Mathematical Modeling of Human Mobility and Vector-Borne Disease Persistence: A Review

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This review aims to provide a comprehensive overview of the mathematical frameworks used to model disease dynamics, focusing on the underlying transmission mechanisms, key influencing factors, and the evaluation of potential control strategies as presented by the authors.

1 Introduction

Cosner et al. [1] examined how human movement between patches with varying vector densities influences the dynamics of vector—borne disease transmission. Cosner et al. employ a spatial meta—population framework inspired by spatial extensions of the classical Ross—Macdonald model. In this framework, space is modeled as a network of patches, each with a distinct vector population. The human population is divided into subgroups based on location, with individuals traveling from their home patch to others. The study adopts a hybrid modeling perspective that integrates both Lagrangian and Eulerian movement concepts — terms originally inspired by classical mechanics or fluid dynamics, though applied differently here. From this point onward, we use these same labels to distinguish between the two movement models in this thesis.

In both frameworks, individuals are categorized based on their place of residence or social group. The Lagrangian movement model tracks individuals based on their home patch or group, focusing on how they move between locations while maintaining a sense of origin. It assumes individuals spend most of their time in their home patch but visit others frequently enough to contribute to transmission elsewhere. It's like tracking labeled individuals and where they go – suitable for modeling commuting behavior. In contrast, the Eulerian movement model focuses on locations rather than individuals, tracking how many people are present in each patch at a given time, regardless of where they came from. It models movement explicitly, and is ideal for describing migration or redistribution across patches.

The disease dynamics follow a Susceptible-Infected-Recovery (SIR) compartmental

structure for humans and an Susceptible–Exposed–Infected (SEI) structure for vectors, with frequency–dependent transmission. Human and vector population sizes are held constant through death and replacement processes. The model is particularly relevant to diseases such as malaria and dengue, where mild or asymptomatic infections mean that human mobility is largely unaffected by infection status.

The primary goal is to understand how human mobility patterns influence disease persistence and spread across spatially heterogeneous environments and how interventions could be optimized under such mobility—driven transmission scenarios. The modeling framework of the study is divided into two parts: the single—patch model and the spatial models. Both Eulerian and Lagrangian approaches are incorporated into the spatial models. From this point forward, we will discuss the model framework used in the study.

2 Single-Patch Model

The model assumes that both the human and mosquito populations are constants, although the mosquito population undergoes turnover due to adult mortality. In some parts of the derivation, the study considered the possibility that the human and/or mosquito populations in each patch may vary due to movement of humans or mosquitoes. To this end, the following variables are introduced,

- N^H : total human population
- I^H : number of infected humans
- N^M : total mosquito population
- I^M : number of infected mosquitoes
- β : number of bites per mosquito per unit time on humans
- λ^{MH} : probability of transmission the disease to humans after bitten by an infected mosquito
- λ^{HM} : probability of transmission the disease to healthy mosquito from an infected human
- μ^M : the mosquito mortality rate, with deaths being offset by the emergence of new, healthy mosquitoes
- μ^H : the recovery rate of humans
- τ : the incubation period from the time a mosquito becomes infected until it becomes infectious

The model equations are given as

$$\frac{dI^H}{dt} = \beta I^M \lambda^{MH} e^{-\mu^M \tau} \frac{(N^H - I^H)}{N^H} - \mu^H I^H,$$

$$\frac{dI^M}{dt} = \beta \lambda^{HM} \frac{I^H (N^M - I^M)}{N^H} - \mu^M I^M$$

In vector–borne disease models, mosquitoes are typically assumed to have a finite expected lifespan. The rate of adult mosquito mortality (μ^M) is high enough that many infected mosquitoes will die before they have the opportunity to spread the infection to humans. Therefore, only a fraction of the initially infected mosquitoes will remain alive long enough to transmit the disease to humans, and this fraction is determined by the rate of mosquito mortality. Hence, the exponential decay factor $e^{-\mu^M\tau}$ captures the effect of mosquito mortality over time and determines the proportion of mosquitoes that remain infectious. That means, if the mortality rate is high, only few mosquitoes will survive to become infectious and transmit the disease.

However, to correctly account for the incubation period in mosquitoes, a delay should be incorporated in the human infection equation. Since mosquitoes are not infectious immediately after acquiring the infection, the mosquitoes that contribute to human infections at time t must have been infected at time $t-\tau$ or earlier, and must have survived until time t. Therefore, the number of infectious mosquitoes at time t should be more accurately calculated.

$$F(s) = \beta \lambda^{HM} \frac{I^{H}(s)(N^{M} - I^{M}(s))}{N^{H}}$$

This gives the rate of new mosquito infections occurring at time $s \in [0, t]$. Mosquitoes infected after time $t-\tau$ are still in the incubation phase and therefore not yet infectious. Then the number of infectious mosquitoes at time t can be calculated as

$$\int_0^{t-\tau} F(s) \cdot e^{-\mu^M(t-s)} \, ds$$

Thus, the correct system of differential equation, where the infection of humans depends on the *past* number of infected mosquitoes who have survived the incubation period:

$$\begin{split} \frac{dI^{H}(t)}{dt} &= \beta \left(\int_{-\infty}^{t-\tau} F(s) \cdot e^{-\mu^{M}(t-s)} \, ds \right) \lambda^{MH} \frac{(N^{H} - I^{H}(t))}{N^{H}} - \mu^{H} I^{H}(t) \\ \frac{dI^{M}(t)}{dt} &= \beta \lambda^{HM} \frac{I^{H}(t)(N^{M} - I^{M}(t))}{N^{H}} - \mu^{M} I^{M}(t). \end{split}$$

This formulation captures the fact that only mosquitoes infected τ time units ago and that have survived until now can contribute to new human infections. However, to remain consistent with the study by Cosner *et al.*, we do not incorporate a delay in

the following analysis, i.e the original model equations are valid under the assumption $\tau = 0$.

2.1 Spatial Models

The model considers space as a network of patches shared by both humans and mosquitoes, though their movement scales may differ. Humans typically move farther than mosquitoes, so their movement patterns operate on different spatial scales. To deal with movements, the models used coefficients to describe how many individuals (human or mosquito) move between patches, or how much time they spend in nonhome patches. These movement coefficients can be adjusted differently for humans and mosquitoes. For example, mosquito movements can be very small or zero since they do not travel far, but for human it can be larger to account their greater mobility. Section 3 of this thesis addresses the significant difference between the coefficients. Despite this different scales of movement between humans and mosquitoes, the study used the same patch network for both species because the focus of the study is on human movements and and its effect on the disease spread. Moreover, since mosquitoes do not move far, there is no need to track their every movement within human-defined patch (e.g. city). It is reasonable to sum up the overall behavior of mosquitoes in the patch, which makes the model simple without losing the accuracy at the human movement scale. However, this context considered as a special case and this might not be valid in other studies where mosquito movement matters more.

2.2 Lagrangian Approach: Patch Models with Commuting

The following assumptions are made in this version of the model,

- Individuals are labeled by their home patch and are assumed not to move permanently but visit other patches temporarily
- Infection risk is determined by the fraction of time individuals spend in each patch and the transmission rates in those patches.

The transmission rates in the model are defined by averaging across patches, where the contributions from each patch are weighted according to the fraction of time individuals spend there. Specifically,

- a_{ij} : the fraction of time a human resident in patch i spends in patch j,
- b_{ij} : the fraction of time a mosquito resident in patch i spends in patch j.

and

$$\sum_{i=1}^{n} a_{ij} = 1 \quad \text{and} \quad \sum_{i=1}^{n} b_{ij} = 1$$

where n is the total number of patches in the network. Define

$$A_{ij} = \beta_j \lambda_j^{MH} a_{ij} \frac{1}{N_j^H}, \quad B_{ij} = \beta_j \lambda_j^{HM} b_{ij} \frac{1}{N_j^H}.$$

 $A_{ij}I_i^M$ quantifies the rate at which a human from patch i spending time in patch j, gets infected by mosquitoes residing in patch j. $B_{ij}I_i^H$ quantifies the rate at which a mosquito from patch i, spending time in patch j, becomes infected by biting infected humans in patch j. Note that Cosner et al. [1] treat $\beta, \lambda^{HM}, \lambda^{MH}, \mu^{M}$ and μ^{H} as variables that depend on the patch when defining A_{ij} and B_{ij} . Treating parameters as patch-specific can be justified in spatial models, but doing so without explanation weakens the model's transparency and credibility. Since spatial heterogeneity is a key motivation for this modelling, it is critical to articulate where and why these heterogeneities are reflected in the parameters. Moreover, the study describes the term N_i^H as the human population of patch j when defining A_{ij} and B_{ij} which is incorrect. According to the model structure, individuals temporarily visit other patches; therefore, the number of humans present in patch j is not limited to the residents of that patch. In both cases, the term N_i^H should be interpreted as the effective human population present in patch j, which includes both residents and visitors. $a_{ij}N_i^H$ is given an expected number of people from patch i presents in patch j at any given time. Therefore, effective human population in patch j given by

$$n_j^{\text{eff}} = \sum_{i=1}^n a_{ij} N_i^H$$

The Lagrangian model then has the form

$$\frac{dI_{i}^{H}}{dt} = \left(\sum_{j=1}^{n} A_{ij} I_{j}^{M}\right) \left(N_{i}^{H} - I_{i}^{H}\right) - \mu_{i}^{H} I_{i}^{H}
\frac{dI_{i}^{M}}{dt} = \left(\sum_{j=1}^{n} B_{ij} I_{j}^{H}\right) \left(N_{i}^{M} - I_{i}^{M}\right) - \mu_{i}^{M} I_{i}^{M}, \quad i = 1, \dots, n$$
(1)

There are several criticisms about this model. The first term of the first equation $N_i^H - I_i^H$ assumes that all non-infected individuals are susceptible. However, this neglects the possibility of recovered individuals, contradicting the study's claim of following the Ross-Macdonald model, which traditionally distinguishes between susceptible, infected, and recovered classes. The same claim applies to the model presented in singlepatchmodel. Moreover the expression $\sum_{j=1}^n A_{ij}I_j^M = A_{i1}I_1^M + A_{i2}I_2^M + \cdots + A_{in}I_n^M$ represents the rate at which a human from patch i becomes infected while spending time in all patches, due to contact with infectious mosquitoes residing in those patches. Since both humans and mosquitoes are assumed to move between patches, I_j^M does

not represent infected mosquitoes present in patch j. To rectify this error, we introduce a new variable: the effective number of infected mosquitoes. For any patch i, this quantity depends on the number of infected mosquitoes that reside in patch i and remain there, as well as those that visit patch i from other patches. It captures the total infected mosquito presence in patch i, accounting for mosquito movement across the network.

$$m_j^{\text{eff}} = \sum_{i=1}^n b_{ij} I_i^M.$$

Therefore, the incorrect term I_j^M in the first equation should be replaced by m_j^{eff} . Additionally, while the first term of the second equation suggests that mosquitoes become infected from humans residing in patch j it implicitly assumes that infected humans are in their home patches. However, nothing in the model restricts the movement of infected humans. This inconsistency is an error, as not all humans stay at their home.

The set $\{(I_1^H,\ldots,I_n^H,I_1^M,\ldots,I_n^M):0\leq I_i^H\leq N_i^H,0\leq I_i^M\leq N_i^M,\,i=1,\ldots,n\}$ is invariant for the system described by the above equations. This means that the model's state space is bounded, ensuring that the number of infected humans I_i^H and infected mosquitoes I_i^M in each patch will always lie within the valid range: between 0 and the total population of humans N_i^H and mosquitoes N_i^M in that patch. Furthermore, the study assumed that the initial conditions satisfy $0\leq I_i^H(0)\leq N_i^H$ and $0\leq I_i^M(0)\leq N_i^M$ for all i, ensuring that the initial population of infected individuals in each patch is within the feasible limits.

2.3 Eulerian Approach: Patch Models with Migration

This model is based on the following assumptions,

- Individuals (humans or mosquitoes) migrate between patches and are not labeled by a home patch
- The infection rate depends solely on the current patch location, focusing on what happens within a patch rather than following individuals.

Under this assumption, the human and vector populations change over time, directly influencing the model. Therefore, it is assumed that these populations have reached the equilibrium predicted by the migration rates, at least in relation to the time scale over which the system is being studied. For that purpose, the study defined:

- C_{ij} : the rate of human migration from patch j to i
- D_{ij} : the rate of vector migration from patch j to i

The migration of humans and mosquitoes are in the form

$$\frac{dN_i^H}{dt} = \sum_{\substack{j=1\\j\neq i}}^n C_{ij} N_j^H - \left(\sum_{\substack{j=1\\j\neq i}}^n C_{ji}\right) N_i^H
\frac{dN_i^M}{dt} = \sum_{\substack{j=1\\j\neq i}}^n D_{ij} N_j^M - \left(\sum_{\substack{j=1\\j\neq i}}^n D_{ji}\right) N_i^M, \quad i = 1, \dots, n$$

However, the study refers to this as discrete diffusion, which can be contextually ambiguous. In mathematics, diffusion typically refers to random, undirected movement, where individuals spread out due to local random walks. This process is modeled by the Laplacian operator and leads to second—order partial differential equations. In contrast, the terms used in the model represent directed migration between patches and do not arise from spatial gradients or random movement. Therefore, we consider the use of the term diffusion in this context to be a misinterpretation.

By expanding the original summations to include all $j = 1, 2, \dots, n$ and defining leaving patch i by negative diagonal entries as

$$C_{ii} = -\sum_{\substack{j=1\\j\neq i}} C_{ji}, \quad D_{ii} = -\sum_{\substack{j=1\\j\neq i}} D_{ji}, \quad i = 1, \dots, n$$

we can absorb the total outflow from each patch and obtain the compact matrix form

$$\frac{d\mathbf{H}}{dt} = \mathbf{C}\mathbf{H},
\frac{d\mathbf{V}}{dt} = \mathbf{D}\mathbf{V}.$$
(2)

where $C = (C_{ij}), D = (D_{ij}), H = (N_1^H, \dots, N_n^H)^T$ and $V = (N_1^M, \dots, N_n^M)^T$. Using

$$N^H = \sum_{i=1}^n N_i^H, \quad N^M = \sum_{i=1}^n N_i^M$$

and summing up for all i, it is clear that the total human population N^H and mosquito population N^M is conserved over time. It is clear that matrices \mathbf{C} and \mathbf{D} have negative diagonal and non-negative off-diagonal elements (Metzler matrix) with column sum zero. Using that property we can say that $\vec{\mathbf{I}}^T\mathbf{C} = 0$ which gives zero is an eigenvalue of \mathbf{C} and $\vec{\mathbf{I}}$ is the related left eigenvector. Analogously, \mathbf{D} has 0 as an eigenvalue. To make \mathbf{C} a non-negative matrices, choose $c_0 = \max_i |C_{ii}|$ and define a new matrix which is assumed to be irreducible with non-negative diagonal,

$$\tilde{\boldsymbol{C}} = \boldsymbol{C} + c_0 \boldsymbol{I}$$

Now, $\tilde{C}_{ij} \geq 0$, irreducible with non-negative entries and hence it is allowed to apply the Perron-Frobenius theorem. Hence, \tilde{C} has a dominant eigenvalue $\tilde{\lambda}_1$ which is real and positive with positive eigenvector $\tilde{v}_1 > 0$ where all other eigenvalues have real parts smaller than $\tilde{\lambda}_1$. Since, $\tilde{C} = C + c_0 I$ all eigenvalues λ_i of C are related to those of \tilde{C} via

$$\lambda_i = \tilde{\lambda}_i - c_0$$

As we mentioned earlier $(1, \dots, 1)$ is a left eigenvector of \mathbf{C} of eigenvalue 0 and hence it is a left eigenvector for $\tilde{\mathbf{C}}$ corresponding to the eigenvalue c_0 . Therefore, $\tilde{\lambda}_1 \geq c_0$. The equality holds when $\lambda_1 = 0$. When $\tilde{\lambda}_i \neq \tilde{\lambda}_1$, $Re(\tilde{\lambda}_i) < Re(\tilde{\lambda}_1) = c_0$ which yields, $Re(\lambda_i) = Re(\tilde{\lambda}_i - c_0) < 0$ which implies that the rest eigenvalue of \mathbf{C} are negative. Therefore, we can conclude that 0 is the dominant eigenvalue of \mathbf{C} which has a positive eigenvector. Suppose the initial condition $(N_1^H(0), \dots, N_n^H(0))$ has some positive components and the matrix \mathbf{C} has the normalized right eigenvector $(N_1^{H*}, \dots, N_n^{H*})$ corresponding to the eigenvalue 0 such that,

$$\sum_{j=1}^{n} C_{ij} N_j^{H*} = 0 \quad \sum_{j=1}^{n} N_j^{H*} = N^H.$$

All eigenvalues of matrix C other than the dominant one have negative real parts, their corresponding modes decay exponentially as time progresses. Therefore, starting from any initial condition with some positive components, the system asymptotically aligns with eigenvector $(N_1^{H*}, \cdots, N_n^{H*})$ as $t \to \infty$. Analogously, for non zero initial condition $(N_1^M(0), \cdots, N_n^M(0))$ over time the system evolves toward the right eigenvector $(N_1^{M*}, \cdots, N_n^{M*})$ of matrix D corresponding to the eigenvalue 0.

The study assumed that the migration process has stabilized, meaning that while individuals may move between patches, the total human and vector populations in each patch remain unchanged. Thus, $N_i^H(t) = N_i^{H*}$ and $N_i^M(t) = N_i^{M*}$. Again, the disease transmission happens only between individuals when they are in the same patch. Let

$$A_i = \beta_i \lambda_i^{MH} \frac{1}{N_i^{H*}}, \quad B_i = \beta_i \lambda_i^{HM} \frac{1}{N_i^{H*}}.$$

Then the Eulerian model becomes,

$$\frac{dI_i^H}{dt} = A_i I_i^M \left(N_i^{H*} - I_i^H \right) - \mu_i^H I_i^H + \sum_{i=1}^n C_{ij} I_j^H,
\frac{dI_i^M}{dt} = B_i I_i^H \left(N_i^{M*} - I_i^M \right) - \mu_i^M I_i^M + \sum_{i=1}^n D_{ij} I_j^M, \quad i = 1, \dots, n$$
(3)

Again it is clear that the set $\{(I_1^H,\ldots,I_n^H,I_1^M,\ldots,I_n^M):0\leq I_i^H\leq N_i^{H*},0\leq I_i^M\leq N_i^{H*}\}$

 $N_i^{M*}, i=1,\ldots,n\}$ is invariant for the system described by above equations and it is assumed $0 \leq I_i^H(0) \leq N_i^{H*}$ and $0 \leq I_i^M(0) \leq N_i^{M*}$ for all i.

2.4 R_0 of the Models

In epidemiological models it is important to examine whether the disease free equilibrium $(I_i^H = I_i^M = 0 \text{ for } i = 1, ..., n)$ is stable upon disease introduction, and to compute the corresponding basic reproduction number.

2.4.1 R_0 of Lagrangian Model

By linearizing the (1) near DFE

$$\frac{d\mathbf{X}}{dt} \approx \mathbf{A}^* \mathbf{Y} - R\mathbf{X}$$
$$\frac{d\mathbf{Y}}{dt} \approx \mathbf{B}^* \mathbf{X} - M\mathbf{Y}$$

where, $\mathbf{X} = (I_1^H, \dots, I_N^H)$, $\mathbf{Y} = (I_1^M, \dots, I_N^M)$, $\mathbf{A}^* = (A_{ij}I_i^M)$, $\mathbf{B}^* = (B_{ij}I_i^H)$, $R = diag(\mu_i^H)$, $M = diag(\mu_i^M)$. Then the system of equations can be written as

$$\frac{d}{dt} \begin{pmatrix} \mathbf{X} \\ \mathbf{Y} \end{pmatrix} = \left(\underbrace{\begin{pmatrix} 0 & \mathbf{A}^* \\ \mathbf{B}^* & 0 \end{pmatrix}}_{F} - \underbrace{\begin{pmatrix} R & 0 \\ 0 & M \end{pmatrix}}_{T} \right) \begin{pmatrix} \mathbf{X} \\ \mathbf{Y} \end{pmatrix}$$

where \boldsymbol{F} gives new infections and \boldsymbol{T} gives transitions between status. Then the NGM \boldsymbol{K} (refer NGM) can be written as,

$$\boldsymbol{K} = \boldsymbol{F}\boldsymbol{T}^{-1} = \begin{pmatrix} 0 & \mathbf{A}^* \\ \mathbf{B}^* & 0 \end{pmatrix} \times \begin{pmatrix} R^{-1} & 0 \\ 0 & M^{-1} \end{pmatrix} = \begin{pmatrix} 0 & \mathbf{A}^*M^{-1} \\ \mathbf{B}^*R^{-1} & 0 \end{pmatrix}$$

Then the basic reproduction number can be written as,

$$R_0 = \rho(\mathbf{A}^* M^{-1} \mathbf{B}^* R^{-1})$$

The Next Generation Theorem implies $R_0 < 1 \Leftrightarrow \lambda_{(F-T)} < 0$ where $\lambda_{(F-T)} = \max(Re(\lambda_i))$ where λ_i is an eigenvalue of matrix (F - T). That means the DFE is stable when $R_0 < 1$. Conversely, if $R_0 > 1$ then the DFE is unstable.

Since we are focusing on the conditions under the which the disease could spread exponentially from low initial, we don't need to pay attention whether there exists an equilibrium if the DFE is unstable. There may be an equilibrium, a limit cycle, or even chaotic dynamics when DFE is unstable.

Lajmanovich and Yorke [2] established that if the disease–free equilibrium is un-

stable, there exists a unique positive equilibrium – satisfying $0 < I_i^{H*} < I_i^H$ and $0 < I_i^{M*} < I_i^M$ for all i – which is globally stable among positive solutions in their study of a gonorrhea model for non–homogeneous populations. Hasibeder and Dye [3] extended this result to vector–borne disease settings, and Cosner $et\ al.$ cited their work for this conclusion. However, checking the uniqueness or existence of this equilibrium is not our interest in this context. Therefore we omit that part.

2.4.2 R_0 of Eularian Model

In similar manner, by linearizing (3) near DFE

$$\frac{d\mathbf{X}}{dt} \approx \mathbf{A}^{**}\mathbf{Y} - \mathbf{C}^{**}\mathbf{X}$$
$$\frac{d\mathbf{Y}}{dt} \approx \mathbf{B}^{**}\mathbf{X} - \mathbf{D}^{**}\mathbf{Y}$$

where, $\mathbf{A}^{**} = (A_i I_i^M \delta_{ij})$ $\mathbf{B}^{**} = (B_i I_i^H \delta_{ij})$ $\mathbf{C}^{**} = (C_{ij} - \mu_i^H \delta_{ij})$ $\mathbf{D}^{**} = (D_{ij} - \mu_i^M \delta_{ij})$ where, δ_{ij} is the Kronecker delta. Then,

$$\frac{d}{dt} \begin{pmatrix} \mathbf{X} \\ \mathbf{Y} \end{pmatrix} = \left(\underbrace{\begin{pmatrix} 0 & \mathbf{A}^{**} \\ \mathbf{B}^{**} & 0 \end{pmatrix}}_{F} - \underbrace{\begin{pmatrix} -\mathbf{C}^{**} & 0 \\ 0 & -\mathbf{D}^{**} \end{pmatrix}}_{T} \right) \begin{pmatrix} \mathbf{X} \\ \mathbf{Y} \end{pmatrix}$$

and

$$\boldsymbol{K} = \boldsymbol{F}\boldsymbol{T}^{-1} = \begin{pmatrix} 0 & \mathbf{A}^*(-\mathbf{C}^{**-1}) \\ \mathbf{B}^*(-\mathbf{D}^{**-1}) & 0 \end{pmatrix}$$

hence,

$$R_0 = \rho(\mathbf{A}^{**}(-\mathbf{D}^{**-1})\mathbf{B}^{**}(-\mathbf{C}^{**-1}))$$

But this is only true when \mathbf{C}^{**} and \mathbf{D}^{**} are invertible and have eigenvalues with strictly negative real parts. This is because, in the absence of new infections – i.e., with only migration and recovery/death – the system should decay to zero as time tends to infinity. It is not straightforward to decide whether these matrices invertible or not. Furthermore, it is clear that \mathbf{C}^{**} and \mathbf{D}^{**} have negative diagonal elements and nonnegative off diagonal elements and hence by definition these are Metzler matrices.

Also, we defined elements of matrices C and D such that the column sum to be equal to zero and together with the definitions of C^{**} and D^{**} we can see that the absolute value of the diagonal entry of each row of C^{**} and D^{**} are greater than the sum of the magnitudes of off-diagonal elements

$$|(\mathbf{C}^{**})_{ii}| > \sum_{j \neq i} (\mathbf{C}^{**})_{ij}, \quad |(\mathbf{D}^{**})_{ii}| > \sum_{j \neq i} (\mathbf{D}^{**})_{ij}.$$

Hence, \mathbf{C}^{**} and \mathbf{D}^{**} are strictly diagonally dominant matrices with negative diagonals. At this point we can use *Gershgorin's Circle Theorem* to conclude the invertibility of \mathbf{C}^{**} and \mathbf{D}^{**} . According to the above–said theorem, every eigenvalue of the square matrix $\mathbf{C}^{**} = (C_{ij} - \mu_i^H \delta_{ij})$ lies within at least one Gershgorin disc in the complex plane. The disc centered at $(\mathbf{C}^{**})_{ii}$ has radius $\sum_{j\neq i} (\mathbf{C}^{**})_{ij}$. And as every $(\mathbf{C}^{**})_{ii}$ is negative and the matrix \mathbf{C}^{**} is strictly diagonal dominant, the rightmost boundary of any disc is $(\mathbf{C}^{**})_{ii} + \sum_{j\neq i} (\mathbf{C}^{**})_{ij} < 0$. Therefore, every Gershgorin disc lies entirely in the open left–half of the complex plane – i.e., all eigenvalues of \mathbf{C}^{**} have strictly negative real parts. This follows that the matrix $-\mathbf{C}^{**}$ has all eigenvalues with positive real parts and hence it is invertible. Analogously, $-\mathbf{D}^{**}$ is invertible. Therefore, we can conclude that the R_0 is well–defined and thus can be computed.

Moreover, unlike in the Lagrangian approach, the effects of μ_i^H and μ_i^M on R_0 are not straightforward to determine analytically. However, an intuitive understanding can still be drawn. When the human recovery rate μ_i^H increases, individuals recover more quickly, reducing the time they remain infectious. As a result, the number of secondary infections caused by each infectious individual decreases, making it harder for the disease to persist in the population. Similarly, when the mosquito death rate μ_i^M increases, mosquitoes have a shorter lifespan, limiting the time available for them to infect humans. This also contributes to a lower potential for disease spread and hence the disease will die out eventually. Thus, an increase in either of these rates reduces the number of secondary infections, thereby decreasing the R_0 and hence the ability of the disease to sustain transmission across generations. However, we can explain this effect using properties of matrices.

- increase of μ_i^H/μ_i^M making diagonal entry of $\mathbf{C}^{**}/\mathbf{D}^{**}$ more negative
- then $-\mathbf{C}^{**}/-\mathbf{D}^{**}$ becomes larger on the diagonal which makes the matrix more strongly diagonally dominant and hence, reduces the magnitude of the entries in its inverse.

Therefore $\rho(\mathbf{A}^{**}(-\mathbf{D}^{**-1})\mathbf{B}^{**}(-\mathbf{C}^{**-1}))$ monotonically decreasing with increasing of μ_i^H . Similarly, they are also monotone decreasing with respect to the mortality rates μ_i^M as well.

3 Two-patch models with no transmission in one patch

Malaria transmission is highly localized due to limited movement range of mosquitoes – typically within a few hundred meters to 2–3 km – and tends to cluster around specific breeding sites and households. To study how the human movement can sustain infection in areas without local transmission, a two–patch model is considered, where only patch

1 has vector-borne transmission. Patch 2 has no mosquitoes and hence, $I_2^M = 0$ and no mosquito movement occurs between patches, but human can move between them. This set up helps investigate whether infection can persist in a non-transmitting patch purely through human mobility, highlighting the role of spatial structure and movement in maintaining malaria transmission.

3.1 The Eulerian model

The relevant model and R_0 are as follows.

$$\frac{dI_1^H}{dt} = A_1 I_1^M (N_1^{H*} - I_1^H) - \mu_1^H I_1^H + C_{12} I_2^H - C_{21} I_1^H
\frac{dI_2^H}{dt} = C_{21} I_1^H - C_{12} I_2^M - \mu_2^H I_2^H
\frac{dI_1^M}{dt} = B_1 I_1^H (N_1^{M*} - I_1^M) - \mu_1^M I_1^M$$
(4)

In this two–patch Eulerian framework, to calculate the expected number of secondary infections generated by a single infectious individual in a fully susceptible population, we can use simple bookkeeping as in previous sections.

Due to the model's assumptions, only individuals located in patch 1 – until their recovery – have the potential to contribute to R_0 . Each infected mosquito in patch 1 lives for an average of $\frac{1}{\mu_1^M}$ days and can produces $\frac{A_1N_1^{M*}}{\mu_1^M}$ new human infections during its lifetime in all susceptible population. Once a human becomes infected, several possibilities arise: they may remain in patch 1 until recovery, relocate to patch 2 and recover there, or migrate to patch 2 and subsequently return to patch 1 before recovering – possibly repeating this cycle multiple times. Therefore, the expected total time an infected human spends in patch 1 before recovery is a crucial component in determining R_0 . Let us define

 T_1 = expected time an infected human spends in patch 1 before recovery

and calculate T_1 using,

$$T_1 = \frac{1}{C_{21} + \mu_1^H} + \frac{C_{21}}{C_{21} + \mu_1^H} \cdot \frac{C_{12}}{C_{12} + \mu_2^H} T_1.$$

By solving this T_1 can be obtained as

$$\frac{C_{12} + \mu_2^H}{C_{12}\mu_1^H + C_{21}\mu_2^H + \mu_1^H\mu_2^H}$$

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Then the basic reproduction number can be obtained as

$$R_0 = \frac{A_1 N_1^{M*} B_1 N_1^{H*}}{\mu_1^M} \cdot \frac{C_{12} + \mu_2^H}{C_{12} \mu_1^H + C_{21} \mu_2^H + \mu_1^H \mu_2^H}$$

In a more practical sense, we observe that as the rate of movement from patch 1 to patch 2 increases, the value of R_0 decreases – R_0 is a decreasing function of C_{21} . This is because individuals leave the infectious environment more frequently, reducing their exposure time to infected vectors. As a result, even if $R_0 > 1$ in patch 1 in isolation, high migration away from the transmission patch can reduce the overall effective reproduction number and potentially eliminate the disease.

Whenever $R_0 > 1$, the system has a positive equilibrium as follows:

$$I_{2}^{H*} = \frac{C_{21}I_{1}^{H*}}{C_{12} + \mu_{2}^{H}},$$

$$I^{M*} = \frac{B_{1}N_{1}^{M*}I_{1}^{H*}}{B_{1}I_{1}^{H*} + \mu_{1}^{M}},$$

$$I_{1}^{H*} = \frac{A_{1}B_{1}N^{M*}N^{H*} - Q\mu_{1}^{M}}{B_{1}(A_{1}N^{M*} + Q)} = \frac{(R_{0} - 1)Q\mu_{1}^{M}}{B_{1}(A_{1}N^{M*} + Q)},$$
(5)

where
$$Q = \frac{C_{12}\mu_1^H + C_{21}\mu_2^H + \mu_1^H\mu_2^H}{C_{12} + \mu_2^H}$$
.

In this situation the equilibrium value I_2^{H*} remains positive, indicating that human mobility can sustain the disease in the patch without mosquitoes. This outcome does not depend on the specific values of human migration rates.

4 Conclusion of the paper

The analysis of two-patch models where pathogen transmission occurs in only one patch shows that the disease can persist in the patch without local transmission only if $R_0 > 1$. Infected individuals moving from the transmitting patch can carry the pathogen to non-transmitting patch, leading to nonzero number of infections there. However, increased migration from the transmitting patch to non-transmitting patch reducing the value of R_0 . Thus, while human movements enables spatial spread, it can also dilute the transmission potential, highlighting the nuanced role of mobility in sustaining vector-borne diseases. This helps explain why infections can appear in areas with low mosquito density, but also why excessive movement might limit long-term persistence of the disease.

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