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Research Paper

The SEIRV-SEI age structured epidemic model for vector born disease

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Abstract:
Vector-borne diseases such as malaria remain a major global health challenge due to their complex transmission dynamics between humans and vectors. In this work, we develop and analyze a coupled deterministic–stochastic age-structured model for malaria transmission. The human population is stratified by age into susceptible, exposed, infectious, recovered, and vaccinated classes, governed by partial differential equations (PDEs), while the mosquito population is described by susceptible, exposed, and infectious compartments using ordinary differential equations (ODEs). To capture demographic and environmental fluctuations, stochastic perturbations are introduced, resulting in a hybrid system of stochastic partial differential equations (SPDEs) for humans and stochastic differential equations (SDEs) for mosquitoes. We establish well-posedness of the model, prove existence and uniqueness of solutions, and derive expressions for the disease-free equilibrium (DFE), endemic equilibrium (EE), and the basic reproduction number R_0using the next-generation operator method. Furthermore, we investigate extinction and persistence conditions of the disease under stochastic influences. Our analysis demonstrates that when $R_0<1$, the DFE is globally stable and malaria dies out, while for $R_0>1$, an endemic equilibrium emerges, with stochastic effects potentially altering persistence outcomes. The incorporation of age structure highlights how immunity acquisition, vaccination, and demographic heterogeneity affect long-term malaria dynamics. This framework provides valuable insights for designing targeted control strategies that account for age-specific risk, vaccination campaigns, and environmental variability.

Key words: *Vector-borne diseases, Malariatransmission, Basic reproduction number, Age-structured mode, SDE.*

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I. Introduction

Stochastic modeling has become an essential tool in epidemic dynamics, providing a framework to capture random fluctuations that deterministic models often overlook. Early work by Gurarie and McKenzie (2007) introduced a stochastic model of malaria infection in children, linking disease progression and immune status to age and prior exposure. Their individual-based approach reproduced several field-observed patterns and emphasized the interplay between immunity, stochasticity, and intervention effects.Subsequently, Gahungu et al. (2017) developed a stochastic age-structured malaria transmission model incorporating Brownian noise into a deterministic system. They demonstrated that when the basic reproduction number$R_0 < 1$, stochastic trajectories converge toward deterministic ones, while for $R_0 > 1$, deviations appear, underlining the influence of random effects. Around the same time, Dang et al. (2017) formulated a PDE-based vector–host model including incubation and infection-age structures, deriving R_0 and proving global stability of equilibrium using Lyapunov function techniques. Similarly, Zhang et al. (2017)analyzed the stochastic dynamics of an SVIR model under random environmental noise, showing extinction when the stochastic reproduction number is below unity and persistence when it exceeds unity.Advancing this line of research, Wang and Wang (2021) conducted a complete analysis of an age–space structured malaria epidemic model within a bounded domain. They established global existence, derived an explicit expression for R_0, and analyzed stability through fixed-point theory and Lyapunov methods. To incorporate additional biological realism, Richard et al. (2022) proposed a human–vector malaria model structured by host age, time since infection, and waning immunity, offering new insights into long-term transmission and immunity loss.Further progress was made by Wang et al.

(2023), who analyzed an age–space structured nonlocal reaction–diffusion malaria model, deriving threshold dynamics and verifying results with simulations. In the same year, Cao and Nie (2023)introduced a stochastic vector–host model with age-dependent vaccination and disease relapse, proving existence, uniqueness, and conditions for extinction and persistence, and demonstrating the role of relapse and immunity duration.The most recent developments include Seck et al. (2025), who proposed an age-structured malaria transmission model for both human and vector populations, proving the positivity, boundedness, and global stability of equilibria via graph-theoretic methods and validating results using real data from Senegal. Likewise, Guo et al. (2025) developed a vector–host model incorporating biological age structure and asymptomatic infections. They derived the basic reproduction number and showed that the disease-free equilibrium is globally asymptotically stable when $R_0 < 1$ and the endemic equilibrium is locally stable when $R_0 > 1$. Their results highlighted that longer asymptomatic durations can reduce symptomatic transmission, offering valuable insights for control strategies.

Vector-borne diseases (VBDs) are infections that are transmitted between humans and other animals through the bites of blood-feeding arthropods such as mosquitoes, ticks, flies, and sand-flies. The pathogen-whether a virus, bacterium or parasite-is carried by the vector and introduced into the host during feeding. These diseases are responsible for a substantial burden of illness worldwide, accounting for millions of cases and deaths each year. They are particularly prevalent in tropical and subtropical regions, where climatic conditions support high vector density and activity. The transmission cycle of a vector-borne disease involves two interacting populations: the human (or animal) host and the vector. Control of such diseases is challenging because it requires not only protecting hosts through vaccination or treatment but also reducing vector populations and interrupting contact between hosts and vectors.

Vector-borne diseases (VBDs) represent a major global health challenge, accounting for a significant fraction of infectious disease morbidity and mortality worldwide. These diseases, transmitted through vectors such as mosquitoes, ticks, or flies, include some of the most persistent public health threats in tropical and subtropical regions. The transmission dynamics of VBDs are inherently complex, as they depend on the interactions between human hosts, vector populations, climatic conditions, ecological changes, and the implementation of control strategies such as vaccination, vector management and prophylaxis.

Mathematical modelling provides a powerful framework for understanding the spread of vector-borne infections and evaluating the impact of interventions. Classical models often adopt compartmental structures that divide the host and vector populations into susceptible, exposed, infectious, and recovered classes. However, such models typically neglect heterogeneity in host populations, particularly differences arising from age. In many vector-borne diseases, age plays a critical role in shaping transmission and disease severity. This motivates the use of age-structuredmodels, where host compartments depend on both age and time, providing a more realistic description of immunity acquisition, disease progression, and intervention outcomes.Vector populations, in contrast, generally have short lifespans and are therefore modelled without explicit age structure. Their dynamics are commonly described by ordinary differential equations that capture recruitment, infection, and mortality processes. The interaction between host and vector populations is mediated through the force of infection, which incorporates contact rates, biting intensity, and transmission probabilities.

Deterministic models capture the mean behaviour of disease transmission but real-world epidemics are subject to stochastic fluctuations. Randomness arises from demographic variability, environmental factors, vector abundance, and irregular contact patterns. These sources of noise can substantially alter disease outcomes, especially in small or heterogeneous populations. To address this, deterministic models can be extended by incorporating stochastic partial differential equations (SPDEs) for age-structured host dynamics and stochastic differential equations (SDEs) for vector dynamics. Such hybrid deterministic–stochastic formulations better reflect the inherent uncertainty of vector-borne disease transmission.

In this study, we present a deterministic–stochastic framework for vector-borne disease transmission. The host population is structured by age and time into susceptible, exposed, infectious, recovered, and vaccinated compartments, governed by PDEs. The vector population is divided into susceptible, exposed, and infectious classes, governed by ODEs. Stochastic perturbations are introduced into both systems, yielding SPDEs for host compartments and SDEs for vector compartments. This unified modelling framework provides a versatile platform to analyse how host heterogeneity, vaccination, and random environmental effects influence the persistence, control, or eradication of vector-borne infections.

II. Description of the model

2.1 Model overview

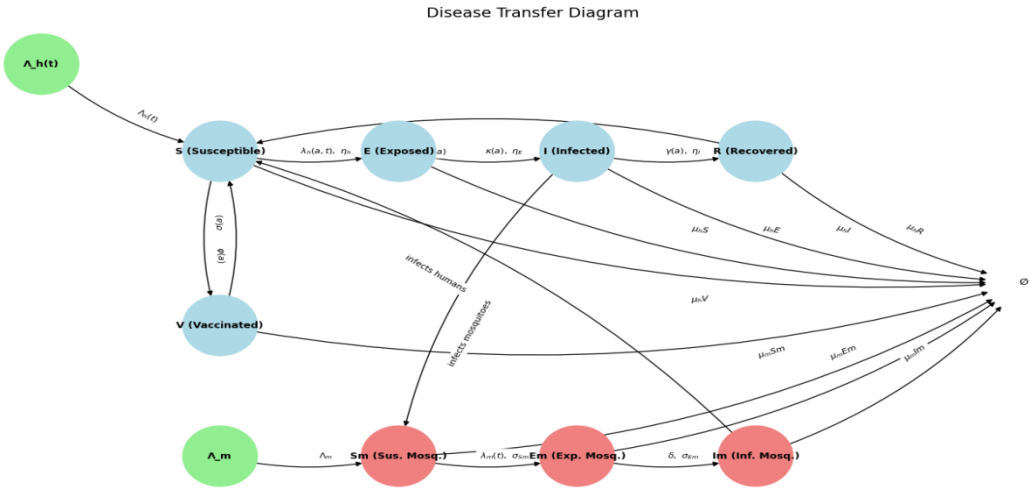


Fig.1.Disease transfer diagram

The disease transfer diagram illustrates the flow of individuals between compartments in the age-structured malaria model, highlighting both human and mosquito dynamics. The cross-links emphasize that infectious humans contribute to mosquito infection, while infectious mosquitoes drive human infections, thereby closing the transmission cycle.

In this age-structured malaria model the human population is divided into five compartments depending on age $a \geq 0$and time $t \geq 0$ susceptible humans $S(a,t)$, exposed humans $E(a,t)$, infectious humans $I(a,t)$, recovered or immune humans $R(a,t)$,and vaccinated or prophylaxis-protected humans $V(a,t)$.The total human population at time t is given by

$$N_h(t) = \int_0^{\infty}[\mathrm{S}(a,t) + \mathrm{E}(a,t) + \mathrm{I}(a,t) + \mathrm{R}(a,t) + \mathrm{V}(a,t)]\mathrm{d}a. \quad (1)$$

Newborn humans enter the population through a recruitment rate Λ_h, which is usually concentrated at age zero so that infants are added to the susceptible class. Humans die at an age-dependent natural death rate $\mu_h(a)$. The epidemiological processes influencing human transitions include the vaccination/prophylaxis rate $\phi(a)$, waning of vaccine-induced immunity $\sigma(a)$, waning of natural immunity $\omega(a)$, progression from exposed to infectious $\kappa(a)$, and recovery of infectious individuals $\gamma(a)$. Transmission from mosquitoes to humans is determined by the age-dependent transmission rate $\beta_h(a)$, with the corresponding force of infection for humans denoted by $\lambda_h(a,t)$.

The mosquito population is not age-structured and is described by three compartments: susceptible mosquitoes $S_m(t)$, exposed mosquitoes $E_m(t)$, and infectious mosquitoes $I_m(t)$. Their total population is

$$N_m(t) = S_m(t) + E_m(t) + I_m(t) \quad (2)$$

Mosquitoes are recruited into the susceptible class at a constant rateΛ_m and die at a natural death rate μ_m. Exposed mosquitoes progress to the infectious class at rate δ. Transmission from humans to mosquitoes is governed by the coefficient β_m, and the corresponding mosquito force of infection is given by $\lambda_m(t)$.

The force of infection for humans depends on the biting activity of the vector population and is given by:

$\lambda_h(a,t) = \beta_h(a)\frac{\mathrm{I_m}(t)}{N_m(t)}$Where,$\beta_h(a)$ is the age- dependent mosquito-to-human transmission rate, similarly,the force of infection for mosquitoes is

$\lambda_m(t) = \beta_m \frac{\int_0^{\infty} I(t,a)da}{N_h(t)}$Where β_m is the human-to-mosquito transmission coefficient.

2.2The deterministic age-structured PDEs for

$$\textbf{humans}\begin{cases} \frac{\partial \mathbf{S}}{\partial \mathbf{t}} + \frac{\partial \mathbf{S}}{\partial \boldsymbol{a}} = \boldsymbol{\Lambda_h}(\boldsymbol{t}) - \boldsymbol{\lambda_h}(\boldsymbol{a},\boldsymbol{t})\boldsymbol{S}(\boldsymbol{a},\boldsymbol{t}) - \boldsymbol{\phi}(\boldsymbol{a})\boldsymbol{S}(\boldsymbol{a},\boldsymbol{t}) + \boldsymbol{\omega}(\boldsymbol{a})\mathbf{R}(\boldsymbol{a},\mathbf{t}) + \boldsymbol{\sigma}(\boldsymbol{a})\mathbf{V}(\boldsymbol{a},\mathbf{t}) - \boldsymbol{\mu_h}(\boldsymbol{a})\mathbf{S}(\boldsymbol{a},\boldsymbol{t}) \\ \frac{\partial \mathbf{E}}{\partial \mathbf{t}} + \frac{\partial \mathbf{E}}{\partial \boldsymbol{a}} = \boldsymbol{\lambda_h}(\boldsymbol{a},\boldsymbol{t})\boldsymbol{S}(\boldsymbol{a},\boldsymbol{t}) - \boldsymbol{\kappa}(\boldsymbol{a})\mathbf{E}(\boldsymbol{a},\mathbf{t}) - \boldsymbol{\mu_h}(\boldsymbol{a})\mathbf{E}(\boldsymbol{a},\boldsymbol{t}) \\ \frac{\partial \mathbf{I}}{\partial \mathbf{t}} + \frac{\partial \mathbf{I}}{\partial \boldsymbol{a}} = \boldsymbol{\kappa}(\boldsymbol{a})\mathbf{E}(\boldsymbol{a},\mathbf{t}) - \boldsymbol{\gamma}(\boldsymbol{a})\mathbf{I}(\boldsymbol{a},\mathbf{t}) - \boldsymbol{\mu_h}(\boldsymbol{a})\mathbf{I}(\boldsymbol{a},\boldsymbol{t}) \\ \frac{\partial \mathbf{R}}{\partial \mathbf{t}} + \frac{\partial \mathbf{R}}{\partial \boldsymbol{a}} = \boldsymbol{\gamma}(\boldsymbol{a})\mathbf{I}(\boldsymbol{a},\mathbf{t}) - \boldsymbol{\omega}(\boldsymbol{a})\mathbf{R}(\boldsymbol{a},\mathbf{t}) - \boldsymbol{\mu_h}(\boldsymbol{a})\mathbf{R}(\boldsymbol{a},\boldsymbol{t}) \\ \frac{\partial \mathbf{V}}{\partial \mathbf{t}} + \frac{\partial \mathbf{V}}{\partial \boldsymbol{a}} = \boldsymbol{\phi}(\boldsymbol{a})\boldsymbol{S}(\boldsymbol{a},\boldsymbol{t}) - \boldsymbol{\sigma}(\boldsymbol{a})\mathbf{V}(\boldsymbol{a},\boldsymbol{t}) - \boldsymbol{\mu_h}(\boldsymbol{a})\mathbf{V}(\boldsymbol{a},\boldsymbol{t}) \end{cases} \quad (\mathbf{3})$$

Mosquito compartments ODEs:

$$\begin{cases} \frac{dS_m}{dt} = \Lambda_m - \lambda_m(t)S_m(t) - \mu_m S_m(t), \\ \frac{dE_m}{dt} = \lambda_m(t)S_m(t) - (\delta + \mu_m)E_m(t), \\ \frac{dI_m}{dt} = \delta E_m(t) - \mu_m I_m(t) \end{cases} \quad (4)$$

2.3 Boundary and Initial Conditions: For humans, newborns enter at age $\boldsymbol{a = 0}$into the susceptible class and For mosquitoes, recruitment occurs into the susceptible class at rate$\boldsymbol{\Lambda_m}$, while no mosquitoes are born directly into the exposed or infectious classes. Thus, the boundary conditions are:

$$\begin{cases} \boldsymbol{S(0,t) = \Lambda_h(t)}, \\ \mathbf{E(0,t) = 0}, \\ \mathbf{I(0,t) = 0}, \\ \boldsymbol{R(0,t) = 0}, \\ \boldsymbol{V(0,t) = 0}. \end{cases} \quad \begin{cases} \boldsymbol{S_m(t,0) = \Lambda_m}, \\ \boldsymbol{E_m(t,0) = 0}, \\ \boldsymbol{I_m(t,0) = 0}. \end{cases} \quad (5)$$

The corresponding initial distributions$\boldsymbol{t = 0}$are:

$$\begin{cases} \boldsymbol{S(a,0) = S_0(a)}, \\ \boldsymbol{E(a,0) = E_0(a)}, \\ \boldsymbol{I(a,0) = I_0(a)}, \\ \boldsymbol{R(a,0) = R_0(a)}, \\ \boldsymbol{V(a,0) = V_0(a)}. \end{cases} \quad \begin{cases} \boldsymbol{S_m(0) = s_{m0}}, \\ \boldsymbol{E_m(0) = E_{m0}}, \\ \boldsymbol{I_m(0) = I_{m0}}. \end{cases} \quad (6)$$

Summing the five human PDEs gives the population balance equation for the total human population:

$$\frac{\partial}{\partial \mathrm{t}}(S + E + I + R + V) + \frac{\partial}{\partial a}(S + E + I + R + V) = \Lambda_h(t)\delta(a) - \mu_h(a)(S + E + I + R + V)$$

Integrating over all ages:$\frac{dN_h(t)}{dt} = \Lambda_h(t) - \int_0^\infty \mu_h(a)(S + E + I + R + V)(a,t)da.$ (7)

For mosquitoes:$\frac{dN_m(t)}{dt} = \Lambda_m - \mu_m N_m(t).$(8)

Thus the human and mosquito total populations are bounded provided $\Lambda_h(t), \Lambda_m$ are finite.

2.4 Normalization: To simplify the analysis, we introduce normalized variables representing the fraction of each compartment relative to the corresponding total population, as given in equation (1)

$\mathrm{s}(a,\mathrm{t}) = \frac{S(a,t)}{N_h(t)}, e(a,t) = \frac{E(a,t)}{N_h(t)}, i(a,t) = \frac{I(a,t)}{N_h(t)}, r(a,t) = \frac{R(a,t)}{N_h(t)}, v(a,t) = \frac{V(a,t)}{N_h(t)}$ (9)

These fractions satisfy$\int_0^\infty [s(a,t) + e(a,t) + i(a,t) + r(a,t) + v(a,t)]da = 1.$Provided$N_h(t) > 0.$

For mosquitoes proportions:as given in equation (2)

$s_m(t) = \frac{S_m(t)}{N_m(t)}, e_m(t) = \frac{E_m(t)}{N_m(t)}, i_m(t) = \frac{I_m(t)}{N_m(t)}$ (10)

With $s_m(t) + e_m(t) + i_m(t) = 1$ whenever $N_m(t) > 0$.

The rescaled model can then be expressed in terms of these normalized fractions, facilitating stability analysis and the calculation of threshold parameters such as the basic reproduction number R_0.

In the rescaled model, we define the forces of infection and total population derivatives in normalized form as follows

$$\lambda_h(a,t) = \beta_h(a)i_m(t), \lambda_m(t) = \beta_m \int_0^\infty i(a,t)da.$$

$$\dot{N}_h(t) = \Lambda_h(t) - \int_0^\infty \mu_h(a)N_h(t)[s + e + i + r + v](a,t)da.$$

$\dot{N}_h(t) = \Lambda_h(t) - \int_0^\infty \mu_h(a)N_h(t)X(a,t)da,$with$X = S + E + I + R + V. \dot{N}_m(t) = \Lambda_m - \mu_m N_m(t).$

2.5 Normalized human PDE:write f for any human fraction s, e, i, r, v.if F satisfies the density transport equation$\partial_t F + \partial_a F = G_F$, then $f = \frac{F}{N_h}$ satisfies

$\partial_t f + \partial_a f = \frac{1}{N_{h(t)}} G_F(a,t) - \frac{\dot{N}_h(t)}{N_h(t)} f(a,t).$ (11)

Applying this to each compartment yields

$$\begin{cases} \partial_t s + \partial_a s = -\lambda_h(a,t)s - \phi(a)s + \omega(a)r + \sigma(a)v - \mu_h(a)s - \frac{\dot{N}_h(t)}{N_h(t)}s, \\ \partial_t e + \partial_a e = \lambda_h(a,t)s - \kappa(a)e - \mu_h(a)e - \frac{\dot{N}_h(t)}{N_h(t)}e, \\ \partial_t i + \partial_a i = \kappa(a)e - \gamma(a)i - \mu_h(a)i - \frac{\dot{N}_h(t)}{N_h(t)}i, \\ \partial_t r + \partial_a r = \gamma(a)i - \omega(a)r - \mu_h(a)r - \frac{\dot{N}_h(t)}{N_h(t)}r, \\ \partial_t v + \partial_a v = \phi(a)s - \sigma(a)v - \mu_h(a)v - \frac{\dot{N}_h(t)}{N_h(t)}v. \end{cases} \quad (12)$$

Here $N_h(t)$ is the time derivative of the total human population given above.For the mosquito ODEs in rescaled form, we apply the quotient rule to obtain

$s_m(t) = \frac{S_m}{N_m(t)}and\dot{s}_m = \Lambda_m - \lambda_m(t)S_m(t) - \mu_m S_m(t)$ using the quotient rule,

$$\dot{s}_m = \frac{s_m}{N_m(t)} - \frac{\dot{N}_m}{N_m}s_m = -\lambda_m(t)s_m + \frac{\Lambda_m}{N_m} - \mu_m s_m - \frac{\dot{N}_m}{N_m}s_m$$

Similarly for e_m, i_m:

$$\begin{cases} \dot{s}_m = -\lambda_m(t)s_m + \frac{\Lambda_m}{N_m}(1 - s_m), \\ \dot{e}_m = \lambda_m(t)s_m - (\delta + \mu_m)e_m - \frac{\dot{N}_m}{N_m}e_m, \\ i_m = \delta e_m - \mu_m i_m - \frac{\dot{N}_m}{N_m}i_m. \end{cases} \quad (13)$$

At demographic steady state($\dot{N}_m \equiv \frac{\Lambda_m}{\mu_m}$), these simplify to the familiar form:

$$\begin{cases} \dot{s}_m = -\lambda_m s_m + \mu_m(1 - s_m), \\ \dot{e}_m = \lambda_m s_m - (\delta + \mu_m)e_m, \\ i_m = \delta e_m - \mu_m i_m. \end{cases}$$

Rescaled Initial and Boundary ConditionsHuman and mosquitonewborn boundary

$$\begin{cases} s(0,t) = \frac{\Lambda_h(t)}{N_h(t)}, \\ \mathrm{e}(0,\mathrm{t}) = 0, \\ \mathrm{i}(0,\mathrm{t}) = 0, \\ r(0,t) = 0, \\ v(0,t) = 0. \end{cases} \quad \begin{cases} s_m(0) = \frac{\Lambda_m}{N_m(0)}, \\ e_m(0) = 0, \\ i_m(0) = 0. \end{cases} \quad (14)$$

Initial distributions(at $t = 0$):

$$\begin{cases} \boldsymbol{s(a,0) = s_0(a)}, \\ \boldsymbol{e(a,0) = e_0(a)}, \\ \boldsymbol{i(a,0) = I_0(a)}, \\ \boldsymbol{r(a,0) = r_0(a)}, \\ \boldsymbol{v(a,0) = v_0(a)}. \end{cases} \quad \begin{cases} \boldsymbol{s_m(0) = s_{m0}}, \\ \boldsymbol{e_m(0) = e_{m0}}, \\ \boldsymbol{i_m(0) = i_{m0}}. \end{cases} \quad (15)$$

To analyse the dynamics,we rescale all compartments by their total populations.

$\int_0^\infty [\, s_0 + e_0 + i_0 + r_0 + v_0](a,t)da = 1$. And $s_m(t) + e_m(t) + i_m(t) = 1$

2.6 Existence and Uniqueness of Solutions

2.6.1 State space and notation

Let $\mathbb{X} := L^1\big((0,\infty); \mathrm{R}^5\big)$ with norm $||X||_1 = \int_0^\infty ||X(a)||_{R^5} da$. And human denote the human age-density vector by$X(a,t) = (S(a,t), E(a,t), I(a,t), R,(a,t), V(a,t))^T \in \mathbb{X}$

Let the mosquito stable be $Y(t) = (S_m(t), E_m(t), I_m(t))^T \in R_+^3$.

We write the coupled human-mosquito model can be abstractly as

$\frac{dX}{dt} = \mathcal{A}_0 X + \mathcal{L}X + \mathcal{N}(X,Y), \frac{dY}{dt} = F(Y,X)$, (16)

Where,$\mathcal{A}_0$ is the transport-mortality generator,L is the bounded transfer operator (progression, recovery, vaccination, waning), and $N$is the nonlinear infection operator.

Define,The linear transport-mortality generator $\mathcal{A}_0$: $D(\mathcal{A}_0) \subset \mathrm{X} \to \mathrm{X}$ by

$(\mathcal{A}_0 X)(a) := -\frac{dX}{da}(a) - M_0(a)X(a), M_0(a) := \mu_h(a)I_{5,}$ (17)

With domain $D(\mathcal{A}_0) := \{X \in W^{1,1}\big((0,\infty); \mathrm{R}^5\big): X(0) = 0\}$.

Under the standing assumption $\mu_h \in L^\infty(0,\infty)$ with $\mu_h \geq 0$,Then $\mathcal{A}_0$ generates a positive C_0- semigroup $\{T_0(t)\}_{t\geq 0}$ on$\mathbb{X}$(left shift transport semigroupwith age-dependent damping).

2.6.2Bounded linear transfer operator$\mathcal{L}$

Collect progression, recovery, vaccination and waning in a bounded multiplication operator $(LX)(a) \in L(a)X(a)$,Where

$$L(a) = \begin{pmatrix} -\phi(a) & 0 & 0 & \omega(a) & \sigma(a) \\ 0 & -\kappa(a) & 0 & 0 & 0 \\ 0 & \kappa(a) & -\gamma(a) & 0 & 0 \\ 0 & 0 & \gamma(a) & -\omega(a) & 0 \\ \phi(a) & 0 & 0 & 0 & -\sigma(a) \end{pmatrix} \quad (18)$$

If $\varphi, \kappa, \gamma, \omega, \sigma \in L^{\infty}(0,\infty)$ then L is bounded on X.By the bounded perturbation theorem,$\mathcal{A} := \mathcal{A}_0 + L$ generates a (positive) C_0-semigroup $\{T_0(t)\}_{t\geq 0}$ on$\mathbb{X}$.Since the entries are bounded measurable in $a, \mathcal{L}$ is bounded on $\mathbb{X}$.

Remark: With $M_0(a) = \mu_h(a)I_5$diagonal**,** the generator $\mathcal{A}_0$is standard. All age-local transfers (which couple components but not ages) are treated as a bounded perturbation$\mathcal{L}$, so $\mathcal{A}$:=$\mathcal{A}_0 + \mathcal{L}$ still generates a positive C_0-semigroup by bounded perturbation theory.

Nonlinear infection operator:The human infection operator is

$$\mathcal{N}(X,Y)(a) = \begin{pmatrix} -\lambda_h(a,Y)S(a) \\ \lambda_h(a,Y)S(a) \\ 0 \\ 0 \\ 0 \end{pmatrix} \lambda_h(a,Y) = \beta_h(a)\frac{I_m}{N_m}, \quad (19)$$

With $\beta_h \in L^{\infty}(0,\infty)$the mosquito force of infection used in F is $\lambda_m(Y) = \beta_m \frac{\int_0^{\infty} I(a)da}{N_h(t)}$.For bounded β_m, β_h, the map $X \to \mathcal{N}(X,Y)$ is locally Lipschitz on $\mathbb{X}$ (indeed Fréchet differentiable), for each fixed Y;indeed,for fixed Y one has the estimate

$\left|\left|N(X,Y) - N\left(\tilde{X},Y\right)\right|\right|_1 \leq C\left|\left|X - \tilde{X}\right|\right|_1$with$C = ||\beta_h||_{L^{\infty}}$.the mosquito ODE right-hand side $F(Y,X)$ because it depends linearly on S_m, E_m, I_m and linearly on the functional $\int I(a)da$.

Births at age $a = 0$are modelled as an inhomogeneous boundary input $b(t) = (\Lambda_h(t), 0,0,0,0)^T$. In the semi-group formulation this appears via the variation-of-constants formula with boundary perturbation (Webb, 1985):

$X(t) = T(t)X_0 + \int_0^t T(t-s)(\ \mathcal{N}\left(X(s),Y(s)\right)ds) + \mathcal{B}[b](t)$,(20)

Where ,$T(t)$ is the semi group of $\mathcal{A} = \mathcal{A}_0 + \mathcal{L}$ and $\mathcal{B}[b]$ is the standard boundary input operator (Miyadera–Voigt type); assuming $\Lambda_h \in L^{\infty}_{loc}, \mathcal{B}[b]$ is well defined and bounded.The mosquito ODEs$\dot{S_m} = \Lambda_m - \lambda_m(Y)S_m - \mu_m S_m, \dot{E_m} = \lambda_m(Y)S_m - (\delta + \mu_m)E_m, \dot{I_m} = \delta E_m - \mu_m I_m$are locally Lipschitz in (S_m, E_m, I_m), hence admit a unique non negative solution for any non-negative initial data by Picard-Lindelof theorem.

2.6.3Well-posedness of the model

We now establish existence, uniqueness, and non-negativityof solutions for the coupled human–mosquito system using semi-group theory for the PDE component and classical ODE theory for the mosquito subsystem.

Theorem 1 (Existence and Uniqueness and positivity of solutions)

Assume the following regularity and positivity conditions $\mu_h, \phi, \sigma, \omega, \kappa, \gamma, \beta_h \in L^{\infty}(0,\infty)$ with $\mu_h(a), \kappa(a), \gamma(a), \phi(a), \sigma(a), \omega(a) \geq 0, \forall a \geq 0, \Lambda_h \in L^{\infty}_{loc}\left([0,\infty)\right)$, and $\Lambda_m, \mu_m, \delta, > 0, \beta_m \geq 0$.then the following hold:

1. $\mathcal{A}$ =$\mathcal{A}_0 + \mathcal{L}$ generates a positive C_0 −semigroup$\{T(t)\}_{t\geq 0}$on $\mathbb{X} = L^1((0\infty); R^5$.
2. The nonlinear map $X \to \mathcal{N}(X,Y)$ is locally Lipschitz on $\mathbb{X}$ for any continuous mosquito trajectory$Y$.
3. For any non-negative initial data $X_0 \in \mathbb{X}$ and non negative mosquito initial data $Y_0 \in R^3_+$, the coupled human-mosquito system admits a unique, non-negative mild solution.$X \in \mathrm{C}([0,\mathrm{T}];\mathbb{X}), Y \in \mathrm{C}([0,\mathrm{T}];R^3)$For everyfinite$T > 0$.

4.Moreover $||X(t)||_1 + \left|\left|Y(t)\right|\right| < \infty$ for all $t \in [0,\mathrm{T}]$remain finite on bounded time intervals.

Proof : Under the hypotheses of the theorem (bounded measurable coefficients) Assume $\mu_h, \phi, \sigma, \omega, \kappa, \gamma, \beta_h \in L^{\infty}(0,\infty)$ with $\mu_h, \kappa, \gamma, \phi, \sigma, \omega \geq 0, \Lambda_h \in L^{\infty}_{loc}\left([0,\infty)\right)$, and $\Lambda_m, \mu_m, \delta > 0, \beta_m \geq 0$,the operator decomposition and fixed-point argument below produce a unique nonnegative mild solution on some interval $[0,T]$. The solution extends as long as norms remain finite.

1. $\mathcal{A}_0$ generates a positive C_0 −semigroup and $\mathcal{A}$ =$\mathcal{A}_0 + \mathcal{L}$ does too

$(a)\mathcal{A}_0$is the transport-mortality generator.Define the transport-mortality operator

$(\mathcal{A}_0 X)(a) = -\frac{\partial \mathrm{X}}{\partial a}(a) - M_0(a)X(a), M_0(a) = \mu_h(a)I_5$(21)

With domain $D(\mathcal{A}_0) = \left\{X \in W^{1,1}\left((0,\infty);R^5\right): X(0)\right\}$.(22)

The operator $\mathcal{A}_0$ is the classical left-shift transport operator with age-dependent absorption. Standard results (transport semi-group theory; see e.g. *Webb,1985, Pazy,1983*) show that $\mathcal{A}_0$ generates a positive C_0-

semigroup$\{T_0(t)\}_{t\geq 0}$ on X. Concretely, the semigroup acts by shifting initial profiles to the right in age and multiplying by an integrating factor

$$[T_0(t)X_0](a) = \begin{cases} X_0(a-t)exp\big(-\int_{a-t}^{a}\mu_h(s)ds\big), & a \geq t, \\ 0 & a < t. \end{cases} \quad (23)$$

positivity follows from non-negativity .

(b)$\mathcal{L}$is bounded, so $\mathcal{A}$ =$\mathcal{A}_0 + \mathcal{L}$ generates a semigroup.

The operator $\mathcal{L}$ is a multiplication operator by the matrix $\mathcal{L}(a)$ whose entries $\varphi, \kappa, \gamma, \omega, \sigma$ are assumed essentially bounded.

For $X(a) \in R^5, (LX)(a) = L(a)X(a)$,(24)

Where, $L(a) = \begin{pmatrix} -\phi(a) & 0 & 0 & \omega(a) & \sigma(a) \\ 0 & -\kappa(a) & 0 & 0 & 0 \\ 0 & \kappa(a) & -\gamma(a) & 0 & 0 \\ 0 & 0 & \gamma(a) & -\omega(a) & 0 \\ \phi(a) & 0 & 0 & 0 & -\sigma(a) \end{pmatrix}$

so$L$ is a bounded linear operator on X.By the bounded perturbation theorem for C_0-semigroup, $\mathcal{A}$ =$\mathcal{A}_0 + \mathcal{L}$ generates a C_0-semigroup$T(t)$ on X. Positivity is preserved because $T_0(t)$ is positive and $\mathcal{L}(a)$ has the sign structure that preserves nonnegativity of components (one can check that off-diagonal entries that create transfers are nonnegative where appropriate); therefore $T(t)$ is a positive semi-group.

2. Local Lipschitz property of N(X,Y)

Recall the nonlinear infection operator

$$\mathrm{N(X,Y)}(a) = \begin{pmatrix} -\lambda_h(a,Y)S(a) \\ \lambda_h(a,Y)S(a) \\ 0 \\ 0 \\ 0 \end{pmatrix}, \lambda_h(a,Y) = \beta_h(a)\frac{I_m}{N_m}. \quad (25)$$

Fix a continuous $Y(t)$ with $N_m(t) > 0$and $|\frac{I_m}{N_m}| \leq 1$. Let $X, \tilde{X} \in$ X.using $|\beta_h(a)| \leq$(boundedness) and $||S||_{L^1} \leq ||X||_1$, compute for the L^1-norm

$$||N(X,Y) - N(\tilde{X},Y)||_1 = \int_0^\infty |\lambda_h(a,Y)||S(a) - \tilde{S}(a)|da \leq B\int_0^\infty |S(a) - \tilde{S}(a)|da \leq B||X - \widetilde{X}||_1 \quad (26)$$

Thus $N(.,Y): X \to X$ is Lipschitz on X (globally Lipschitz in fact, with constant B) for fixed Y. If β_h were only locally bounded you still obtain local Lipschitzness on balls. The dependence on Y is continuous as well because λ_h depends continuously on $\frac{I_m}{N_m}$.Since$\frac{I_m}{N_m} \leq 1$ in the positive cone,B is finite.

3. Fixed-point construction of a mild solution (local existence & uniqueness)

Write the variation-of-constants (mild solution) formula for the coupled system:

$X(t) = T(t)X_0 + \int_0^t T(t-s)N\big(X(s),Y(s)\big)ds + B[b](t)$,(27)

where$B[b](t)$ denotes the bounded boundary-input operator produced by birth term $b(t) = (\Lambda_h(t), 0,0,0,0)^T$. The mosquito ODEs are $\dot{Y}(t) = F\big(Y(t),X(t)\big)$,With F locally Lipschitz in (Y,X)because $\lambda_m = \beta_m \int \frac{I(a)}{N_h}$ depends linearly on X.Set up the banach space $\mathcal{B}_T := C([0,T]; X \times R^3)$

With norm $||(X,Y)||_T = \sup_{t\in[0,T]}\big(||X(t)||_1 + \big||Y(t)\big|\big)$.Choose$R > 0$ large enough so that initial data lie in the ball of radius R,and define the closed ball $\bar{B}_R \subset \mathcal{B}_T$.

Define the mapping Φ on $\bar{B}_R$ by

$$\Phi\colon (\mathrm{X,Y}) \mapsto \left(T(t)X_0 + \int_0^t T(t-s)N\big(X(s),Y(s)\big)ds + B[b](t), Y_0 + \int_0^t F\big(Y(s),X(s)\big)ds\right). \quad (28)$$

Using boundedness of $T(t)$ on finite intervals,Lipschitz bounds for N (from step 2),local Lipschitznessof$F$,and boundedness of $B[b]$(because $\Lambda_h \in L^\infty_{\mathrm{loc}}$),one shows:

- For small enough $T > 0$,Φ maps $\bar{B}_R$ into itself.
- For possibly smaller T, Φ is a contraction on $\bar{B}_R$.

Hence a unique fixed point $(X,Y) \in \bar{B}_R$ exists by Banach's fixed-point theorem. This fixed point is exactly the unique mild solution on $[0,T]$.Standard continuation arguments extend the solution as long as $||X(t)||_1 + \big||Y(t)\big||$ remains finite; thus either the solution exists globally or the norm blows up in finite time.

4. Preservation of non-negativity

The semi-group $T(t)$ generated by $A$is positive (maps nonnegative functions to nonnegative functions).The boundary operator $B[b]$ is nonnegative when $\Lambda_h \geq 0$.The infection operator $N(X,Y)$ maps nonnegative X to

vectors whose first two components are $-\lambda_h S \; and \; \lambda_h S$ (which preserve non-negativity of E because production terms are nonnegative and depletion terms are of the correct sign).Moreover, the mosquito ODE vector field F preserves non-negativity (right-hand sides are standard bilinear/nonnegative forms).
Given nonnegative initial data $X_0 \geq 0, Y_0 \geq 0$,the fixed-point iteration can be started with nonnegative iterates and all iterates remain nonnegative by induction; the limit (the mild solution) is therefore nonnegative.Thus non-negativity is preserved for the mild solution.

(if you prefer the single operator form)If you want everything in one operator,you can write
$\frac{dX}{dt} = \big(-\partial_a - M(a)\big)X + \mathcal{N}(X,Y)$, With

$$M(a) = \begin{pmatrix} \mu_h + \phi(a) & 0 & 0 & -\omega(a) & -\sigma(a) \\ 0 & \mu_h(a) + \kappa(a) & 0 & 0 & 0 \\ 0 & -\kappa(a) & \mu_h(a) + \gamma(a) & 0 & 0 \\ 0 & 0 & -\gamma(a) & \mu_h + \omega(a) & 0 \\ 0 & 0 & 0 & 0 & \mu_h(a) + \sigma(a) \end{pmatrix} \quad (29)$$

Note that here $M(a)$ is not diagonal (because it includes transfers).This is mathematically fine –the same bounded-perturbation argument shows $\mathcal{A}$ still generates a positive C_0-semigroup-but many readers find the split $\mathcal{A}_0 + \mathcal{L}$ clearer:$\mathcal{A}_0$ diagonal $\mathcal{L}$ bounded.

2.7 Steady States

A steady state is a time-independent solution $(S^*, E^*, I^*, R^*, V^*; S_m^*, E_m^*, I_m^*)$Satisfying the human age-ODEs ((transport in age only) and mosquito algebraic equations with the birth boundary at$a = 0$.
Let $X^*(a) = (S^*(a), E^*(a), I^*(a), R^*(a), V^*(a))^T . Y^* = (S_m^*, E_m^*, I_m^*)^T$,satisfying the age-transport ODEs together with the mosquito algebraic steady equations and the birth boundary condition $X^*(0) = (\Lambda_h^*, 0,0,0,0)^T$.
$\frac{dX^*}{da}(a) = -A(a; i_m^*)X^*(a), a > 0$,With boundary$X^*(0) = (\Lambda_h^*, 0,0,0,0)^T$,Where$i_m^* := \frac{I_m^*}{N_m^*}$ and

$$A(a; i_m^*) = \begin{pmatrix} \mu_h + \phi + \lambda_h & 0 & 0 & -\omega & -\sigma \\ \lambda_h(a) & \mu_h + \kappa & 0 & 0 & 0 \\ 0 & -\kappa & \mu_h + \gamma & 0 & 0 \\ 0 & 0 & -\gamma & \mu_h + \omega & 0 \\ -\phi & 0 & 0 & 0 & \mu_h + \sigma \end{pmatrix}(a),$$

$\lambda_h(a) = \beta_h(a) i_m^*$ (30)

(Here each rate is a function of age a; suppressing $(a$) for readability.)Let$\Phi(a; i_m^*)$ be the fundamental matrix of$\frac{d}{da}Z = -A(.; i_m^*)Z$,
with$\Phi(a; i_m^*) = I_5$.Then$X^*(a) = \Phi(a; i_m^*)(= (\Lambda_h^*, 0,0,0,0)^T, a \geq 0$.
The total human population is $N_h^* = \int_0^\infty 1^T X^*(a)da, 1 = (1,1,1,1,1)^T$,
And stationarityrequires the demographic balance $\Lambda_h^* = \int_0^\infty \mu_h(a) 1^T X^*(a)da$,
Mosquito equations at steady state:Define$\lambda_m^* := \beta_m \frac{\int_0^\infty I^*(a)da}{N_h^*}$The mosquito steady algebraic system is$0 = \Lambda_m - \lambda_m^* S_m^* - \mu_m S_m^*, 0 = \lambda_m^* S_m^* - (\delta + \mu_m) E_m^*, 0 = \delta E_m^* - \mu_m I_m^*$
Solving gives$S_m^* = \frac{\Lambda_m}{\mu_m + \lambda_m^*}, E_m^* = \frac{\lambda_m^*}{\delta + \mu_m} S_m^*, I_m^* = \frac{\delta}{\mu_m(\delta + \mu_m)} \lambda_m^* S_m^*$
Moreover, The total mosquito population is always $N_m^* = \frac{\Lambda_m}{\mu_m}$ (independent of λ_m^*),
And the infectious fraction satisfies the convenient scalar identity
$i_m^* = \frac{I_m^*}{N_m^*} = \frac{\delta \lambda_m^*}{(\mu_m + \lambda_m^*)(\delta + \mu_m)}$ (31)

2.7.1Disease-Free Equilibrium (DFE)

At the DFE,$E^* \equiv 0, I^* \equiv 0, E_m^* = 0, I_m^* = 0, i_m^* = 0, \lambda_h \equiv 0, \lambda_m^* = 0$ Thus X^* solve a linear age system without infection:$\frac{dX^*}{da} = -A_{DFE}(a)X^*(a), A_{DFE}(a) = A(a; 0)$With $X^*(0) = (\Lambda_h^*, 0,0,0,0)^T$.In particular,$E^*(a) = I^*(a) \equiv 0, S_m^* = \frac{\Lambda_m}{\mu_m}, E_m^* = I_m^* = 0$.Closed-form expressions for (S^*, R^*, V^*) are given by$\Phi_{DFE}(a)$ (matrix exponential along age; see constant-rate corollary below).
$\lambda_h^* = \int_0^\infty \mu_h(a)[S^*(a), R^*(a), V^*(a)]da$.(32)

2.7.2Endemic Equilibrium (EE)

At an endemic equilibrium has $E^*, I^*, E_m^*, I_m^* > 0, i_m^* > 0, \lambda_h(a) = \beta_h(a) i_m^* > 0$, $\lambda_m^* = \beta_m \frac{\int I^*}{N_h^*} > 0$.Given a candidate $i_m^* \in (0,1)$, the human age profile is $X^*(a; i_m^*) = \Phi(a; i_m^*)(\Lambda_h^*, 0,0,0,0)^T$,from which we

compute$\ell(i_m^*) = \int_0^\infty I^*(a; i_m^*)da, N_h^*(i_m^*) := \int_0^\infty 1^T X(a; i_m^*)da$.Then$\lambda_m^* = \beta_m \frac{I(i_m^*)}{N_h^*(i_m^*)}, i_m^*(i_m^*) = \frac{\delta\lambda_m^*(i_m^*)}{(\delta+\mu_m)(\mu_m+\lambda_m^*(i_m^*))}$.An endemic equilibrium correspondence to a fixed point.

$$i_m^* = \frac{\delta\beta_m \frac{I(i_m^*)}{N_h^*(i_m^*)}}{(\delta+\mu_m)(\mu_m+\beta_m \frac{I(i_m^*)}{N_h^*(i_m^*)})}.$$

Existence of a positive fixed point is guaranteed when $R_0 > 1$(see the R_0 section): at $i_m = 0$ the derivative of the right-hand side equals $R_0 - 1$ up to a scaling,so a nontrivial intersection emerges for $R_0 > 1$ (standard bifurcation argument).

2.7.3Constant-rate corollary

Assume all host rates are age-independent constants$\mu_h, \phi, \sigma, \omega, \kappa, \gamma$ and $\beta_h(a) \equiv \beta_h$are constant.

At DFE$E^* = I^* \equiv 0$.then $R^*(a) = 0$(because$\frac{dR^*}{da} = -(\omega + \mu_h)R^*, R^*(0) = 0$).the (S^*, V^*) pair solves

$$\frac{d}{da}\begin{pmatrix}S^*(a)\\V^*(a)\end{pmatrix} = -\begin{pmatrix}\mu_h+\phi & -\sigma\\ -\phi & \mu_h+\sigma\end{pmatrix}\begin{pmatrix}S^*(a)\\V^*(a)\end{pmatrix}, \qquad \begin{pmatrix}S^*(0)\\V^*(0)\end{pmatrix} = \begin{pmatrix}\Lambda_h^*\\0\end{pmatrix}.$$

Hence $\begin{pmatrix}S^*(a)\\V^*(a)\end{pmatrix} = \exp(-aM)\begin{pmatrix}\Lambda_h^*\\0\end{pmatrix}. M = \begin{pmatrix}\mu_h+\phi & -\sigma\\ -\phi & \mu_h+\sigma\end{pmatrix}$

The demographic balance gives $\Lambda_h^* = \mu_h \int_0^\infty [S^*(a) + V^*(a)]da$ (which determinesΛ_h^* up to scaling/ normalization).

Let$i_m^* > 0$ be unknown. Then $\lambda_h = \beta_h i_m^*$ is constant and the (E^*, I^*) components admit volterra integral forms: $E^*(a) = \int_0^\infty e^{-(\kappa+\mu_h)(a-s)}\lambda_h S^*(s)ds, I^*(a) = \int_0^\infty e^{-(\gamma+\mu_h)(a-s)}\kappa E^*(s)ds$.

Meanwhile (S^*, R^*, V^*) solve a constant-coefficient linear system driven by E^*, I^*;In compact form $\frac{d}{da}X^* = -A(i_m^*)X^*, X^*(0) = (\Lambda_h^*, 0,0,0,0)^T, X^*(a) = e^{-aA(i_m^*)}X^*(0)$,With

$$A(i_m^*) = \begin{pmatrix}\mu_h+\phi+\beta_h i_m^* & 0 & 0 & -\omega(a) & -\sigma(a)\\ -\beta_h i_m^* & \mu_h+\kappa(a) & 0 & 0 & 0\\ 0 & -\kappa(a) & \mu_h+\gamma(a) & 0 & 0\\ 0 & 0 & -\gamma(a) & \mu_h+\omega(a) & 0\\ -\phi(a) & 0 & 0 & 0 & \mu_h+\sigma(a)\end{pmatrix}.$$

Compute $I(i_m^*) = \int_0^\infty I^*(a)da, \lambda_m^* = \beta_m \frac{I(i_m^*)}{N_h^*}$, and close with

$i_m^* = \frac{\delta\lambda_m^*}{(\delta+\mu_m)(\mu_m+\lambda_m^*)}$. (33)

Solving this scalar fixed-point yields the endemic i_m^*; then all steady profiles follow from the formulas above.

2.8 Basic Reproduction Number

The basic reproduction number $R_0$is the key threshold parameter that determines whether malaria persists in the population or dies out. It represents the expected number of new human infections generated by a single infected human introduced into a fully susceptible population, through the human–mosquito–human transmission cycle.We compute R_0 using the next-generation operator method(van den Driessche and Watmough, 2002). The infected compartments of the system are

$$\chi(t) = \big(E(a,t), I(a,t), E_m(t), I_m(t)\big),$$

with transmission occurring via $I(a,t)$(infectious humans) and $I_m(t)$(infectious mosquitoes).

2.8.1 Next-Generation Matrix Derivation

For completeness, we derive the same result using the NGM approach.

New infection matrix F and Transition matrix V:

$$F = \begin{bmatrix}0 & 0 & 0 & \frac{\beta_m S_m^*}{N_h^*}\\ 0 & 0 & 0 & 0\\ 0 & \beta_h & 0 & 0\\ 0 & 0 & 0 & 0\end{bmatrix}, \quad V = \begin{bmatrix}\kappa+\mu_h & 0 & 0 & 0\\ -\kappa & \gamma+\mu_h & 0 & 0\\ 0 & 0 & \delta+\mu_m & 0\\ 0 & 0 & -\delta & \mu_m\end{bmatrix}$$

The next-generation matrix is$K = FV^{-1}$,The eigenvalues of K are

$$\lambda = \pm\frac{\sqrt{S_m^*\beta_h\beta_m\kappa\delta}}{\sqrt{N_h^*}\mu_m\sqrt{(\kappa+\mu_h)(\gamma+\mu_h)(\delta+\mu_m)}}, \qquad 0, \qquad 0.$$

Therefore, the spectral radius is$R_0 = \frac{\sqrt{S_m^* \beta_h \beta_m \kappa \delta}}{\sqrt{N_h^*}\mu_m\sqrt{(\kappa+\mu_h)(\gamma+\mu_h)(\delta+\mu_m)}}$

Substituting $S_m^* = \frac{\Lambda_m}{\mu_m}$ again gives$R_0 = \sqrt{\frac{\beta_h \beta_m \kappa \delta \Lambda_m}{(\kappa+\mu_h)(\gamma+\mu_h)(\delta+\mu_m)\mu_m^2 N_h^*}}$. (34)

III. Age structured stochastic differential equation

To account for environmental and demographic randomness, the model is extended into a stochastic framework. The human compartments are governed by stochastic partial differential equations (SPDEs):

$$\begin{cases} \frac{\partial \mathrm{S}}{\partial \mathrm{t}} + \frac{\partial \mathrm{S}}{\partial a} = -\lambda_h(a,t)S(a,t) - \phi(a)S(a,t) + \omega(a)\mathrm{R}(a,t) + \sigma(a)\mathrm{V}(a,t) - \mu_h(a)\mathrm{S}(a,t) + \eta_S(a,t)W_S(t), \\ \frac{\partial \mathrm{E}}{\partial \mathrm{t}} + \frac{\partial \mathrm{E}}{\partial a} = \lambda_h(a,t)S(a,t) - \kappa(a)\mathrm{E}(a,\mathrm{t}) - \mu_h(a)\mathrm{E}(a,t) + \eta_E(a,t)W_E(t), \\ \frac{\partial \mathrm{I}}{\partial \mathrm{t}} + \frac{\partial \mathrm{I}}{\partial a} = \kappa(a)\mathrm{E}(a,\mathrm{t}) - \gamma(a)\mathrm{I}(a,\mathrm{t}) - \mu_h(a)\mathrm{I}(a,t) + \eta_I(a,t)W_I(t), \\ \frac{\partial \mathrm{R}}{\partial \mathrm{t}} + \frac{\partial \mathrm{R}}{\partial a} = \gamma(a)\mathrm{I}(a,\mathrm{t}) - \omega(a)\mathrm{R}(a,\mathrm{t}) - \mu_h(a)\mathrm{R}(a,t) + \eta_R(a,t)W_R(t), \\ \frac{\partial \mathrm{V}}{\partial \mathrm{t}} + \frac{\partial \mathrm{V}}{\partial a} = \phi(a)S(a,t) - \sigma(a)\mathrm{V}(a,t) - \mu_h(a)\mathrm{V}(a,t) + \eta_V(a,t)W_V(t). \end{cases} \quad (35)$$

Here,$\eta_i(a,t)$represent the of stochastic perturbations in compartment ($i \in \{\mathrm{S},\mathrm{E},\mathrm{I},\mathrm{R},\mathrm{V}\}$,while$W_i(t)$ areGaussian white noise processes modelling environmental or demographic stochasticity.

The mosquito dynamics are modelled by stochastic differential equations (SDEs):

$$\begin{cases} \frac{dS_m}{dt} = \Lambda_m - \lambda_m(t)S_m(t) - \mu_m S_m(t) + \xi_S W_S^m(t), \\ \frac{dE_m}{dt} = \lambda_m(t)S_m(t) - (\delta+\mu_m)E_m(t) + \xi_E W_E^m(t), \\ \frac{dI_m}{dt} = \delta E_m(t) - \mu_m I_m(t) + \xi_I W_I^m(t). \end{cases} \quad (36)$$

Here, ξ_i are noise intensities for mosquito compartments$(i \in \{S_m, E_m, I_m\}), and\ W_i^m(t)$are independent Gaussian white noise processes modelling random fluctuations in mosquito dynamics.Thus, the full model captures malaria transmission under both deterministic and stochastic influences. The deterministic part accounts for biological and epidemiological processes such as aging, vaccination, immunity waning, recovery, and vector biting, while the stochastic terms model random variations due to environmental and demographic factors.

Integrate the human SPDEs over age $a \in (0,\infty)$ and sum compartments to obtain a formal evolution for the total host population$\lambda_h(t)$. Using the transport terms and boundary $X(0,t) = b(t)$one obtains (formally)

$$N_h(t) = \int_0^\infty [S(a,t) + E(a,t) + I(a,t) + R(a,t) + V(a,t)]da$$

Starting from the compartmental SPDEs in conservation (transport)form $\frac{\partial X}{\partial t} + \frac{\partial X}{\partial a} = (\text{local reactions}) + \eta_X(a,t)W_X(t), X \in \{\mathrm{S},\mathrm{E},\mathrm{I},\mathrm{R},\mathrm{V}\}$ andintegrate each compartment over$a \in [0,\infty)$ and sum.using

$$\int_0^\infty \left(\frac{\partial X}{\partial t} + \frac{\partial X}{\partial a}\right) da = \frac{d}{dt}\int_0^t X(a,t)da + [X(\infty,t) - X(0,t)],$$

And assuming $X(\infty,t) = 0$ for each compartment,the transport terms produce the boundary contribution $-\sum X(0,t)$ on the left-hand side. Summing the right-hand side reaction terms, all internal transfers (infectionλ_h,progression κ,recovery γ, vaccination ϕ,waning σ) cancel pairwise,leaving only natural deaths and the integrated stochastic forcing.Thus the formal stochastic balance for $N_h(t)$ is

$$\frac{dN_h(t)}{dt} = \underbrace{\sum_{X\in\{\mathrm{S},\mathrm{E},\mathrm{I},\mathrm{R},\mathrm{V}\}} X(0,t)}_{\text{newborn/boundary inflow}} - \int_0^\infty \mu_h(a)N_h(a,t)da + \sum_{X\in\{S,E,I,R,V\}} \int_0^\infty \eta_X(a,t)da\ dW_i(t) \quad (37)$$

If your model prescribes only susceptible new-borns, i.e.$S(0,t) = \Lambda_h(t)$ and $E(0,t) = I(0,t) = R(0,t) = V(0,t) = 0$,the equation simplifies to

$\frac{dN_h(t)}{dt} = \Lambda_h(t) - \int_0^\infty \mu_h(a)N_h(a,t)da + \sum_X\left(\int_0^\infty \eta_X(a,t)da\right)W_X(t)$.(38)

Notes on the stochastic term: the formal integral $\int_0^\infty \eta_X(a,t)W_X(t)da$ aggregates age-dependent noise intensities into a single time-noise driving term. If you prefer the stochastic differential notation with standard Brownian motions$B_X(t)$,you can write

$dN_h(t) = \left[\Lambda_h(t) - \int_0^\infty \mu_h(a)N_h(a,t)da\right]dt + \sum_X\left(\int_0^\infty \eta_X(a,t)da\right)dB_X(t)$.(39)

Where,$dB_X(t)$are the increments corresponding to the W_X −type white noise processes.
$dN_h(t) = [\Lambda_h(t) - M(t)]dt + \sum_X \tilde{\eta}_X(t)dB_X(t)$,(40)
Where $M(t) = \int_0^\infty \mu_h(a)N_h(a,t)da$ and $\tilde{\eta}_X(t) = \int_0^\infty \eta_X(a,t)da$ .Let the aggregated quadratic variation of the martingale part be $Q_h(t) = \tilde{\eta}_X(t)^T R_h \tilde{\eta}(t)$ (if the Brownian drives B_X have correlation matrix R_h).
Derivation (Itoson N_h^2)

$$d(N_h^2) = 2N_h dN_h + d\langle N_h\rangle = 2N_h(\Lambda_h(t) - M)dt + 2N_h \sum_X \tilde{\eta}_X(t)dB_X(t) + Q_h dt.$$

Take expectations:$\frac{d}{dt}E[N_h^2] = 2E[N_h(\Lambda_h - M)] + Q_h$
Mean equation :$m'(t) = \Lambda_h(t) - E[M(t)]$.then(41)

$$\frac{d}{dt}\mathrm{Var}(N_h) = \frac{d}{dt}E[N_h^2] - 2mm' = 2E[N_h(\Lambda_h - M)] + Q_h - 2m(\Lambda_h - E[M]).$$

Rearrange the deterministic terms:$\frac{d}{dt}\mathrm{Var}(N_h) = -2(E[N_h M] - mE[M]) + Q_h$
But $[N_h M] - mE[M] = \mathrm{Cov}(N_h, M)$,
Thus $\frac{d}{dt}\mathrm{Var}(N_h) = -2\,\mathrm{Cov}\big(N_h(t), M(t)\big) + Q_h(t)$. (42)
Where,$M(t) = \int_0^\infty \mu_h(a)N_h(a,t)da$ and $Q_h(t)$aggregates the noise intensities and correlations.

3.1 Mosquito total population$N_m(t)$:$\frac{dN_m(t)}{dt} = \Lambda_m - \mu_m N_m(t) + \xi_S W_S^m(t) + \xi_E W_E^m(t) + \xi_I W_I^m(t)$
Or in Itosform (with Brownian increments dB^m):

$$\frac{dN_m(t)}{dt} = \Lambda_m - \mu_m N_m(t) + \xi_S B_S^m(t) + \xi_E B_E^m(t) + \xi_I B_I^m(t).$$

SDE:$dN_m(t) = (\Lambda_m - \mu_m N_m(t))dt + \xi_S B_S^m(t) + \xi_E B_E^m(t) + \xi_I B_I^m(t)$
Where,B_i are Brownian motions with covariation $d\langle B_i, B_j\rangle_t = \rho_{ij}dt$ and $\rho_{ij} = 1$.
Define $\xi = (\xi_S\xi_E\xi_I)^T$and $\mathrm{R} = (\rho_{ij})$. Then the instantaneous quadratic variation of the martingale part is $d\langle M\rangle_t = \xi^T \mathrm{R}\xi\, dt \equiv Qdt, Q = \sum_i \xi_i^2 + 2\sum_{i<j}\rho_{ij}\xi_i\xi_j$.
Derivation (Itoson N_m^2):
$d(N_m^2) = 2N_m dN_m + d\langle N_m\rangle = 2N_m(\Lambda_m - \mu_m N_m)dt + 2N_m \sum_i \xi_i dB_i + Qdt$. (43)
Take expectations (cross-term with dB vanishes):$\frac{d}{dt}E[N_m^2] = 2\Lambda_m m(t) - 2\mu_m E[N_m^2] + Q$.
Now $\mathrm{var}(N_m) = E[N_m^2] - m^2$.Differentiate:

$$\frac{d}{dt}\mathrm{var}(N_m) = \frac{d}{dt}E[N_m^2] - 2mm' = 2\Lambda_m m - 2\mu_m E[N_m^2] + Q - 2m(\Lambda_m - \mu_m m).$$

Simply (terms with $\Lambda_m m$ cancel):$\frac{d}{dt}\mathrm{var}(N_m) = -2\mu_m \mathrm{var}(N_m) + Q$.
Steady variance (if $\mu_m > 0$): $\mathrm{var}\big(N_m(\infty)\big) = \frac{Q}{2\mu_m}$(44)

3.2 Multiplicative-noise examples for N_m

I.Proportional (linear) multiplicative noise

SDE:$dN = (\Lambda - \mu\mathrm{N})\mathrm{dt} + \sigma\mathrm{NdB}$.
Ito on N^2:$d(N^2) = 2NdN + (dN)^2 = 2N(\Lambda - \mu\mathrm{N})\mathrm{dt} + 2\sigma N^2 dB + \sigma^2 N^2 dt$
Take expectations:$\frac{d}{dt}E[N^2] = 2\Lambda\mathrm{m} - 2\mu\mathrm{E}[N^2] + \sigma^2 E[N^2]$.
Hence $\frac{d}{dt}E[N^2] = 2\Lambda\mathrm{m} - (2\mu - \sigma^2)E[N^2]$
Mean:$m' = \Lambda - \mu m$. Then the variance satisfies

$$\mathrm{Var}' = \frac{d}{dt}E[N^2] - 2mm' = (\sigma^2 - 2\mu)\mathrm{E}[N^2] + 2\Lambda m - 2m(\Lambda - \mu m)$$

After simplification (use $E[N^2] = \mathrm{Var} + m^2$):$\frac{d}{dt}\mathrm{Var(N)} = (\sigma^2 - 2\mu)\mathrm{Var(N)} + \sigma^2 m^2$.

II. Square-root (CIR) noise

SDE:$dN = (\Lambda - \mu N)dt + \sigma\sqrt{N}dB$.
Ito's on N^2 (note$(dN)^2 = \sigma^2 N dt$): $d(N^2) = 2NdN + \sigma^2 N dt = 2N(\Lambda - \mu N)dt + 2\sigma N^{3/2}dB + \sigma^2 N dt$
Take expectations:$\frac{d}{dt}E[N^2] = 2\Lambda m - 2\mu\mathrm{E}[N^2] + \sigma^2 m$.
Mean:$m' = \Lambda - \mu m$.ThusVar$' = \frac{d}{dt}E[N^2] - 2mm' = -2\mu\mathrm{E}[N^2] + 2\Lambda m + \sigma^2 m - 2m(\Lambda - \mu m)$.
Simply (use $E[N^2] = \mathrm{var} + m^2$ and cancel):$\frac{d}{dt}\mathrm{var(N)} = -2\mu\,\mathrm{Var(N)} + \sigma^2 m$.

3.3 Extinction and Persistence of the Disease

In this section, we rigorously analyse the long–term behaviour of the stochastic age–structured malaria model. We derive sufficient conditions for extinction and persistence by employing tools from stochastic differential equations (Itô's formula, martingale theory, and Lyapunov function methods).

3.3.1 Setting and assumptions

Let the age-aggregated infected state be $X(t) = (E_h(t), I_h(t), E_m(t), I_m(t))^T \in \mathcal{R}^4_+$,

Where $E_h(t) = \int_0^{a_{max}} E(a,t)da \; and \; I_h(t) = \int_0^{a_{max}} I(a,t)da$,Near the disease-free state, the infected dynamics can be written (standard linearization plus higher-order terms) as

$dX(t) = [(F-V)X(t) - \Psi(X(t))]dt + \Sigma \mathrm{diag}\big(X(t)\big)dB(t)$, (A1)

where:

- $F \geq 0$ collects new-infection terms and V is an M-matrix (progression/removals);$\Psi: \mathcal{R}^4_+ \to \mathcal{R}^4_+$ captures nonlinear saturation/depletion (e.g.,finiteS, S_m); crucially,
$$\Psi(X) = \mathrm{o}\big(||\mathrm{X}||\big)\text{as } ||\mathrm{X}|| \to 0;$$
- $B(t)$is a 4-dim Brownian motion and $\Sigma = \mathrm{diag}\big(\sigma_{E_h}, \sigma_{I_h}, \sigma_{E_m}, \sigma_{I_m}\big) \geq 0$gives multiplicative noise intensities after age aggregation (the σ_k are the effective aggregated noise amplitudes obtained from the original $\eta_i(a,t)$and ξ_j).
- Global well-posedness/positivity/invariance**.** The full model implies a compact, positively invariant set $\kappa \subset \mathcal{R}^4_+$that attracts all trajectories a.s. (bounded human and mosquito totals). Standard Lipschitz/linear-growth conditions ensure a unique, nonnegative strong solution.

Threshold matrices

Let $K \coloneqq V^{-1}F, R_0 \coloneqq \rho(\mathrm{K}), A \coloneqq F - V$(Metzler,irreducible). (A2)

For Metzler A, its spectral abscissa $s(A)$ satisfies under the usual next-generation interpretation,

$s(A) > 0 \Leftrightarrow R_0 > 1, \mathrm{s}(A) < 0 \Leftrightarrow R_0 < 1$.(A3)

Effective noise level.Define$\sigma_* \coloneqq \max\{\sigma_{E_h}, \sigma_{I_h}, \sigma_{E_m}, \sigma_{I_m}\}$(A4)

Sublinearity of nonlinear terms**.** There exists $L > 0$ such that, for all $X \neq 0$,

$0 \leq \frac{\alpha^{\mathrm{T}}\Psi(X)}{\alpha^{\mathrm{T}}X} \leq L\big||X|\big|$, (A5)

We define the stochastic reproductionthresholdR$_0^{\mathrm{s}} \coloneqq \exp\left(\mathrm{s(A)} - \frac{1}{2}\sigma_*^2\right)$(A6)

So that R$_0^{\mathrm{s}} > 1 \Leftrightarrow \mathrm{s(A)} - \frac{1}{2}\sigma_*^2 > 0$

(This is the continuous-time SDE analogue of the well-known Itô penalty; in discrete-time or generation-scaled forms one often writesR$_0^{\mathrm{s}} = \mathrm{R}_0 \exp\left(-\frac{1}{2}\sigma_*^2\right)$.)

3.3.2 Extinction

Theorem 2: If$\mathbf{R_0^s < 1}$(equivalently $\mathbf{s(A) - \frac{1}{2}\sigma_*^2 < 0}$), then $\lim_{t\to\infty} \boldsymbol{X(t) = 0}$ almost surely.In particular,$\boldsymbol{I_h(a,t) \to 0}$ for all$\boldsymbol{a \in [0, a_{max}]}and\boldsymbol{I_m(t) \to 0}$almost surely.

Proof: Let$\alpha \in \mathcal{R}^4_+\{o\}$ be the positive left eigenvector of A^T to $s(A)$: $A^T\alpha = s(A)\alpha$.(B1)

Define $Y(t) \coloneqq \alpha^T X(t) > 0$ and $U(t) \coloneqq \ln Y(t)$. From (A1),$dY = \alpha^T dX = \alpha^T[(AX - \Psi(\mathrm{X}))\mathrm{dt} + \Sigma\mathrm{diag(X)dB}]$With quadratic variation is

$d\langle \mathrm{Y} \rangle = (\sum_{k=1}^4 \alpha_k \sigma_k X_k)^2 dt$.By Itô's formula,$dU = \frac{1}{Y}dY - \frac{1}{2Y^2}d\langle \mathrm{Y} \rangle$

$= \left[\frac{\alpha^T AX}{\alpha^T X} - \frac{\alpha^T \Psi(\mathrm{X})}{\alpha^T X} - \frac{1}{2}\frac{(\sum \alpha_k \sigma_k X_k)^2}{(\alpha^T X)^2}\right]dt + \frac{\alpha^T \Sigma \mathrm{diag(X)dB}}{\alpha^T X}$.Using $A^T\alpha = s(A)\alpha$ gives $\frac{\alpha^T AX}{\alpha^T X} = s(A)$.since $\Psi \geq 0, -\frac{\alpha^T \Psi(\mathrm{X})}{\alpha^T X} \leq 0$.also,$0 \leq \frac{(\sum \alpha_k \sigma_k X_k)^2}{(\alpha^T X)^2} \leq \sigma_*^2$.

Hence$dU \leq \left(s(A) - \frac{1}{2}\sigma_*^2\right)dt + dM_t$ (B2)

wheredM_t is a continuous local martingale with$\langle \mathrm{M} \rangle_t = \int_0^t \frac{(\sum \alpha_k \sigma_k X_k)^2}{(\alpha^T X)^2} ds \leq \sigma_*^2 t$.Integrating (B2) and dividing by t,$\frac{U(t)-U(0)}{t} \leq s(A) - \frac{1}{2}\sigma_*^2 + \frac{M_t}{t}$.By the strong law for continuous local martingales,$\frac{M_t}{t} \longrightarrow 0$ a.s. Thus$\limsup_{n\to\infty} \frac{U(t)}{t} \leq s(A) - \frac{1}{2}\sigma_*^2 < 0$.By the martingale strong law $\frac{M_t}{t} \longrightarrow 0$a.s., so the right side has negative limit. Hence $\limsup_{n\to\infty} \frac{U(t)}{t} \leq s(A) - \frac{1}{2}\sigma_*^2 < 0$, which implies $U(t) \longrightarrow -\infty$ linearly and thus $Y(t) \longrightarrow 0$ a.s.Since $\min_k \alpha_k =: c > 0$ we have $||X(t)|| \leq (\min_k \alpha_k)^{-1} Y(t) \longrightarrow 0$a.s., so$X(t) \longrightarrow 0$ componentwise almost surely.

3.3.3Persistence

Theorem 3: If$\mathbf{R_0^s > 1}$(equivalently $\boldsymbol{s(A) - \frac{1}{2}\sigma_*^2 > 0}$), then there exists $\boldsymbol{\varepsilon > 0}$ such that $\boldsymbol{\liminf_{t\to\infty} \frac{1}{t}\int_0^t ||X(s)||ds > \varepsilon a.s}$.,i.e., the infection persists stochastically**.**

Proof:

With the same $\alpha, Y = \alpha^T X > 0$ and $U = \ln Y$,the Itô computation above yields the complementary lower bound (now using $-\frac{1}{2}(\ldots) \geq -\frac{1}{2}\sigma_*^2$):

$dU \geq \left(s(A) - \frac{1}{2}\sigma_*^2\right)dt - \frac{\alpha^T \Psi(X)}{\alpha^T X}dt + dM_t$. (C1)

Integrate from 0 to t: $U(t) - U(0) \geq \left(s(A) - \frac{1}{2}\sigma_*^2\right)t - \int_0^t \frac{\alpha^T \Psi(X(s))}{\alpha^T X(s)}ds + M_t$.

Divide by t and rearrange: $\frac{1}{t}\int_0^t \frac{\alpha^T \Psi(X(s))}{\alpha^T X(s)}ds \geq \left(s(A) - \frac{1}{2}\sigma_*^2\right) + \frac{U(0)-U(t)}{t} + \frac{M_t}{t}$.(C2)

On the positively invariant compact $\kappa, U(t) = \ln(\alpha^T X(t))$ is bounded, hence $\frac{U(0)-U(t)}{t} \longrightarrow 0$a.s., so taking lim inf in (C2),

$\liminf_{n\to\infty} \frac{1}{t}\int_0^t \frac{\alpha^T \Psi(X(s))}{\alpha^T X(s)}ds \geq s(A) - \frac{1}{2}\sigma_*^2 > 0$ a.s (C3)

By the sub linearity (A5),$\frac{\alpha^T \Psi(X)}{\alpha^T X} \leq L||X||$,so from (C3),

$$\liminf_{n\to\infty} \frac{1}{t}\int_0^t ||X(s)||ds \geq \frac{1}{L}\left(s(A) - \frac{1}{2}\sigma_*^2\right) =: \varepsilon > 0 \quad \text{a.s.}$$

This is the desired persistence-in-the-mean (time-average bounded away from zero).

If $R_0^s < 1$: Strong noise or weak transmission → extinction almost surely.

If $R_0^s > 1$: Weak noise or strong transmission → persistence with fluctuations.

IV. Numerical illustration

In this study, we developed an age-structured SEIR malaria transmission model coupled with an SEI mosquito model, parameterized with biologically realistic values. The Newborn humans enter the susceptible class at a constant recruitment rateΛ_h , while mosquitoes are recruited at a constant rate $\Lambda_m = 10$ per day. Humans die at an age-dependent natural death rate $\mu_h(a) = 1/(70 \times 365)$ per day, representing an average life expectancy of 70 years, whereas mosquitoes die at a natural rate $\mu_m = 1/14$, corresponding to an average lifespan of two weeks. Transmission between humans and mosquitoes occurs through age-dependent rates: the mosquito-to-human transmission rate is denoted by $\beta_h(a)$ (0.25 in the deterministic model and 0.20 in the stochastic model), and the human-to-mosquito transmission coefficient by β_m (0.25 and 0.20, respectively). The force of infection for humans is given by $\lambda_h(a,t) = \beta_h(a)\frac{I_m(t)}{N_m(t)}$), depending on the proportion of infectious mosquitoes, while the mosquito force of infection is $\lambda_m(t) = \beta_m \frac{\int_0^\infty I(t,a)da}{N_h(t)}$, depending on the proportion of infectious humans. Disease progression and recovery dynamics are characterized by several age-dependent parameters: the rate at which exposed humans become infectious, $\kappa(a) = 1/10$per day; the human recovery rate, $\gamma(a) = 1/7$ per day; and the mosquito progression rate from exposed to infectious, $\delta = 1/10$ per day. Immunological processes include the vaccination or prophylaxis rate $\phi(a)$, waning of vaccine-induced immunity $\sigma(a)$, and waning of natural immunity $\omega(a)$, all assumed to be age-dependent. In the stochastic formulation, random environmental and demographic effects are incorporated through noise intensities: human compartments (S, E, I, R, V) are perturbed by white noise with intensity $\eta_i = 0.01$, while mosquito compartments (S_m, E_m, I_m)experience fluctuations with intensity $\xi_i = 0.05$. These stochastic perturbations capture natural variability and uncertainty in malaria transmission while maintaining biological realism in the system's dynamics.

Figure 2.compares the deterministic and stochastic age-structured malaria SEIR model outcomes across susceptible (S), exposed (E), infectious (I), and recovered (R) populations over time and age. The deterministic plots display smooth epidemic trajectories under high transmission conditions, showing that younger age groups (0–20 years) experience faster depletion of susceptibles, earlier and higher peaks in the exposed and infectious classes, and rapid accumulation in the recovered compartment. In contrast, the stochastic plots, which incorporate environmental noise and lower baseline transmission, exhibit fluctuating and dampened patterns: susceptible decline is irregular, exposed and infectious peaks are smaller and delayed, and recovery accumulates more gradually. Biologically, these results suggest that while deterministic dynamics predict consistent and intense outbreaks dominated by infections among children, the stochastic simulations reflect realistic environmental variability-where mosquito abundance, biting rates, and climate factors can suppress, delay, or even prevent outbreaks. Overall, the figure highlights both the age-dependent vulnerability of the population and the uncertainty introduced by environmental stochasticity in malaria transmission dynamics.

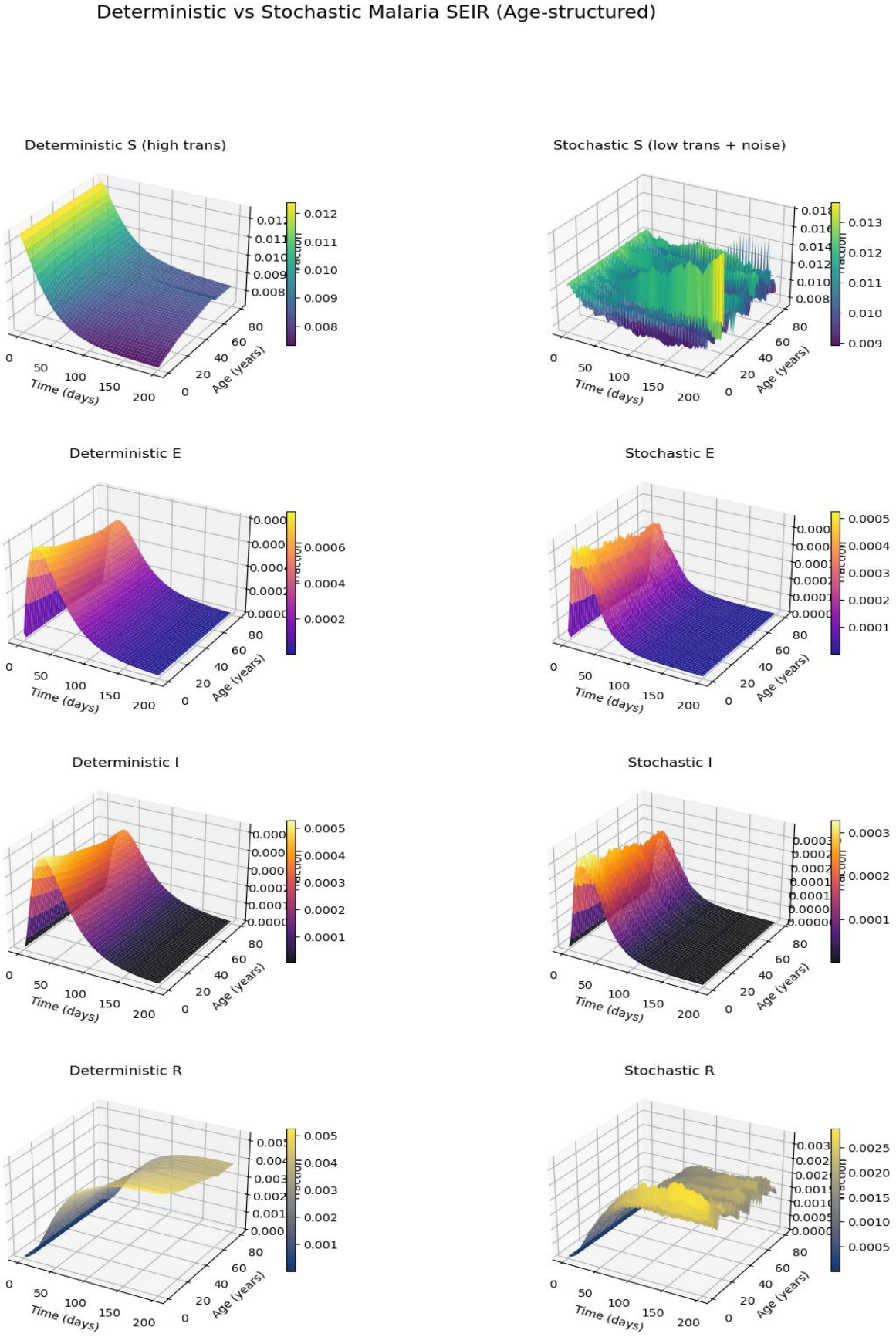
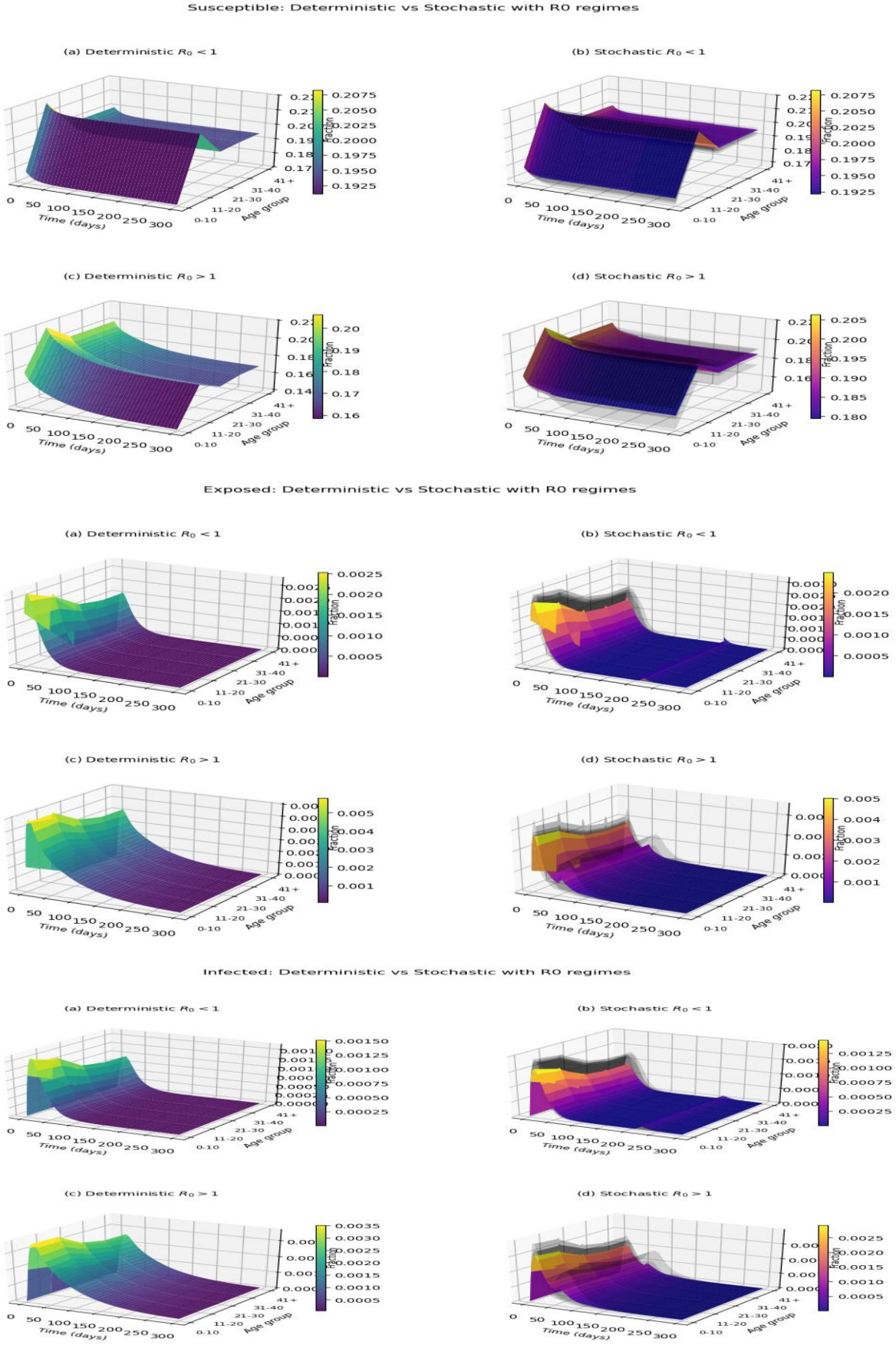


Fig.2.Deterministic vs Stochastic SEIR


Susceptible: Deterministic vs Stochastic with R0 regimes
(a) Deterministic $R_0 < 1$
(b) Stochastic $R_0 < 1$
(c) Deterministic $R_0 > 1$
(d) Stochastic $R_0 > 1$
Exposed: Deterministic vs Stochastic with R0 regimes
(a) Deterministic $R_0 < 1$
(b) Stochastic $R_0 < 1$
(c) Deterministic $R_0 > 1$
(d) Stochastic $R_0 > 1$
Infected: Deterministic vs Stochastic with R0 regimes
(a) Deterministic $R_0 < 1$
(b) Stochastic $R_0 < 1$
(c) Deterministic $R_0 > 1$
(d) Stochastic $R_0 > 1$
Time (days)
Age group
Fraction

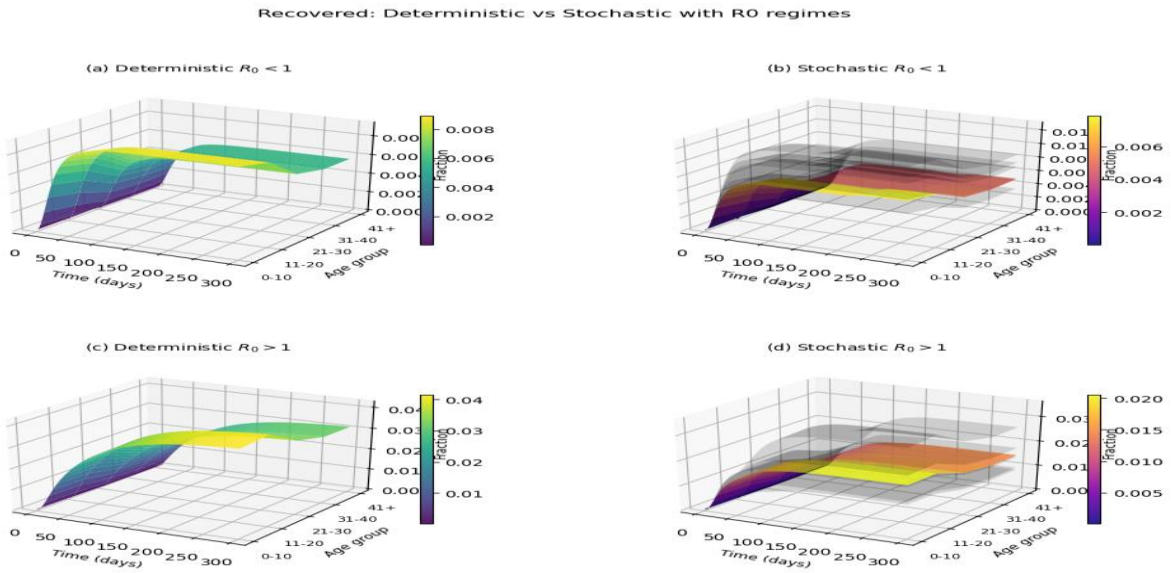


Fig.3.Reproduction number:deterministicvs stochastic

The figures 3.illustrate how the malaria epidemic dynamics differ under low ($R_0 < 1$) and high ($R_0 > 1$) transmission regimes for both deterministic and stochastic formulations of the age-structured SEIR model. When $R_0 < 1$, the disease fails to establish in the population: the susceptible fraction remains nearly constant, while the exposed, infected, and recovered compartments stay at negligible levels or decline over time. This indicates that transmission is insufficient to sustain infection, and both deterministic and stochastic simulations converge to disease-free equilibrium, though the stochastic version exhibits minor fluctuations due to random environmental effects.

In contrast, when $R_0 > 1$, sustained transmission occurs. The susceptible fraction decreases significantly over time-most notably among younger age groups due to higher susceptibility-while the exposed and infected compartments rise sharply before stabilizing or declining as recovery increases. The deterministic model produces smooth, well-defined epidemic waves, whereas the stochastic simulations reveal irregular and fluctuating patterns, capturing random variations in mosquito abundance, biting rates, and environmental noise that affect disease spread. The recovered fraction grows steadily in both regimes, with higher levels achieved when $R_0 > 1$, reflecting the buildup of immunity following infection.

V. Conclusion

This study demonstrates that incorporating both age structure and stochasticity into malaria transmission models significantly enhances our understanding of disease dynamics compared to traditional deterministic approaches. Deterministic models provide smooth epidemic trajectories that describe average long-term behaviour, while stochastic models capture fluctuations, randomness, and extinction possibilities that reflect real-world epidemiological uncertainty. Our analysis confirms that malaria persistence or elimination is governed by the basic reproduction number R_0, but stochastic variability can shift outcomes even when deterministic predictions suggest stability. Importantly, the model highlights the role of age-specific susceptibility, immunity waning, and vaccination in shaping transmission, emphasizing that interventions must be tailored to demographic heterogeneity. Overall, this combined theoretical–computational framework offers valuable tools for public health planning, enabling policymakers to anticipate different epidemic scenarios, evaluate intervention strategies and design more effective community-based malaria control programs.Total epidemic curves provide a visual tool to understand how malaria spreads and fluctuates across different age groups in a community. Deterministic models help identify the average expected epidemic burden, while stochastic models capture the uncertainty and variability present in real-world malaria transmission. By combining mathematical equations, simulation codes, and visual plots, this modelling framework can support public health decision-making, guide vaccination and vector-control strategies, and serve as a reproducible tool for researchers and policymakers to evaluate intervention outcomes under both predictable and uncertain conditions

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Author contributions

A.S.Talawar: suggestion of problem and methodology to be used.
Rathod J.M: All the derivations,Simulation study and preparation of manuscript.
Conflicts of interest:
The authors declare that they have no conflict of interest.

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