Vector–Borne Epidemics Driven by Human Mobility: 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

Since this thesis is inspired by the work of Soriano–Paños *et al.* [1], we briefly outline their approach here. The primary objective of their study was to investigate large–scale vector–borne epidemics by integrating available data on vector incidence with human demographic and mobility information. To achieve this, they employed a discrete–time meta–population framework and validated their findings using real–world data from the city of Cali, Colombia.

The model considers the proportions of infected humans and vectors at time t, denoted by $\rho^H(t)$ and $\rho^M(t)$, respectively. Susceptible humans become infected with probability λ^{MH} upon being bitten by an infected mosquito, while susceptible mosquitoes become infectious with probability λ^{HM} after biting an infected human. Each mosquito is assumed to have β number of contacts with humans regardless of the health status of those humans. Thus, the model excludes any direct human–to–human or mosquito–to–mosquito transmission.

Infected humans recover and return to the susceptible class with probability μ^H, while mosquitoes (regardless of infection status) die with probability μ^M and are immediately replaced by healthy adult mosquito. Given the limited mobility of vectors by nature, the model assumes that mosquitoes remain confined to their local patch, while humans can move between patches according to a predefined mobility matrix $\boldsymbol{R}$. Each patch i hosts N_i^H humans and N_i^M mosquitoes, and the entire system is composed of n distinct meta–population patches. The contagion process align with the classical Ross–Macdonald framework, while incorporating human mobility as the key

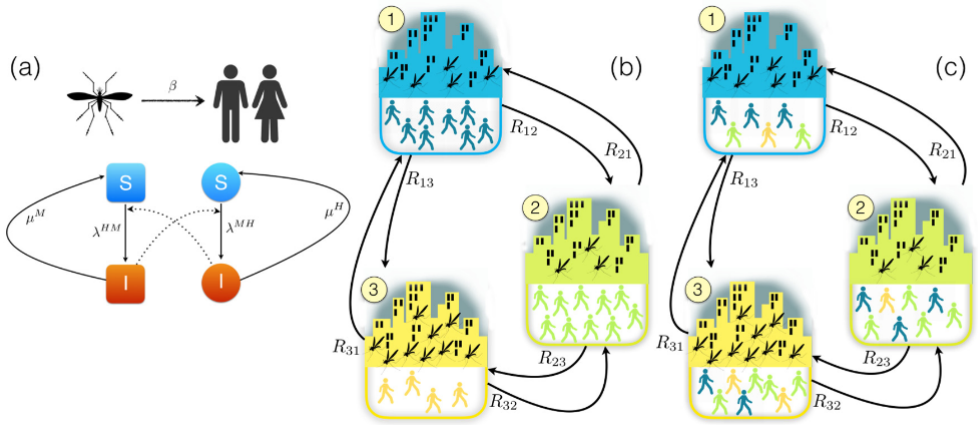

Figure 1: Ross–Macdonald model and meta–population approach extracted from [1]. a) Schematic representation of the processes described in the RM model. (b) Humans and mosquitoes inhabit three distinct population patches, and they move between these patches based on probabilities R_{ij}. Different colors are used to easily distinguish individuals based on their home patch.(c) Once individuals have mixed across the patches, each patch contains humans from various resident patches. The interactions between humans and mosquitoes then follow the Ross–Macdonald model. Afterward, all humans return to their original patches, and the cycle repeats.

driver of spatial disease spread. It is therefore assumed that human movement is the main mechanism through which the disease propagates across the system.

In the model healthy humans leave their home patch with probability p, while infected individuals do so with probability αp, where $\alpha \in [0, 1]$ is the mobility correction factor accounting for reduced mobility due to infection. When an individual leaves their home patch i, they visit another patch j with probability R_{ij}, as defined by the human mobility matrix $\boldsymbol{R}$. After all movement occurs at time t, disease transmission dynamics within each patch follow the Ross–Macdonald model. Both human and vector states (susceptible or infected) are updated according to these interactions. Once the status updates are complete, all individuals return to their home patches, and the process repeats at the next time step, $t + 1$.

2 Model Equations

This modeling framework is illustrated in Figure 1. The authors formulated two coupled Markovian equations to iteratively update the state of the system over time:

$$\begin{aligned} \rho_i^H(t+1) &= \rho_i^H(t)(1-\mu^H) + \left(1-\rho_i^H(t)\right) P_i^H(t) \\ \rho_i^M(t+1) &= \rho_i^M(t)(1-\mu^M) + \left(1-\rho_i^M(t)\right) P_i^M(t) \end{aligned} \tag{1}$$

where $P_i^H(t)$ and $P_i^M(t)$ represent the probabilities that a susceptible human residing in meta–population i, and a susceptible vector associated with patch i, become infected

at time t, respectively. And, the former probability is

$$P_i^H(t) = (1-p)Q_i^H(t) + p\sum_{j=1}^{n} R_{ij}Q_j^H(t) \tag{2}$$

where $Q_i^H(t)$ is the probability that a human located in patch i at time t gets infected, and is given by

$$Q_i^H(t) = 1 - \left(1 - \frac{\lambda^{MH}\rho_i^M}{n_i^{\text{eff}}(\rho^H(t),\alpha,p)}\right)^{\beta N_i^M}. \tag{3}$$

Here $n_i^{\text{eff}}(\rho^H(t),\alpha,p)$ represents the effective number of humans present in subpopulation i, regardless of their place of residence. However, it can be observed that equation (3) is mathematically incorrect and dimensionally inconsistent. Given that the model assumes a deterministic setting, the values of N_i^M and $n_i^{\text{eff}}(\rho^H(t),\alpha,p)$ are typically large and are expected to be measured in the same units. Therefore, the equation does not yield a probability, which should be a dimensionless real number less than 1 ($\frac{\lambda^{MH}\rho_i^M}{n_i^{\text{eff}}(\rho^H(t),\alpha,p)}$ and βN_i^M are both not unit less). The issues related to this equation will be discussed further in section **??**.
The effective human population in patch i is given by

$$n_i^{\text{eff}}(\rho^H(t),\alpha,p) = \left[1 - p\left(1-(1-\alpha)\rho_i^H(t)\right)\right]N_i^H + p\sum_{j=1}^{n} R_{ji}\left(1-(1-\alpha)\rho_j^H(t)\right)N_j^H. \tag{4}$$

Analogously, the probability that a susceptible mosquito in patch i becomes infected is

$$P_i^M(t) = 1 - \left(1 - \lambda_{HM}\frac{i_i^{\text{eff}}(t)}{n_i^{\text{eff}}}\right)^{\beta} \tag{5}$$

where $i_i^{\text{eff}}(t)$ represents the number of infected humans present in population i at time t, and is gives by

$$i_i^{\text{eff}}(t) = (1-\alpha p)N_i^H\rho_i^H(t) + \alpha p\sum_{j=1}^{n} R_{ji}N_j^H\rho_j^H(t). \tag{6}$$

This set of equations describes how infection levels evolve over time – i.e., how the disease either spreads throughout the system or fades out. The vectors $\vec{\rho}^H(t)$ and $\vec{\rho}^M(t)$ represent the fraction of infected humans and mosquitoes, respectively, in each patch at time t. By initializing the system with $\vec{\rho}^H(0)$ and $\vec{\rho}^M(0)$, and iteratively applying equation 1, the authors were able to track the spatiotemporal progression of the vector–borne disease and identify its possible steady states.

Furthermore, the authors examined a scenario in which human mobility is structured by an unweighted, undirected Barabási–Albert network consisting of $n = 50$ patches, with an average degree $\langle k \rangle = 4$. All patches were assumed to be homogeneously populated, each with $N_i^H = 1000$ human agents. The ratio of vectors to humans in patch i was denoted by γ_i with values of γ_i drawn from a uniform distribution within the range $[0.3, 1.7]$.

3 Analysis of the Model

To understand the conditions under which an epidemic emerges, it is crucial to analyze the behavior of the epidemic threshold. In mathematical terms, this threshold corresponds to the point where the disease–free equilibrium loses stability and a non–zero infected equilibrium becomes stable. To investigate this, the study consider an early early stage of the epidemic where the prevalence of infection is very small, $\rho_i^H = \epsilon_i^H \ll 1$ and $\rho_i^M = \epsilon_i^M \ll 1$ for all i. In this regime system can be linearized near DFE to assess whether small perturbations grow or decay over time.

For this purpose, the study analyzed the system under the assumption that the infection levels evolve slowly in time, i.e.,

$$\rho_i^H(t+1) \approx \rho_i^H(t), \quad \rho_i^M(t+1) \approx \rho_i^M(t)$$

not because the system has reached an equilibrium, but because this quasi–static assumption simplifies the linearized system. Furthermore, this step removes the time dependence and simplifies the system into steady–state equations. Under this condition, equation 1 reduces to

$$\begin{aligned} \mu^H \rho_i^H &= (1 - \rho_i^H)\, P_i^H \\ \mu^M \rho_i^M &= (1 - \rho_i^M)\, P_i^M. \end{aligned} \tag{7}$$

Now, the system of equations 7 can be linearized, leading to a simplified set of equations suitable for analyzing the epidemic threshold as follows. Let us consider the first equation of 7 first.

Due to the assumption $(1 - \rho_i^H) \approx 1$ and then

$$\epsilon_i^H = \frac{1}{\mu^H} \left((1-p) Q_i^H(t) + p \sum_{j=1}^{n} R_{ij} Q_j^H(t) \right)$$

The term $n_i^{\text{eff}}(\rho^H(t), \alpha, p)$ is very large number and hence, the quantity inside the parentheses of equation 3 is close to 1. Also, βN_i^M is a large number and therefore, we

can approximate the value of Q_i^H using using a binomial expansion.

$$Q_i^H(t) = 1 - \left(1 - \lambda^{MH}\beta N_i^M \epsilon_i^H \frac{1}{\tilde{n}_i^{\text{eff}}} + H.O.T\right) \approx \lambda^{MH}\beta N_i^M \epsilon_i^H \frac{1}{\tilde{n}_i^{\text{eff}}}$$

Therefore

$$\begin{aligned}
\epsilon_i^H &= \frac{1}{\mu^H}\left((1-p)\,\lambda^{MH}\beta N_i^M \epsilon_i^H \frac{1}{\tilde{n}_i^{\text{eff}}} + p\sum_{j=1}^{n} R_{ij}\lambda^{MH}\beta N_j^M \epsilon_j^H \frac{1}{\tilde{n}_j^{\text{eff}}}\right) \\
&= \frac{\lambda^{MH}\beta}{\mu^H}\left((1-p)\,N_i^M \epsilon_i^H \frac{1}{\tilde{n}_i^{\text{eff}}} + p\sum_{j=1}^{n} R_{ij} N_j^M \epsilon_j^H \frac{1}{\tilde{n}_j^{\text{eff}}}\right) \\
&= \sum_{j=1}^{n} \frac{\lambda^{MH}\beta}{\mu^H}\left(p\,R_{ij}\frac{N_j^M}{\tilde{n}_j^{\text{eff}}} + (1-p)\,\delta_{ij}\frac{N_i^M}{\tilde{n}_i^{\text{eff}}}\right)\epsilon_j^H
\end{aligned}$$

Analogously we can derive the linear system for ϵ_i^M and both yield

$$\begin{aligned}
\epsilon_i^H &= \sum_{j=1}^{n} \frac{\lambda^{MH}\beta}{\mu^H} \underbrace{\left(pR_{ij}\frac{N_j^M}{\tilde{n}_j^{\text{eff}}} + (1-p)\delta_{ij}\frac{N_i^M}{\tilde{n}_i^{\text{eff}}}\right)}_{\boldsymbol{M}_{ij}} \epsilon_j^M \\
\epsilon_i^M &= \sum_{j=1}^{n} \frac{\lambda^{HM}\beta}{\mu^M} \underbrace{\left(\alpha p R_{ji}\frac{N_j^H}{\tilde{n}_i^{\text{eff}}} + (1-\alpha p)\delta_{ij}\frac{N_i^H}{\tilde{n}_i^{\text{eff}}}\right)}_{\tilde{\boldsymbol{M}}_{ij}} \epsilon_j^H
\end{aligned} \tag{8}$$

where

$$\tilde{n}_i^{\text{eff}} = n_i^{\text{eff}}(0, \alpha, p)$$

which denotes the effective number of humans in patch i under the assumptions of low disease prevalence and given mobility parameters and δ_{ij} is Kronecker delta where

$$\delta_{ij} = \begin{cases} 1, & \text{if } i = j, \\ 0, & \text{if } i \neq j. \end{cases}$$

The matrices $\boldsymbol{M}$ and $\tilde{\boldsymbol{M}}$ represent the transmissions from vectors to humans and from humans to vectors, respectively. Both $\boldsymbol{M}$ and $\tilde{\boldsymbol{M}}$ are functions of the mobility probability p, the mobility matrix $\boldsymbol{R}$, and the population distributions $\vec{N^H}$ (humans) and $\vec{N^M}$ (mosquitoes). Therefore, equation 8 can be rewritten accordingly.

$$\begin{pmatrix} \vec{\boldsymbol{\epsilon}}^{*H} \\ \vec{\boldsymbol{\epsilon}}^{*M} \end{pmatrix} = \begin{pmatrix} 0 & \dfrac{\beta\lambda^{MH}}{\mu^H}\boldsymbol{M} \\ \dfrac{\beta\lambda^{HM}}{\mu^M}\tilde{\boldsymbol{M}} & 0 \end{pmatrix} \begin{pmatrix} \vec{\boldsymbol{\epsilon}}^{*H} \\ \vec{\boldsymbol{\epsilon}}^{*M} \end{pmatrix} \tag{9}$$

Since equation 9 represents the linearized dynamics near the disease–free equilibrium

for a vector – borne disease, it naturally exhibits a bipartite structure – reflecting the fact that infections occur only between humans and vectors, not within each group. This structure is intrinsic to the nature of vector–borne transmission. Therefore to fully capture the chains of infection over time it is essential to iterate the linearized system of equations. By computing powers of the system matrix – such as squaring or cubing – it becomes possible to study indirect transmission pathways, for example:

- Human → Mosquito → Human → Mosquito → Human

The second iteration of 9 provide insight into how indirect interactions contribute to the propagation of the disease throughout the network as follows.

$$\begin{pmatrix} \vec{\boldsymbol{\epsilon}}^{H} \\ \vec{\boldsymbol{\epsilon}}^{M} \end{pmatrix} = \frac{\beta^2 \lambda^{MH} \lambda^{HM}}{\mu^M \mu^H} \begin{pmatrix} \boldsymbol{M}\tilde{\boldsymbol{M}} & 0 \\ 0 & \tilde{\boldsymbol{M}}\boldsymbol{M} \end{pmatrix} \begin{pmatrix} \vec{\boldsymbol{\epsilon}}^{H} \\ \vec{\boldsymbol{\epsilon}}^{M} \end{pmatrix} \tag{10}$$

Hence, if the largest eigenvalue of $\boldsymbol{M}\tilde{\boldsymbol{M}}$ is Λ_{max} then,

$$R_0 = \frac{\beta^2 \lambda^{MH} \lambda^{HM}}{\mu^M \mu^H} \Lambda_{max}$$

Note that the eigenvalues of $\boldsymbol{M}\tilde{\boldsymbol{M}}$ and $\tilde{\boldsymbol{M}}\boldsymbol{M}$ are the same. Therefore, using $\boldsymbol{M}\tilde{\boldsymbol{M}}$ is not a matter of arbitrary choice. It is known that λ^{MH} and λ^{HM} are real numbers, and there is a proportional relationship between them always, such that $\lambda^{MH} = \delta \lambda^{HM}$ where $\delta \in \mathbb{R}$. This allowed the derivation of the critical epidemic threshold as follows.

$$\lambda_c^{MH} = \sqrt{\frac{\mu^H \mu^M}{\delta \beta^2 \, \Lambda_{max}(\boldsymbol{M}\tilde{\boldsymbol{M}})}}$$

However, even though the study focuses on investigating the critical contagion rate λ_c it is not a parameter that can be directly controlled, unlike other model parameters which are more easily adjustable. Nevertheless, identifying λ_c is important for understanding which patch contributes most to the epidemic, as this insight supports further analysis.

To validate this finding, they conducted several numerical simulations under the assumption that $\lambda_{HM} = \lambda_{MH} = \lambda$, and analyzed the fraction of infected humans, ρ^H at the equilibrium, as a function of λ and the human mobility parameter p. Their findings revealed that for each value of p, there exists a critical contagion rate λ_c, beyond which ($\lambda > \lambda_c$) an epidemic emerges. This indicates that the epidemic threshold λ_c is influenced by the level of human mobility. Interestingly, their results also showed that increased human mobility can, in some cases, hinder the spread of the epidemic – challenging the common assumption that mobility always aggravates disease transmission. This is because in diseases like vector–borne diseases, transmission depends on the ratio of vectors (e.g., mosquitoes) to humans in a patch: $\gamma_i = \frac{N_i^M}{N_i^H}$. If one patch has

a high ratio (lots of vectors per human), it is high risk for transmission. But when people move around, they increase the number of humans temporarily present in each patch (n_i^{eff}). As n_i^{eff} increases in high–risk patches (due to more people moving in), the effective ratio γ_i decreases. That is, the risk per person drops because there are more people sharing the same number of vectors.

Moreover, the curve $\lambda_c(p)$ sometimes shows sudden changes in slope (Figure 2).

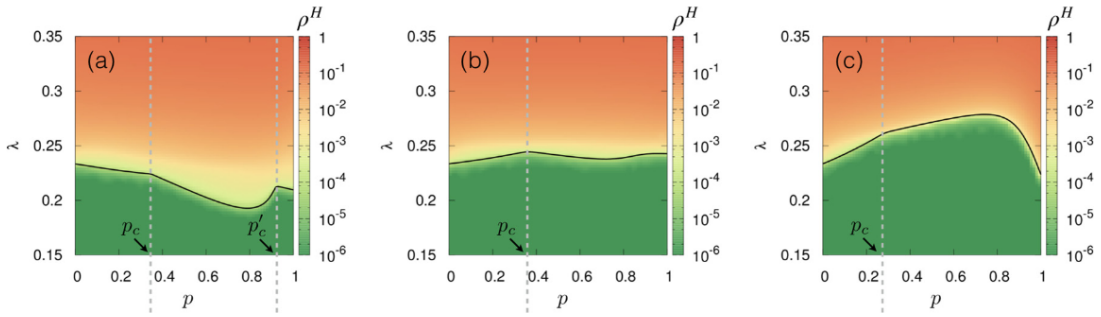

Figure 2: Epidemic threshold and evolution of the leading eigenvector of matrix $\boldsymbol{M}\tilde{\boldsymbol{M}}$ extracted from [1] (a) $\alpha = 0$, (b) $\alpha = 0.5$, (c) $\alpha = 1$

These sharp changes are not random; they happen because of something specific in the math behind the model. They come from collisions between the two largest eigenvalues of $\boldsymbol{M}\tilde{\boldsymbol{M}}$ that's used in the analysis. As the human mobility p changes, these two largest eigenvalues can switch places – meaning one becomes larger than the other at a particular critical value of mobility called p_c. These collisions between eigenvalues do not significantly affect the actual risk of an epidemic because that the function $\lambda_c(p)$ is continuous. While the maximum eigenvalue of $\boldsymbol{M}\tilde{\boldsymbol{M}}$ determines λ_c, the eigenvector $\vec{v}_{max}$ tells which patches are most responsible for driving the epidemic. When this vector changes suddenly, it means the *epidemic – driving* patches have shifted. This insight is crucial for designing effective containment strategies, as a measure that works well at one level of human mobility may become entirely ineffective at another.

References

[1] David Soriano-Paños et al. “"Vector–Borne Epidemics Driven by Human Mobility"”. In: *Physical Review Research* 2.1 (2020), p. 013312.