\alpha = 0.071$, $\gamma_1 = 0.143$, $\gamma_2 = 0.033$), we compute the sensitivity indices for each parameter. The results are presented in Table 3.

Table 3. Normalized forward sensitivity indices of R_0 with respect to model parameters at baseline values. Index defined as $Y^pR_0 = (\partial R_0/\partial p) \times (p/R_0)$

Parameter	Sensitivity Index ($\Upsilon_{R_0}^p$)	Interpretation
β_2	+0.883	Highest positive influence
β_1	+0.117	Moderate positive influence
α	+0.766	Strong positive influence
γ_1	-0.766	Strong negative influence
γ_2	-0.117	Moderate negative influence

The sensitivity analysis reveals that R_0 is most sensitive to the late-stage transmission rate β_2 $(\Upsilon = +0.883)$. A 10% reduction in β_2 would decrease R_0 by approximately 8.83%. The progression rate $\alpha$and early-stage recovery rate γ_1 also show high sensitivity (in opposite directions). In contrast, β_1 and γ_2 have relatively smaller effects.

These findings confirm that interventions aimed at reducing late-stage transmission (such as prompt isolation and treatment of crusted cases) and accelerating early-stage recovery (through active screening and immediate treatment) are the most effective strategies for bringing R_0 below the threshold value of 1 and controlling scabies outbreaks in Islamic boarding schools.

Several model limitations should be acknowledged explicitly. First, the closed-population assumption excludes demographic turnover: in a real pesantren, students enroll and graduate over time, and births or migration could sustain a pool of susceptibles, potentially enabling genuine endemic persistence. Second, permanent immunity is a strong simplification for scabies. Clinical evidence shows that immunity wanes and reinfestation is common, particularly in institutional settings; incorporating waning immunity or reinfection dynamics would likely raise the effective reproduction number and could produce endemic equilibria absent here. Third, environmental transmission via contaminated bedding, clothing, or shared surfaces is excluded, even though fomite-mediated spread contributes meaningfully to scabies persistence in close-quarters environments. Fourth, the model is deterministic and homogeneous, ignoring stochastic extinction at low infection levels and individual heterogeneity in contact rates. Future extensions incorporating demographic renewal, waning immunity, environmental reservoirs, and stochastic effects would substantially increase the model's realism and predictive validity for informing public health policy in Indonesian Islamic boarding schools.

4. CONCLUSION

This study develops a stage structured S-I_E-I_L-R compartmental model (Susceptible, Early-stage Infected, Late-stage Infected, Recovered) to analyze the transmission dynamics of scabies in high density congregate settings, particularly Islamic boarding schools (*pesantren*) in Indonesia. The model explicitly incorporates differential mite burden and infectivity between early- and late-stage infections, along with frequency dependent transmission appropriate for closed residential environments. Qualitative analysis shows that the diseasefree equilibrium is locally and globally asymptotically stable when the basic reproduction number $R_0 < 1$. Under baseline parameters derived from the literature, $R_0 > 1$, indicating the potential for significant epidemic spread in the absence of intervention. Sensitivity analysis identifies the late-stage transmission rate (β_2) and early-stage recovery rate (γ_1) as the most influential parameters. Interventions targeting prompt detection, isolation, and treatment of late-stage cases (including crusted scabies) combined with active screening to accelerate early recovery offer the greatest potential to reduce R_0 below the epidemic threshold. Due to the model's assumptions of a closed population and permanent immunity, the system describes a single epidemic wave that eventually exhausts the pool of susceptibles, with no biologically meaningful endemic equilibrium. These idealizations facilitate analytical tractability while highlighting the selflimiting nature of outbreaks under current assumptions; however, they also underscore the need for extensions that incorporate demographic turnover, waning immunity, and environmental transmission to better reflect real world *pesantren* settings. Overall, the model provides a rigorous theoretical framework for understanding stage specific transmission dynamics of scabies and supports evidence based, resource efficient control strategies in high-risk institutional environments. Future research should validate and refine parameter estimates through field studies in Indonesian *pesantren*, integrate stochastic effects or optimal control, and evaluate the cost effectiveness of targeted interventions focusing on late-stage cases.

ACKNOWLEDGEMENT

I would like to express my sincere gratitude to my advisors in the Master of Mathematics program at Diponegoro University for their invaluable guidance, and to my family and colleagues at Tazakka Islamic Boarding Schools for their unwavering support throughout this research. I hope these findings can contribute to scabies prevention efforts in Islamic boarding schools.

5. REFERENCES

[1] L. Romani *et al.*, "Mass Drug Administration for Scabies Control in a Population with Endemic Disease," *N. Engl. J. Med.*, vol. 373, pp. 2305–2313, Dec. 2015, doi: 10.1056/nejmoa1500987.

[2] D. Engelman *et al.*, "The public health control of scabies: priorities for research and action," *Lancet*, vol. 394, no. 10192, pp. 81–92, Jul. 2019, doi: 10.1016/S0140-6736(19)31136-5.

[3] S. Sukmawati, H. Ahmad, F. Islam, and F. Akbar, "Risk Factors for Scabies Incidence Among Students at Islamic Boarding Schools in Mamuju Regency, Indonesia," *J. Med. Heal.*, vol. 7, no. 2, pp. 163–171, Aug. 2025, doi: 10.28932/jmh.v7i2.9245.

[4] P. Mawardi *et al.*, "Scabies Risk Factor Analysis in Students at Islamic Boarding School," *Berk. Ilmu Kesehat. Kulit dan Kelamin*, vol. 36, pp. 168–173, 2024.

[5] S. Wahdini, S. Sungkar, S. Widaty, F. Nurcandra, and I. P. Sari, "Scabies and related factors among students in a traditional urban Islamic boarding school," *Discov. Public Heal.*, vol. 22, no. 1, p. 754, Nov. 2025, doi: 10.1186/s12982-025-01175-4.

[6] K. Ali, M. Rizaldi, and S. Putri, "Environmental Sanitation and Scabies Incidence among Santri in Islamic Boarding School in Jember Regency," *Insights Public Heal. J.*, vol. 5, Nov. 2024, doi: 10.20884/1.iphj.2024.5.2.13462.

[7] J. Jauhari, S. Mulyani, and V. Widyaningsih, "Factors Influencing Scabies among Islamic Boarding School Students: A Scoping Review," in *BIO Web of Conferences*, EDP Sciences, Nov. 2025. doi: 10.1051/bioconf/202519300042.

[8] R. M. Anderson and R. M. May, *Infectious Diseases of Humans: Dynamics and Control*. Oxford University Press, 1991. doi: 10.1093/oso/9780198545996.001.0001.

[9] P. Nhek, M. Seng, and S. Pol, "A Narrative Review on Crusted Scabies: Pathogenesis, Clinical Manifestations, Diagnosis, and Treatment Options," *J. Cambodian Heal.*, vol. 3, no. 1, Dec. 2025, doi: 10.70243/jch2025.3105.

[10] T. Wang *et al.*, "Proteomic analysis of Sarcoptes scabiei reveals that proteins differentially expressed between eggs and female adult stages are involved predominantly in genetic information processing, metabolism and/or host-parasite interactions," *PLoS Negl. Trop. Dis.*, vol. 16, no. 12, p. e0010946, 2022.

[11] E. Fadhal *et al.*, "Dynamical analysis of scabies delayed epidemic model with second-order global stability," *PLoS One*, vol. 20, no. 4, p. e0319095, Apr. 2025, doi: 10.1371/journal.pone.0319095.

[12] J. Shah, H. Khan, E. A. A. Ismail, F. A. Awaad, and A. Kumar, "Modeling scabies transmission dynamics: a stochastic approach with spectral collocation and neural network insights," *Eur. Phys. J. Plus*, vol. 140, no. 1, p. 66, Jan. 2025, doi: 10.1140/epjp/s13360-025-06025-5.

[13] K. E. C. Ainslie, M. Hooiveld, and J. Wallinga, "Estimation of the epidemiological characteristics of scabies," *Nat. Commun.*, vol. 16, no. 1, p. 10524, Nov. 2025, doi: 10.1038/s41467-025-65544-y.

[14] M. Vink *et al.*, "Exploring transmission dynamics of the Sarcoptes scabiei mite in humans by combining molecular typing and epidemiological variables, the Netherlands 2016–2023," *Parasit. Vectors*, vol. 17, no. 1, p. 419, Oct. 2024, doi: 10.1186/s13071-024-06488-y.

[15] D. Engelman *et al.*, "A framework for scabies control," *PLoS Negl. Trop. Dis.*, vol. 15, no. 9, p. e0009661, Sep. 2021, doi: 10.1371/journal.pntd.0009661.

[16] J. Al-Dabbagh, R. Younis, and N. Ismail, "The current available diagnostic tools and treatments of scabies and scabies variants: An updated narrative review," May 2023, *Lippincott Williams and Wilkins*. doi: 10.1097/MD.0000000000033805.

[17] S. Uzun *et al.*, "Clinical practice guidelines for the diagnosis and treatment of scabies," *Int. J. Dermatol.*, vol. 63, no. 12, pp. 1642–1656, 2024, doi: 10.1111/ijd.17327.

[18] S. Halder, S. Panda, A. Samadder, and J. Chattopadhyay, "Enhancing Disease Control in Resource-Limited Settings Through Bidirectional Behavioral Responses," *Bull. Math. Biol.*, vol. 87, no. 10, p. 149, Sep. 2025, doi: 10.1007/s11538-025-01514-1.

[19] S. A. T. S. Estri and M. Khotibudin, "Incidence and Management of Scabies in Boarding School: Perception from Residents," *IJNP (Indonesian J. Nurs. Pract.*, vol. 6, pp. 18–27, Jun. 2022, doi: 10.18196/ijnp.v6i1.13355.

[20] S. Sungkar, "Linking the Life Cycle of Sarcoptes scabiei to Diagnosis, Treatment, and Control," *eJournal Kedokt. Indones.*, vol. 13, no. 3, p. 1, Dec. 2025, doi: 10.23886/ejki.13.1162.1.

[21] F. Brauer, C. Castillo-Chavez, and Z. Feng, *Mathematical Models in Epidemiology*, vol. 69. in Texts in Applied Mathematics, vol. 69. New York, NY: Springer, 2019. doi: 10.1007/978-1-4939-9828-9.

[22] J. Baafi and A. Hurford, "Modeling the Impact of Seasonality on Mosquito Population Dynamics: Insights for Vector Control Strategies," *Bull. Math. Biol.*, vol. 87, Feb. 2025, doi: 10.1007/s11538-024-01409-7.

[23] N. Holmes, "New Study Clarifies Transmission Dynamics of Scabies in Europe," Dec. 2025. [Online]. Available: https://www.emjreviews.com/dermatology/news/new-study-clarifies-transmission-dynamics-of-scabies-in-europe/

[24] A. T. Alabdala, Y. Adel, and W. Adel, “Optimal control strategies for infectious disease management: Integrating differential game theory with the SEIR model,” *Partial Differ. Equations Appl. Math.*, vol. 12, Dec. 2024, doi: 10.1016/j.padiff.2024.100943.

[25] M. J. Hasan, M. A. Rafi, T. Choudhury, and M. G. Hossain, “Prevalence and risk factors of scabies among children living in Madrasahs (Islamic religious boarding schools) of Bangladesh: A cross-sectional study,” *BMJ Paediatr. Open*, vol. 8, Jun. 2024, doi: 10.1136/bmjpo-2023-002421.

[26] P. van den Driessche, “Reproduction numbers of infectious disease models,” *Infect. Dis. Model.*, vol. 2, no. 3, pp. 288–303, Jun. 2017, doi: 10.1016/j.idm.2017.06.002.

[27] D. D. Fernando *et al.*, “Scabies,” *Nat. Rev. Dis. Prim.*, vol. 10, no. 1, p. 74, Oct. 2024, doi: 10.1038/s41572-024-00552-8.

[28] E. Browne *et al.*, “Sustaining Transmission in Different Host Species: The Emblematic Case of Sarcoptes scabiei,” *Bioscience*, vol. 72, no. 2, pp. 166–176, Feb. 2022, doi: 10.1093/biosci/biab106.

[29] I. J. Hindle, L. K. Forbes, and S. Carver, “The effect of spatial dynamics on the behaviour of an environmentally transmitted disease,” *J. Biol. Dyn.*, vol. 16, no. 1, pp. 144–159, Dec. 2022, doi: 10.1080/17513758.2022.2061614.

[30] H. W. Hethcote, “The Mathematics of Infectious Diseases,” *SIAM Rev.*, vol. 42, no. 4, pp. 599–653, 2000, doi: 10.1137/S0036144500371907.