Assessing the interplay between human mobility and mosquito–borne diseases in urban environments: 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

E. Massaro *et al.* [1] conducted a study exploring the relationship between human mobility and dengue outbreaks within the complex urban setting of Singapore. However, this work contains several shortcomings and numerous instances of incorrect mathematical reasoning. Therefore, we aim to both explain the authors' approach and identify the errors they have made.

The study developed an agent–based dengue transmission model, where both humans and mosquitoes are treated as individual agents, and humans transition through the various epidemic states of dengue. To capture the dynamics of disease spread, the study employed a stochastic population model grounded in an ordinary differential equation (ODE) framework. We use the same notations used by the authors even though the notations are not consistent over the paper and complex.

$$
\begin{aligned}
S_t^{(\cdot)} &= \text{Susceptible humans (h) or mosquitoes (v) at time } t \\
E_t^{(\cdot)} &= \text{Exposed humans (h) or mosquitoes (v) at time } t \\
I_t^{(\cdot)} &= \text{Infectious humans (h) or mosquitoes (v) at time } t \\
R_t^h &= \text{Recovered humans at time } t \\
A_t^v &= \text{Mosquitoes in the aquatic stage at time } t
\end{aligned}
$$

The epidemiological model incorporated both temperature–dependent and constant parameters, as detailed in Table 1 and Table 2 (T stands for temperature). The spatial

environment for the simulations was a regular grid of $320m \times 320m$ cells, mapped over the city of Singapore. The values of γ^h and σ^h were obtained from former literature such as Wesolowski *et al.* [2].

Notation	Description
$\epsilon_A^v(T)$	Transition rate from aquatic to adult mosquito life–stages
$\mu_A^v(T)$	Mortality rate of aquatic mosquito life–stages
$\mu_V^v(T)$	Mortality rate of adult mosquito life–stage
$\theta_V^v(T)$	Intrinsic oviposition rate of adult mosquito life–stage
$\gamma_V^v(T)$	Extrinsic incubation period of adult mosquito life–stage
$\phi^{h\to v}(T)$	Human–to–vector probability of transmission per infectious bite
$\phi^{v\to h}(T)$	Vector–to–human probability of transmission per infectious bite

Table 1: Temperature dependent parameters

Notation	Description	Value
γ^h	transition rate from exposed E to infected I for humans	0.5 $days^{-1}$
σ^h	transition rate from infected I to recovered R for humans	0.25 $days^{-1}$
c	mosquito eggs hatching to larvae	1
f	female mosquitoes hatched from all eggs	1

Table 2: Constant parameters

The model operates in two distinct phases:

- a reaction phase, governed by the epidemiological model, during which disease transmission occurs within each grid cell (Figure 1)
- a diffusion phase, where agents relocate between grid cells based on the specified mobility model.

The study examined *four* distinct mobility models and compared their ability to predict the dengue outbreaks of 2013 and 2014 in Singapore. In each model, every agent was assigned a home and a work location (grid cell) and was assumed to commute between the two locations daily. The models differed in the method of assigning these locations:

- **Mobile phone data:** Used anonymized call detail records from a mobile phone operator in Singapore to estimate home and work cells for each agent.
- **Random work location:** Assigned home cells based on estimates from mobile phone data, while work locations were assigned randomly.
- **Lévy distribution** Assigned random home locations based on mobile phone data, with work locations chosen such that commuting distances followed a truncated Lévy distribution.

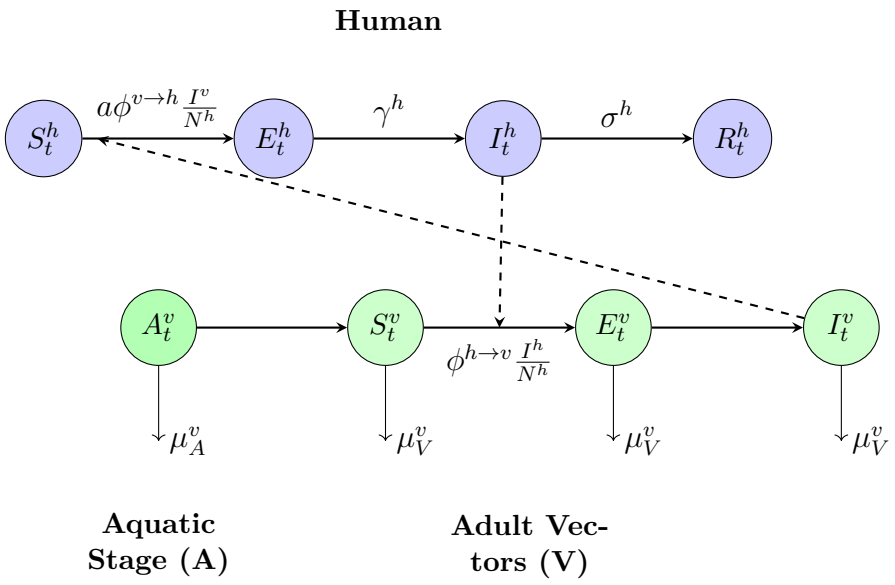

Figure 1: Flowchart of the transmission dynamics between humans and vectors, showing the progression through susceptible, exposed, infectious, and recovered stages.

- **Radiation model:** Distributed home locations using census data of Singapore and selected work cells according to established methods by Simini *et al.* [3]

Besides the mobility models, the study includes two key variables: the number of mosquitoes per human x_v and the average number of bites per day of mosquitoes a. Although x_v is not an independent parameter – it is determined by the ratio of mosquitoes and human population sizes – it was deliberately varied within a ranges $x_v \in [0.004, 0.0]$ alongside $a \in [0.14, 0.26]$ to reflect plausible scenarios in Singapore. Since accurately estimating these quantities in real–world settings is extremely challenging, the study adopted this sensitivity analysis approach to explore their impact on model predictions.

While most of the parameters listed in Table 1 were estimated using the best available values from the literature, x_v and a were varied to account for this inherent uncertainty. The optimal combination of these two parameters was selected for each mobility model to evaluate its performance. Observed dengue data from January 2013 was used as the initial condition for the agents. Although most of the parameter values were taken from the literature, it is not always clear whether these values were critically assessed or chosen primarily to fit the model to the desired outcome. This raises concerns about potential confirmation bias in parameter selection. A more rigorous sensitivity analysis or justification for the choice of each parameter would have strengthened the reliability of the model calibration.

The paper provides a detailed explanation of the data used, the development of the four different mobility models, and how the study compared their predictive power using various statistical methods and simulations. However, to remain consistent with

the scope of this thesis, we focus primarily on the mathematical formulation of the epidemiological model and its results.

2 Epidemiological model

2.1 Humans

Mosquitoes were assumed to remain fixed, meaning there was no interaction between mosquito populations across different grid cells. As a result, humans are modeled as individual agents, while mosquito populations were treated in an aggregated manner at the cell level. Each human agent i at time t is described by the pair $(N, c)_{t,i}$, where N represents the epidemiological state – susceptible (S), exposed (E), infected (I), or recovered (R) – and c indicates the grid cell the agent currently occupies. The location c alternates between a predefined home and work cell, which is either inferred from mobile phone data (in realistic scenarios) or randomly assigned. The variables $S^h_{t,c}, E^h_{t,c}, I^h_{t,c}, R^h_{t,c}$ denote the total number of susceptible, exposed, infected, and recovered individuals in cell c at time t respectively. Thus, the total number of human agents in cell c at time t can be expressed as:

$$(N, c)_{t,i} = S^h_{t,c} + E^h_{t,c} + I^h_{t,c} + R^h_{t,c}$$

There is an inconsistency in notation:
N is used both to represent the epidemiological state of human agents and to denote the total number of human agents, which may lead to confusion.

Following exposure to an infectious mosquito bite, individuals transition to the exposed state (E), where they remain for an average of γ_h^{-1} days. They then become infectious (I) for an average duration of σ_h^{-1} days before finally entering the recovered state (R), acquiring lifelong immunity. All agents are initially susceptible, and their state transitions follow the dynamics illustrated in Figure 1.

Each time step in the model corresponds to half a day. Therefore, the study assumes that state transitions occur at half the daily probabilities: $\frac{\lambda^{v\to h}_{t,c}}{2}$, $\frac{\gamma^h}{2}$, and $\frac{\sigma^h}{2}$ represent the probabilities of transitioning from $S \to E$, $E \to I$, and $I \to R$, respectively, within a single half–day step (see below for more details). However, this assumption introduces an error into the model. Probabilities do not scale linearly over time, and if treated as linear, they can eventually exceed 1, which is not meaningful in a probabilistic framework. Adjusting by a factor such as $\frac{1}{2}$ to account for two updates per day is only justifiable in the limit as the time step approaches zero, assuming constant rates. In practice, this simplification is not mathematically rigorous.
The *transmission rate*, which is associated with the mosquito population in the grid

cell where the human agent is located at that moment $\lambda_{t,c}^{v\to h}$ is

$$\lambda_{t,c}^{v\to h} = a\phi^{v\to h}(T)\frac{I_{t,c}^{v}}{N_{t,c}}$$

where

$$\begin{aligned} a &= \text{average number of mosquito bites per day} \\ \phi^{v\to h}(T) &= \text{disease transmission probability per bite} \\ I_{t,c}^{v} &= \text{total number of infected mosquitoes in cell } c \text{ at time } t \\ N_{t,c} &= \text{total number of human agents in cell c at time t} \end{aligned}$$

Another point of confusion arises here. It is evident that $\lambda_{t,c}^{v\to h}$ is not unit less but has units of $days^{-1}$ indicating that it is a rate, not a probability. Consequently, the value of $\lambda_{t,c}^{v\to h}$ can exceed 1 for suitable combinations of parameter values. However, in the previous paragraph, this rate was directly used to define a transition probability, which is mathematically incorrect. This suggests that the study may have treated the terms *rate* and *probability* as interchangeable, which is a serious conceptual error.

Accordingly, adopting similar notation and the SEIR framework, the study described the progression of human agents through the different compartments by introducing random variables for the number of transitions between each compartment.

Susceptible $\to$ Exposed

$$t_{t,c}^{S^h\to E^h} \sim Bin\left(S_{t,c}^{h}, \frac{\lambda_{t,c}^{v\to h}}{2}\right)$$

- $S_{t,c}^{h}$: number of susceptibles in cell c at time t
- $\lambda_{t,c}^{v\to h}$: force of infection from vectors (mosquitoes)
- $\frac{1}{2}$: due to two updates per day (e.g., morning and evening)

Once again, there is a misuse of fundamental concepts. The binomial distribution $Bin(n, p)$ requires n to represent the number of independent trials and p to be a valid probability (i.e., a value between 0 and 1). However, as previously discussed, $\lambda_{t,c}^{v\to h}$ is not a probability but a rate with units of $days^{-1}$. Moreover, the justification for using a factor of $\frac{1}{2}$ to account for two time steps per day is mathematically incorrect unless additional assumptions are explicitly stated and justified. Therefore, the transition from S to E denoted as $t_{t,c}^{S\to E}$, is incorrectly defined in the model. To correct this, the study may use the conversion of rate to probability,

$$1 - exp\left(-\lambda_{t,c}^{v\to h}\Delta t\right)$$

where $\Delta t = \frac{1}{2}$. New infections occurs at the rate $\lambda_{t,c}^{v \to h}$, randomly and independently – the chance of infection in the next moment does not depend on the past. Therefore, the number of infections occurring during a time interval Δt follows a Poisson distribution with mean $\lambda_{t,c}^{v \to h} \Delta t$. As a result, the probability that at least one infection occurs within this interval is given by $1 - exp\left(-\lambda_{t,c}^{v \to h} \Delta t\right)$.

Exposed $\to$ Infected

$$t_{t,c}^{E^h \to I^h} \sim Bin\left(E_{t,c}^h, \frac{\gamma^h}{2}\right)$$

- γ^h: human incubation rate of

The second chapter of this section states that humans remain in the exposed state E for γ_h^{-1} days, which confirms that γ^h is a rate. However, a few lines later, $\frac{\gamma^h}{2}$ is incorrectly referred to as a probability. Furthermore, this value – mistakenly treated as a probability – is then used to calculate a binomial distribution. These are serious issues in the context of scientific literature, as they reflect a fundamental misunderstanding between rates and probabilities, which are not interchangeable.

Infected $\to$ Recovered

$$t_{t,c}^{I^h \to R^h} \sim Bin\left(I_{t,c}^h, \frac{\sigma^h}{2}\right)$$

- σ^h: human recovery rate

Analogously, this is also incorrect. Similar misuse occurs with the parameters $\lambda_{t,c}^{v \to h}$ and σ^h, where rate values are treated as probabilities. Applying rate parameters directly in probabilistic distributions without proper transformation misrepresents the underlying stochastic processes. Such inconsistencies undermine the model's theoretical soundness and may lead to inaccurate simulation outcomes.
Then, the dynamics of human population expressed as follows.

$$\begin{aligned}
S_{t+1,c}^h &= S_{t,c}^h - t_{t,c}^{S^h \to E^h} \\
E_{t+1,c}^h &= E_{t,c}^h + t_{t,c}^{S^h \to E^h} - t_{t,c}^{E^h \to I^h} \\
I_{t+1,c}^h &= I_{t,c}^h + t_{t,c}^{E^h \to I^h} - t_{t,c}^{I^h \to R^h} \\
R_{t+1,c}^h &= R_{t,c}^h + t_{t,c}^{I^h \to R^h}
\end{aligned}$$

Although the model describes the transitions between epidemiological compartments in discrete–time as above, it does not account for births and deaths of human population. While individuals assumed to acquire lifelong immunity upon recovery the absence in

death process – including natural and disease induced mortality – implicitly suggests closed and fixed human population. However, this assumption is not stated in the paper. For completeness and clarity, it would have been beneficial to mention that the study neglects the demographic changes.

Furthermore, the assumption that the number of transitions from one compartment to another follows a binomial distribution is also flawed. For example, under this assumption, the number of susceptible agents in cell c at time t ($S^h_{t,c}$) is treated as independent across individuals. However, this is not the case in reality. Since the number of mosquito bites is finite, when one agent is bitten by a mosquito, that specific bite cannot be shared with another agent. Therefore, the assumption of independence – and hence the use of the binomial distribution – is not valid in this context. However, the study did not calculate the number of transitions by directly sampling from a binomial distribution. Instead, it performed independent Bernoulli trials for each agent using the appropriate transition probabilities and recorded the number of successes. While this approach yields a number of transitions equivalent to sampling from a binomial distribution, tracking each agent individually allowed the study to monitor the progression of every agent and maintain accurate information about their individual epidemic status – a detail that was crucial for the model's accuracy.

2.1.1 Mosquitoes

As mentioned in the earlier sections, the mosquito population in each grid cell has been modeled stochastically, taking into account their two–stage life cycle: Aquatic (A) and Adult Female (V) (refer to Figure 1). The total number of mosquitoes in each class is denoted by $A_{t,c}$ and $V_{t,c}$ respectively, for timestep t and cell c. The changes in the number of mosquitoes in each class for each cell are then calculated based on the following rules.

Aquatic deaths:

$$d^A \sim Bin\left(A^v_{t,c}, \frac{\mu^v_A(T)}{2}\right)$$

Sample the number of aquatic mosquitoes dying using a Binomial Distribution. The death probability is half the mortality rate $\mu^v_A(T)$. This is another example of treating a rate as a probability. Additionally, in such situations, probabilities should not be treated as linear. Therefore, the use of $\frac{1}{2}$ is incorrect.

Aquatic → Adult transition:

$$t^{A\to V} \sim Bin\left(A^v_{t,c} - d^A, \frac{\epsilon^v_A(T)}{2}\right)$$

From the surviving aquatic population, sample how many transition to adult mosquitoes based on the emergence rate $\epsilon_A^v(T)$. Similarly, the use of probability within the Binomial Distribution is incorrect here as well.

Adult deaths:

$$d^V \sim Bin\left(V_{t,c}, \frac{\mu_V^v(T)}{2}\right)$$

Sample how many adult mosquitoes die using the adult mortality rate $\mu_V^v(T)$. The same conceptual flaw appears here, where a rate is again treated as a success probability.

Adult → Egg–laying (new aquatics):

$$t^{V\to A} \sim Poisson\left[cf \cdot \frac{\theta_A^v(T)}{2}\left(1 - \frac{A}{K_{t,c}}\right)V\right]$$

Adult females lay eggs (new aquatics) following a Poisson distribution with the mean based on:

- $\theta_A^v(T)$ is the intrinsic oviposition rate of adult mosquito. Again, model erroneously treats rates and probabilities as interchangeable, which is mathematically incorrect.
- The logistic term for the ecological capacity to receive eggs is given by $\left(1 - \frac{A}{K_{t,c}}\right)$ where the carrying capacity in each cell $K_{t,c}$ defined as $x_v\frac{W_c+H_c}{2}$. Here, W_c and H_c represent the number of people whose work or home location is in cell c. According to this, the study assumed that the number of mosquitoes in a cell depends on the average number of humans who live or work in that particular cell c. This is a significant assumption that overlooks commonly understood constraints. Biologically mosquito breeding sites depends on water bodies, humidity, rainfall, vegetation etc. not simply on human presence. Even if human density is a vital factor for mosquito breeding in urban environment settings, simply averaging the work and home locations does not make a strong mathematical sense. In contrast, if any cell c has low W_c and H_c but ideal physical conditions for breeding, this assumption artificially lowers the carrying capacity. In addition, the model assumes a constant mosquito biting rate, regardless of local human density. This implies that mosquitoes will successfully feed at their full capacity even when only a few humans are present in the cell. If low human density is meant to limit the mosquito population via carrying capacity, then allowing mosquitoes to bite freely regardless of host availability creates a contradiction, as it unrealistically permits high mosquito survival and reproduction even in areas with few humans.
- f: fraction of female mosquitoes hatched from all eggs (assumed to be 1). This

is not a practical assumption. Although the study cited Yang et al.(2009) [4] as the source for this parameter value, that source actually used a value of $f < 1$, indicating that the fraction should be less than 1 in realistic scenarios.

- c: fraction of eggs hatching into larvae (assumed to be 1). Again, this is not a practical assumption. The same study, Yang et al.(2009) [4], was cited as the source for this parameter value, but it actually used a value of $c < 1$, indicating that the hatching success rate is realistically lower than 100%.

Furthermore, the study assumed that each adult mosquito independently lays a certain expected number of eggs per timestep, with this number influenced by both temperature and the carrying capacity described above. Since the mosquito population was modeled stochastically, the Poisson distribution was used to sample the total number of new aquatic mosquitoes based on the expected egg–laying rate. The expression inside the Poisson mean is dimensionally consistent and represents an expected count of eggs laid, which is exactly what the Poisson distribution parameter should be.
Then the mosquito population updated for the $(t+1)$ time step as follows.

$$\begin{aligned} A_{t+1,c} &= A_{t,c} - d^A - t^{A\to V} + t^{V\to A} \\ V_{t+1,c} &= V_{t,c} - d^V + t^{A\to V} \end{aligned} \tag{1}$$

Next, the study focused on formulating mosquito dynamics by extending the standard SEI model to include an *aquatic* stage and established the corresponding equations for mosquito dynamics (see Figure 1).

$$\begin{aligned} t_{t,c}^{S^v\to E^v} &\sim Bin\left(S_{t,c}^v, \frac{\lambda_{t,c}^{h\to v}}{2}\right), \\ t_{t,c}^{E^v\to I^v} &\sim Bin\left(E_{t,c}^v, \frac{\gamma^v(T)}{2}\right), \end{aligned}$$

The common mistake of mixing rates and probabilities, along with the incorrect assumption of additive probabilities, also appears here. These rates must first be properly converted into probabilities before being used in Binomial Distributions. The dynamics of the mosquito population are presented as follows.

$$\begin{aligned} S_{t+1,c}^v &= S_{t,c}^v - t_{t,c}^{S^v\to E^v}, \\ E_{t+1,c}^v &= E_{t,c}^v + t_{t,c}^{S^v\to E^v} - t_{t,c}^{E^v\to I^v}, \\ I_{t+1,c}^v &= I_{t,c}^v + t_{t,c}^{E^v\to I^v}. \end{aligned}$$

Notably, when summing these three equations, we obtain $V_{t+1,c} = V_{t,c}$, which directly contradicts the second equation in Equation 1, where mosquito deaths and transitions from the aquatic stage to the adult stage are explicitly accounted for. Without in-

corporating these demographic processes, the total adult mosquito population at time $t+1$ cannot be correctly determined.

The rate of disease transmission from human to mosquito $\lambda_{t,c}^{h\to v}$ is,

$$\lambda_{t,c}^{h\to v} = a\,\phi^{v\to h}(T)\,\frac{I_{t,c}^{h}}{N_{t,c}},$$

Therefore, by using simple bookkeeping, we can derive R_0. On average a single infected human produces

$$\frac{Va\phi^{h\to v}(T)}{N\sigma^{h}}$$

number of infected mosquitoes during their infectious period. But not all of them survive, and suppose the rate at which exposed mosquitoes become infectious ($E_t^v \to I_t^v$) is γ^v. Hence, one infectious mosquito can infect

$$\frac{\gamma^v}{\gamma^v+\mu_V^v}\cdot\frac{a\phi^{v\to h}(T)}{\mu_V^v(T)}$$

number of humans. By multiplying these two terms to account the human–to–human transmission we can get the R_0 as follows.

$$\left(\frac{Va\phi^{h\to v}(T)}{N\sigma^{h}}\right)\times\left(\frac{\gamma^v}{\gamma^v+\mu_V^v}\cdot\frac{a\phi^{v\to h}(T)}{\mu^v(T)}\right)=\frac{V}{N}\frac{a^2\phi^{h\to v}(T)\phi^{v\to h}(T)}{\sigma^h\mu^v(T)}\cdot\frac{\gamma^v}{\gamma^v+\mu_V^v}$$

However, the study ignored the possibility of vector death during the exposed stage and consequently derived an incorrect expression for R_0.

2.2 Results of the Paper

According to the study,running the model involves repeating two main steps:

- For each human, determine state changes using individual Bernoulli trials, and update mosquito populations in each cell according to mosquito dynamics
- Move human agents according to the mobility model and update the counts of humans in each category within each cell.

The model successfully reproduced the main temporal and spatial patterns of Singapore's dengue outbreaks in 2013 and 2014. Human mobility was found to be a critical driver of dengue spread, even within city–scale distances. This was shown by comparing real outbreak patterns to those generated under a random mobility model (perfect mixing), which produced very different spatial distributions. The study extended previous work (e.g., Wesolowski *et al.* [2]) by demonstrating that mobility matters not just between cities or regions but also within cities, challenging the common assumption that cities can be treated as well–mixed environments for disease modeling.

However, it is unclear how temperature was incorporated into the model. While it is true that the dynamics of humans and mosquitoes do not solely depend on temperature, it is nevertheless an important external driver influencing survival and transmission dynamics. Therefore, it is understandable that the study may not include temperature as an explicit dynamic variable. However, it remains questionable what functional forms were used for the temperature–dependent parameters listed in Table 1. In the summary of the paper, the authors mention that these temperature–dependent forms were extracted from previous literature such as [2, 4], but no explicit details were provided. It would have been more informative if the study had clearly stated how temperature dependence was modeled and what specific functional forms were used.

References

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