Spatial Patterns of Spread of Dengue with Human and Vector Mobility

Murali Krishna Enduri *1 and Shivakumar Jolad†1

1Indian Institute of Technology Gandhinagar Chandkheda, Ahmedabad 380005, INDIA

September 4, 2014

Abstract

Background: Dengue is a vector borne disease transmitted to humans by Aedes Aegypti mosquitoes carrying Dengue virus of different serotypes. Primarily an urban epidemic, Dengue exhibits complex spatial and temporal dynamics, influenced by many biological, human and environmental factors. However, most of the existing models neglect the spatial factors influencing the spread of Dengue. This work sheds light on how Dengue parameters and human mobility changes the spatial spread of the infection and size of the epidemic.

Methodology/Principal findings: We model the Dengue as a stochastic Cellular Automata (CA) process following Susceptible, Exposed, Infected, Recovered (SEIR) - Susceptible, Exposed, Infected (SEI) for human and vector dynamics respectively in each cell, and analyze the spatial and temporal spreading disease using parameters from field studies. We use the data on mosquito density from Ahmedabad city of India as input to our model to predict the dynamics of Dengue incidence and compare it to the reported data on the prevalence of the disease from 2006-2012. We find that for certain infection rates, CA model closely reproduces observed peaks and intensity. We used data based statistical models of human mobility such as exponential step length and super diffusive Lévy flight to study mobility effects on Dengue spreading within the city. We find an interesting result that inclusion of human mobility in many cases can decrease the incidence of Dengue, and may suppress the infection completely. The scale and intensity of reduction depend on the relative strengths of infection transmission rate and mobility step length. The primarily reason for decline can be attributed to the significant fraction of the susceptible and exposed population moving to the regions where majority have already recovered and can no longer be infected.

Conclusions/Significance: The current study is aimed at more realistic model of spatial spread of Dengue and understand how human mobility factors in the spread of vector borne diseases. It highlights certain counter intuitive results such as suppression of Dengue spreading when we include statistical patterns of human mobility. In supplementary information, we show that even for Lévy flight broadly same conclusions hold. The random mobility pattern produces large peak initially, but suppresses Dengue completely within a short period of time even for large infection rate, deviating largely from the observed pattern.

Keywords: Dengue, Epidemics, Spatial Epidemiology, Cellular Automata, Vector Borne Diseases, Lévy Flight, Human Mobility, Reaction-diffusion

1 Introduction

Dengue fever (DF) is a vector borne disease widely prevalent in tropical and subtropical regions in about 100 countries worldwide. The World Health Organization (WHO) estimates that currently over 2.5 billion people (40% of the world population) are at the risk for Dengue, with close to a million cases reported 2007 alone [23]. Dengue is transmitted to humans mainly through Aedes Aegypti female
mosquito bites carrying Dengue virus [16]. Dengue fever (DF), Dengue hemorrhagic fever (DHF) and Dengue shock syndrome (DSS) are different forms of Dengue infection, caused by four serotypes of Dengue virus (DENV:1-4) [23]. The people who recover from one serotype can become permanently immune to it, but may not be immune to other serotypes. Dengue is becoming a major public health concern in various South Asian and Latin American countries. In India, Dengue epidemic has spread to almost all the states and is posing a serious public health problem. In 2010 alone, 28000 confirmed cases were reported (see Fig. 1). At present there is no effective vaccination or treatment for dengue. It is believed that any future dengue vaccination is imperfect, [2] and may not offer protection against all serotypes. Many Dengue infections may not produce severe symptoms, thereby evading early detection. At present, known effective way to prevent dengue outbreak is to devise vector control strategies and minimize vector-human transmission. A sound understanding of the spatial and temporal dynamics of the Dengue can help in devising strategies for containing the spread of urban populations.

Numerous human, biological, social and environmental factors affect the transmission of Dengue. Several mathematical models have been proposed [see 16, 8, 1 for reviews] for studying the Dengue. Many of these are the compartmental ordinary differential equation (ODE) models, which divide the human population into Susceptible, Exposed, Infected, and Recovered (SEIR) groups; and vectors into Susceptible, Exposed, Infected (SEI) groups, and studying their temporal dynamics. These ODE models are essentially mean field models, which neglect the spatial patterns of the spread of Dengue. Attempts at spatial modeling of Dengue includes those based on spatial data mapping and statistical analysis [3, 4, 18], reaction-diffusion partial differential equation (PDE) with vector or larval mobility [22, 14], and Cellular Automata with vector and simplified models of human mobility [7, 20]. In this work, we first report results of SEIR-SIR reaction-diffusion PDE model with data driven inputs as a reference to our full scale study of spatio-temporal dynamics through Cellular Automata.

Human mobility, especially of the infected individuals can create multiple Dengue waves resulting in substantial deviation from mean field results. However, the current approaches to modeling human mobility have been restricted to simple methods such as movement with fixed step size or introducing a global field altering transition probabilities [7, 20]. In this work we use stochastic Cellular Automata (CA) approach (closely following [7]), with realistic models of human mobility patterns derived from statistical studies of human movements observed through the circulation of currency notes, tracking of phone calls through Cellular towers, and location based social networks such as Foursquare. These works have shown varied patterns such as Lévy flight (Brockman et al. [6, 5]), truncated Lévy flight (Gonzalez et al. [9]), exponential distribution in intra-urban movements (Liang et al. [13].
and Noulas et al. [17]). The differences arise possibly due to the difference in scale and resolution of study (large distance, intra city movements, mobile tower coverage etc.) and the methodology used. Since Dengue is primarily an urban disease, we focus on the spread within an urban area and use exponential tail [15] distribution for studying human mobility affects on spread of Dengue. A comparative study with Lévy flight, exponential and uniform distribution of step length of human mobility on vector borne disease is described in the supplementary information.

We model the spatial dynamics of Dengue using the CA formalism by placing human and vector “agents” on two overlapping lattices and different disease states of these agents. We impose transition probabilities between the disease states based on field studies. For vectors we impose diffusive pattern, and for humans we use the exponential distribution of step length as described before. As inputs to the model, we use the data from a Ahmedabad city in the mid-western part of India with a population of about 6 million people (In Fig.2, we show the Dengue cases at ward level in Ahmedabad in 2010). We compare the results of simulation Dengue incidence with the recorded cases in Ahmedabad from 2006-2012.

Our paper is organized as below. Section 1 is the current introduction. In Section 2, we discuss compartmental structure (SEIR-SEI) of our Reaction-Diffusion (RD) and Cellular Automata models. For the RD model, we also discuss main results in this section. CA model has sub sections describing vector and human lattice and their interaction. For vectors, we discuss how we model their population based on the available data, and assumptions on their mortality and mobility. For Humans, we describe distribution of people on the lattice, and mobility patterns derived from statistical distribution. Human-vector interactions we describe how infection gets transmitted from vector to human lattice and vice versa. In Section 3, we describe the results from CA simulations. We divide this section into two sub-sections describing the Dengue spread with and without human mobility. In Section 3.1, we describe the spatio patterns of the disease spread without human mobility for different infection rates over a span of 76 months from the start of the disease. We compare the temporal results so obtained with the actual Dengue cases in Ahmedabad from 2006-2012. In Section 3.2, we move on to describe spatial patterns including human mobility. We will describe different models of human mobility, and explain the rationale behind using exponential distribution for our model. We also compare the spatio-temporal patterns with and without mobility and describe the counter intuitive and contrasting results caused by human mobility (In supplementary information we provide a comparison of the other two models of human mobility viz: Lévy flight and Random step length distribution with that of immobile humans). In the last section we conclude by summarizing the main features and results of our model.

2 Model Formulation

In this study, we use the standard compartmental model to divide the humans into SEIR and vector population into SEI groups. Only infected vectors and infected humans can transmit the dengue virus to susceptible population (See Fig.3 for illustration). Exposed population is infected with the vector but not infectious (i.e. they cannot transmit
Figure 3: SEIR-SEI model of human-vector interactions

the Dengue virus). In Fig 3, we show the flow diagram of SEIR-SEI model. The corresponding ordinary differential equations (non-spatial) describing temporal evolution of Human and vector population are given below in Eqs. 1 and 2 (see [16, 1] for review):

Disease dynamics in humans:

\[
\begin{align*}
\frac{dS_h}{dt} &= \mu_h N_h - \beta_{vh} S_h I_v - \mu_h S_h \\
\frac{dE_h}{dt} &= \beta_{vh} S_h I_v - \alpha_h E_h - \mu_h E_h \\
\frac{dI_h}{dt} &= \alpha_h E_h - rI_h - \mu_h I_h \\
\frac{dR_h}{dt} &= rI_h - \mu_h R_h,
\end{align*}
\]

(1)

Disease dynamics in vectors:

\[
\begin{align*}
\frac{dS_v}{dt} &= \mu_v N_v - \beta_{hv} S_h I_v - \mu_v S_v \\
\frac{dE_v}{dt} &= \beta_{hv} S_h I_v - \alpha_v E_v - \mu_v E_v \\
\frac{dI_v}{dt} &= \alpha_v E_v - rI_h - \mu_v I_v.
\end{align*}
\]

(2)

Here \( \mu_h, \mu_v \) represent human and vector mortality (death rates); \( \beta_{vh}, \beta_{hv} \) represent the vector to human and human to vector infection rates; \( \alpha_h, \alpha_v \) are the transition rates from exposed to infected states in humans and vectors; \( r \) is the recovery rate. The above equations assume that the total population of humans and vectors is conserved. The death rate for humans (about \( 1/(75\text{yrs}) \)) is much lower than that of vectors (about \( 1/(50\text{days}) \)), hence the dynamical time scales vary widely. The temporal evolution of these equations, stationary states and stability conditions have been investigated and reported in many works [16, 19], and will not be discussed in this work. We focus on the spatial approaches to modeling Dengue based on reaction diffusion equations in the next section.

2.1 Reaction-diffusion approach

In the ODE models described above assumes mixing of population and hence no spatial information is encoded in it. Spatial approaches are needed to analyze local and global dynamics, and present more realistic picture of the disease propagation. In vector borne diseases, spatial spreading is possible only when there mobility of vectors, humans or both. Vectors, especially A. Aegypti rarely fly long distances by itself, and their mobility can be modeled a diffusion process [22, 14]. The ODEs will be modified to Partial Differential Equations (PDE), where the diffusion terms will be added only to the vector equations. Human mobility is more complex and harder integrate into the PDE paradigm, requiring use of non-local fractional derivatives [5]. We will consider human mobility patterns in the Cellular Automata approach.

The modified PDE equations for humans in stages \( \{S, E, I, R\} \) with spatial \((x, y)\) and temporal dependence are:

\[
\begin{align*}
\frac{\partial S_h}{\partial t} &= \mu_h N_h - F^2 \beta_{vh} S_h I_v - \mu_h S_h \\
\frac{\partial E_h}{\partial t} &= F^2 \beta_{vh} S_h I_v - \alpha_h E_h - \mu_h E_h \\
\frac{\partial I_h}{\partial t} &= \alpha_h E_h - rI_h - \mu_h I_h \\
\frac{\partial R_h}{\partial t} &= rI_h - \mu_h R_h.
\end{align*}
\]

(3)

For the vectors, the modified equations with the diffusion term and flight range \( F \) are:

\[
\begin{align*}
\frac{\partial S_v}{\partial t} &= \mu_v N_v - F^2 \beta_{hv} S_h I_v - \mu_v S_v \\
\frac{\partial E_v}{\partial t} &= \mu_v N_v - F^2 \beta_{hv} S_h I_v - \mu_v S_v.
\end{align*}
\]
\[
\frac{\partial E_v}{\partial t} = D \nabla^2 E_v + F^2 \beta_h v_s I_h - \alpha_v E_v - \mu_v E_v, \\
\frac{\partial I_v}{\partial t} = D \nabla^2 I_v + \alpha_v E_v - \mu_v I_v, \tag{4}
\]

where \( \nabla^2 = \frac{\partial^2}{\partial x^2} + \frac{\partial^2}{\partial y^2} \) is the Laplacian operator. The parameter values are shown in Table 1. The flight range \( F = 7 \) indicates the maximum number of cells, vectors can hop in at a time.

The results of our simulation on a 500 \( \times \) 500 mesh grid, running for 580 days are shown at various intervals in Fig. 3. The top (bottom) row shows the density of humans (vectors) in various stages of the disease. Initially (at \( t=0 \)), all humans are susceptible and all vectors, except the ones at the center are susceptible. Only the central cell vectors are initially infected with Dengue. The coloring scheme follows Fig. 3 with the following rules: If the susceptible (recovered) density exceeds 0.5, the cell color is blue(green), or if the infected(exposed) density exceeds 0.25, it is painted red(orange).

The overall pattern of the Dengue spread appears like a circular wave spreading outwards. This is due to the assumption of homogeneous and isotropic distribution of population and diffusion constants being same in all directions. We observe that Infection in the vector grid spreads faster than the Human grid. We should note that the death rate of vectors is much higher than that of humans, but the incubation period is longer in vectors than humans \( (\alpha_h > \alpha_v) \). Hence we can clearly observe the exposed (in orange) population in vectors than humans.

The corresponding temporal variations of humans and vectors in different disease states (extended upto 1200 days) is shown in Fig. 5. It exhibits typical SIR behavior upto 27 months. The decline in infected population afterwards is mainly due to boundary effect. Infections cannot spread beyond that and only possibility is recovery of the infected.

### 2.2 Cellular Automata

In Cellular Automata, space is divided into discrete grid and a finite state machine is assumed to operate at each grid point. Time is discrete, and at every step each cell is in one of a finite set of possible states. The transition probabilities depend only on the present state of the cell and its neighbors [21].

In this work, we choose a two layer stochastic Cellular Automata model where each cell can have multiple agents and their number and states change with time [7]. It consists of two overlapping square lattices \( H \) and \( M \) of the same size (see Fig. 6) representing the space occupied by humans and mosquitoes respectively. Each cell \( H(i,j) \) of human layer can have multiple agents (Humans) in different disease states \( \{S,E,I,R\} \), which can change with time. For example consider a cell \( H_{23} \) with 5 humans with \( \{3S,2E,0I,0R\} \). In the next time step, it can be \( \{3S,1E,1I,0R\} \). Similarly, in Vector cell, mosquitoes can have states \( \{S,E,I\} \), which can change with time. In Table 2, we summarize the main parameters used in our CA simulations.

#### 2.2.1 Vector population and mobility

In each cell of the vector lattice \( V(i,j) \), mosquitoes are distributed according to the Poisson distribution \( P(\lambda_v) \), where \( \lambda_v \) is the average density of the \( A.Aegepti \) mosquitoes in the cell. We use the periodic (monthly) data, denoted by \( \{N_{m_1}, N_{m_2}, \ldots N_{m_23}\} \), on the \( Aedes \) vector density available for Ahmedabad city in India from 2006-2012, plotted in Fig. 7, for simulations. At the start of the simulation, in each cell the mean occupation is assumed to be \( \lambda_v = N_m \) the measured average per room density of mosquitoes. To interpolate between the monthly data, we assume an exponential growth \( n(t) = n(t_0)e^{r(t-t_0)} \) between month \( m_k \) and \( m_{k+1} \), with growth rate \( r = \frac{1}{30}\ln(N_{m_{k+1}}/N_{m_k}) \), \( n(t_0) = N_{m_k} \) and \( t_0 \leq t < t_0 + 30 \). We denote the population in each cell from simulations be \( n_i(t) \) and assign new births/deaths depending whether \( \delta n = n(t) - n_i(t) \) is positive or negative. If \( \delta n > 0 \), we generate new susceptible mosquitoes with Poisson distribution \( P(\delta n) \). If \( \delta n < 0 \), we calculate the a random number \( m = P(-\delta n) \) and kill them in that cell.
Table 1: Table of parameters for the ODE and Reaction and Diffusion Equation equations [19]

| Parameter | Description                                      | Value                  |
|-----------|--------------------------------------------------|------------------------|
| $\mu_h$  | Death rate of humans                             | 0.0000391 per day      |
| $\mu_v$  | Death rate of vectors                            | 0.07142 per day        |
| $\beta_{hv}$ | Infection rate from human to vector             | 0.00008                |
| $\beta_{vh}$ | Infection rate from vector to human            | 0.00005                |
| $r_h$    | Recovery rate of human population               | 0.07142 per day        |
| $\alpha_h$ | Rate at which exposed human change to infected human | 0.2 per day            |
| $\alpha_v$ | Rate at which exposed vector change to infected vector | 0.1 per day            |
| $F$      | Flight range                                     | 7                      |
| $D$      | Diffusion Coefficient                            | 0.2                    |

Figure 4: PDE model with vector mobility with grid size is 500 × 500. The upper row corresponds to human grid and the bottom row to vector grid.

Figure 5: Temporal dynamics of Reaction Diffusion series - (a) SIR for humans, (b) S-I for vectors (c) Infected Humans and Vectors.

**Vector Mortality:** A. Aegepti life span is short (average 45 days) and its death rate is not significantly affected by whether it is carrying the Dengue virus or not. We model the mosquito as dying with constant rate $\mu_v$. 
Vector Mobility: We model the vector mobility by hopping of mosquitoes between cells. At each time step, 50% of the mosquitoes decide to move out of the cell. The probability of hopping to a Moore neighborhood of range \( r \) is \( \frac{1}{2r} \). Within this range, the probability of choosing any cell is uniform \( p = \frac{1}{((2r + 1)^2 - 1)} \). If we take \( a \) to be the width of each cell, the average distance along sides is \( a \sum_{r=1}^{\infty} \frac{r}{2^r} = 2a \) (maximum flight range \( f_r = 25m \)), and along the diagonal is \( 2a\sqrt{2} \).

### 2.2.2 Humans Lattice and Mobility

In each cell, human occupation is chosen to be Poisson distribution \( P(\lambda) \) with the mean, \( \lambda = 5 \). The average population of our model with grid size 500 \( \times \) 500 is about 1.25 million. The birth rate in the cell is balanced with the death rate to keep the population constant. Human mortality rate \( (1/70\text{yrs}) \) is much smaller compared to the vector and does not account for significant deaths during the simulation time. We also assume that the death rate due to Dengue is negligible compared to the actual cases.

Human mobility is primarily responsible for carrying communicable diseases to large distances, both within the city and across the cities. In vector borne diseases, disease can be spread by both human and vector mobility. Typically human mobility cause the virus to carry across different regions in much shorter time period than the vectors. Mobility of infected humans or vectors create multiple waves of diseases at different locations. Mobility can also lead to depletion of infected in a particular region and hence local reduction in the transmission rates. Such competing forces call for a careful study of the mobility effects on the spread of vector borne diseases.

Many factors influence mobility within a city-population density, transportation networks and traffic patterns, economic activity in different parts etc (home, work, school, shops, hospitals, transportation networks etc.). What is clear is that mobility patterns are not diffusive. Modeling spatial human mobility in cities (especially in India) in the absence of credible data is a daunting task. Several ingenious methods have been used in the past to study the statistical patterns of human mobility. Tracking of currency notes yielded a scale free Lévy flight pattern\(^6\) \( P(\Delta r) \sim (\Delta r)^{-(1+\beta)} \) across large scale. Later, a study based on the trajectory of 100,000 anonymized mobile phone users \(^9\) in US showed that the step length distribution behaves like a truncated Lévy flight \( P(\delta r) = \frac{A}{(\delta r + \delta r_0)^\sigma} \exp(-\delta r/\kappa) \).
The advantage of these methods is that statistical patterns are robust and do not critically depend on the variations in transportation networks or population density. Dengue is primarily an urban disease, where mobility within the city is more important than across. Study of intra-urban mobility received special attention in the recent years. Recently Liang et al. have produced a strong evidence of exponential distribution in intra-urban movements [13]. This work is supported by a recent study by Noulas et al., when explored at intra city level [17] using location data by social networking Foursquare. In this work, we choose exponential step length model:

\[ P(\Delta r) = \lambda e^{-\lambda \Delta r}, \]  

(5)

where \( 1/\lambda \) is the average step length (which in general is not universal). Liang et al. found this exponent to vary from 0.08 \( km^{-1} \) (Los Angeles) to 0.22 \( km^{-1} \) (Beijing, and London). We choose \( \lambda = 0.2 km^{-1} \) as a representative parameter for our study. In supplementary materials, we provide a detailed comparison of results from different models of human mobility.

In this work, we restrict the mobility to S, E and R population. Infected are immobile as they are at rest/hospitalized. With a 50% probability of S, E or R type people move from their current cell following above distribution. Once a person decides to move, a random number following exponential distribution (as in Eq. 5) is drawn and step length is determined. The angle is from uniform distribution \( U(0, 2\pi) \). The cell which contains the location \((\delta r, \theta)\) from the current point is chosen as the destination cell. If the range is outside the CA boundary, periodic boundary conditions to bring the person back into the CA world. This kind of ensures that net migration (in and out of CA) is zero.

2.2.3 Interactions between humans and vectors

Human-vector and vector-human interaction happens mainly through mosquito biting. We have set the maximum biting rate of two and assumed that the probability of \( \{0, 1, 2\} \) bites are \( \{1/4, 1/2, 1/4\} \) respectively. In each cell, an A. Aegypti mosquito (of any type S, E or I) randomly selects a human to bite and after each bite, the vector stays in the same cell or follows the vector mobility pattern described before. The Dengue virus is transmitted from human to vector (or vice-versa) when an infected vector \( I_v \) bites a human in a susceptible state \( (S_h) \) or an infected human \( I_h \) gets bitten by a susceptible mosquito \( S_v \) with rate \( \beta_{hv}(\beta_{vh}) \) of 0.4/bite (see Table 2). Spatial spread across the cells happens due to vector or human mobility as described before.

3 Results

We have chosen CA grid size to be \( L \times L = 500 \times 500 \) and an average human occupancy of 5 agents per cell. This gives us a large grid population of 1.25 million, suitable to emulate a typical large city in India. If we assume each cell to represents an area \( a^2 = 10m \times 10m = 100m^2 \), (where \( a \) is the width of each cell), we get the model world size to be \( 25km^2 \). Our focus is to compare the spatio-temporal patterns of Dengue with and without human mobility and test how closely it can explain observed Dengue dynamics in Ahmedabad city (located in the middle-western part of the country) for which Dengue cases and mosquito density has been is available from 2006-2012.

3.1 Mobile vectors and immobile humans

In Fig 8 we show panels of spatio-temporal spread of Dengue on human layer. The parameters are chosen from Table 2 with diffusive vector mobility (Section 2.2.1), and four different values of \( \beta_{hv} \) and \( \beta_{vh} \) viz. : 0.15, 0.4, 0.6, 0.9. We choose the following color code: Black represents no human (vector) occupation in the cell. The cell is colored blue at least one person is susceptible. It is colored orange or red depending on whether more than 25% of the population is exposed or infected. In case of conflict, infected color red gets priority. If all persons have recovered, then the cell is colored green.
Table 2: Value of Parameters

| Parameter | Description                                      | Values                          |
|-----------|--------------------------------------------------|---------------------------------|
| \( \tau_{hE} \) | The duration for exposed human to become Infected | Uniform distribution: 4-7 days [11] |
| \( \tau_{vE} \) | Infected vectors incubation period               | Uniform distribution: 8 to 12 days [23, 10] |
| \( f_r \) | Maximum flight range of vectors                  | 25 m [12]                      |
| \( B_r \) | Biting rate of vectors                            | 0 to 2/day [7]                  |
| \( \beta_{hv}, \beta_{vh} \) | Human to vector and vector to human transmission rates | \{ 0.15, 0.4, 0.6, 0.9 \} |

Figure 8: Cellular Automata model with immobile humans. The parameters values are listed in Table 2. Grid size is taken to be 500 × 500.

We start the disease dynamics by setting all vectors in the central cell to be infected. Based on these figures, we make the following observations:

- At \( \beta_{hv} (0.15) \) infection levels are very low and spatial patterns cannot observed. In Fig[10] we see that infections persist at very low levels even after 72 months. But we cannot ascertain distance from the center to which disease goes extinct from the figure.
- For higher \( \beta_{hv} \geq 0.4 \), disease enters the endemic state and spreads outwards precise calculation of transition point in is beyond the scope of this work.
- The Dengue spreads on the human and mosquito lattice in a circular wave pat-
tern. Vector wave leads the infection (not shown).

- In the initial phase, the Infected humans are present throughout the inner circle. Later the infected are mainly in the periphery. In the core infected humans recover (green) over time, passing through incubation period.

- People in the core who were not infected previously are at the risk of infection due to secondary regeneration of vectors. These periodic outbreaks have high correlation with the temporal variation of vector density (not shown). They occur with different intensity and scale show high correlation with the rainfall pattern (cross correlations not shown in this work).

- In the absence of human mobility, Dengue takes long time to spread to the boundary. Speed of propagation increases with the infection rate.

The temporal patterns of human and vector population, infected human and infected vector and SEI groups of humans are shown in Fig.9. Here we observe that at any time, the fraction of infected is quite small comparable to susceptibles (Panel a). But, over time large fraction of the population will be infected and move to recovery phase (as expected from SIR or SEIR dynamics R is monotonically increasing in active phase if there no deaths). Similar trend can be seen in for vectors also in panel b. On a closer look, (in panel c), we see that both infected human and infected vectors show substantial variations in time and are correlated with annual periodicity of monsoon-rainfall and other harmonics (not shown).

In Fig.10, we compare the simulation results for immobile humans (red dash) with the actual Dengue cases in Ahmedabad (black dot-dash) for different infection rates for a period of 6 years. As before, we make all vectors in the central cell of the vector lattice infected. These vectors will infect agents in the human lattice, and the disease spreads to the neighboring cells due to both vector and human mobility, and also due to interaction between human and vector lattice. Color coding follows the description in section 3.1. Based on the Fig.11, we make the following observations:

In Fig.12 (a), we show SI dynamics of vectors for the special case of \( \beta_{hv} = 0.15 \). Susceptible vector patterns closely follow the mosquito density pattern obtained from Ahmedabad data in Fig.7. Infected vector population initially raises till 12 months and then decreases. Compared to the corresponding immobile case (see Fig.9), infection will almost die down after 24 months. A comparison of Infected vectors (IV) and humans (IH) is shown in panel (b). We clearly see a close correlation between the two, highlighting that disease propagates in both human and vector lattices in sync. The IV population is higher than that of IH. Possibly this is due to the different dynamics of vec-

3.2 With both Vector and Human mobility

The process of modeling human mobility has been described in Section 2.2.2. In this section we describe the results with both vector and human mobility.

3.2.1 Spatial and Temporal Variation

In Fig.11, we show spatial variations of SEIR groups in human lattice for different infection rates for a period of 6 years. As before, we make all vectors in the central cell of the vector lattice infected. These vectors will infect agents in the human lattice, and the disease spreads to the neighboring cells due to both vector and human mobility, and also due to interaction between human and vector lattice. Color coding follows the description in section 3.1. Based on the Fig.11, we make the following observations:

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Figure 9: Temporal dynamics of Dengue ($\beta_{hv} = 0.4$) (a) Susceptible, Infected and Recovered Humans (b) susceptible and Infected vectors (c) Infected humans and Infected Vectors.

Figure 10: Comparison of Dengue cases from our simulation with recorded cases from Ahmedabad city from 2006-2012 neglecting mobility of humans. (a) $\beta_{hv} = 0.15$ (b) $\beta_{hv} = 0.4$ (c) $\beta_{hv} = 0.6$ (d) $\beta_{hv} = 0.9$. (Note the difference in scale in all panels).

In our framework, we consider: SEI compared to SEIR. Infectious state is the last state for vectors, which in the absence of mortality should raise to the initial number of susceptibles. However competing dynamics
of birth rates following observed data and high mortality rate, we observe higher vector population than humans. It also keeps the vectors from completely going into absorbing state.

3.2.2 Comparison with and without human mobility

In Fig.13, we explicitly compare infected human population with and without human mobility for different infection rates. Based on the this figure and the spatial spread in Fig.11 we make the following observations.

- For $\beta_{hv} = 0.15$ infection initially rises to a maximum of 12 (see Panel a) and is completely suppressed in a short span of 11-12 months time, highlighting that the dynamics goes to an absorbing state. Where as for the immobile case, infections are still active, even though the magnitude is small. Spatial spread (Fig.11 a) cannot be observed as the infection level does not exceed 25% threshold in most cells.

- For $\beta_{hv} = 0.4$ (Panel b), infected population rises initially and reaches a peak in less than year and is completely suppressed after 20 months. In the later months we see some statistical fluctuations. In the immobile humans case, the disease is in an active state throughout. These observations highlight that human mobility has suppressed infection contrary to intuitive understanding.

- At $\beta_{hv} = 0.6$ (Panel c), we see Infection levels two orders of magnitude higher that of $\beta_{hv} = 0.4$ (signaling active phase). The scale of infection with and without human mobility are roughly same, and also the relative position of peaks in infections. However, mobility is causing higher infections in the beginning compared to the
Figure 12: SIR dynamics of humans and vectors with exponential human mobility model with $\beta_{hv} = 0.4$. (a) Susceptible and Infected vectors (b) Infected humans and Infected Vectors.

Figure 13: Comparison of Dengue cases with exponential human mobility and with out human mobility (a)$\beta_{hv} = 0.15$ (b)$\beta_{hv} = 0.4$ (c)$\beta_{hv} = 0.6$ (d)$\beta_{hv} = 0.9$. (Note the difference in scale).

- There is still suppression of infections due to mobility. The spatial maps for ($\beta_{hv} = 0.6$ in Fig.11) shows the infection does not spread like a circular wave in contrast to immobile. Mobility has diffused the infections. By 60 months most people have recovered and spreading has slowed down drastically.
- The pattern for $\beta_{hv} = 0.9$ (Panel d) is much more similar to the case without human mobility, with position of infection peaks matching up to 60 months and keeping order of magnitude same. Spatial
patterns show circular wavy spread in the upto 32 months and later hit the boundary lattice. Infection in still active due to high $\beta_{hv}$. After 70 months, people have fully recovered to spread any infection.

In all the above cases, we clearly see that infection levels with human mobility is less than that immobile case. We remind readers that we have chosen S, E, and R individuals mobile, keeping infected immobile. We observe that exposed population have induced significant secondary waves, but also deplete the original region where they were spreading the infection. When the exposed people move to cells which have completely recovered, it is likely that the corresponding vector cells are also filled with susceptible mosquitoes. This is due to high correlations in the dynamics of $I_h, I_v$, and makes it is unlikely to spread the infection there. Susceptibles can get infected when they move to cells where the vectors in the neighborhood cells are infected. Mobility of recovered people will not affect the infection dynamics. A combination of the above factors have ensured that mobility has reduced or fully suppressed the infection levels in all the cases we have studied.

We however caution the reader that the spatio-temporal patterns are not universal. In the supplementary information, we study spread of Dengue with two other models of human mobility (Lévy Flight (LF) and Random movement (RM)) and compare it with the immobile case. We observe different spatio-temporal patterns for LM and EM models, but both suppress the infection levels. It is only in case of largely unrealistic RM model that we see enhancement of infection levels in the early phase, but it gets completely suppressed in a short time at all infection rates.

4 Summary and future outlook

In this paper we have studied the spatio-temporal dynamics of transmission of Dengue in human and vector population through reaction-diffusion and stochastic Cellular Automata (CA) formalism. We have divided human and vector population into SEIR-SEI compartments and distributed them on a bi-layer CA lattice. Our lattice size and human-vector population is big enough to simulate a large city. Coupling between the lattices is through vector bites, which transmits the disease across the layers. Mobility of vectors and humans spread the disease both within and across their lattice. As inputs to the model, we have used parameters from field studies, and vector population data from Ahmedabad city in India. We have imposed a statistical pattern of human mobility (Exponential distribution) on the human lattice to understand how mobility affects the spread of vector borne diseases.

For the case without human mobility, we find a close agreement between simulation results of infected humans and data on confirmed Dengue cases in Ahmedabad between 2006-2012. For low $\beta_{hv}$ (0.15), we could reproduce many observed peaks and roughly match the scale. For higher $\beta_{hv}$ many peaks can be reproduced, although their magnitude is much higher. We should note that much of the scale difference might arise due to absence of data on Dengue patients recovering without hospitalization.

Our main work is to understand the effect of human mobility on Dengue spreading has shown some surprising results. Movement of susceptible and exposed humans can actually decrease the incidence of epidemic. For low infection rates ($\beta_{hv}$), initially infection levels rise, however they get suppressed faster than the case with immobile humans. This suppression is due to multiple factors involving mobility of exposed and susceptible to regions which are less likely to spread the infections and also suppressing new infections in the regions where they originally hailed from. This observation is true even for Lévy flight model.

In this work we have made several assumptions for simplifying simulations and analysis. Some of these assumptions can be relaxed to give more realistic picture of the Dengue spread. Heterogeneous distribution of popu-
lation by changing human occupation in lattices based on ward level population density data and excluding regions like rivers and green areas. Exclusion of regions acts as a barrier and prevents secondary epidemic waves emerging from that part. Inclusion of environmental variables such as rainfall patterns can serve as proxy water clogging which acts as breeding sites for vectors. In this work, statistical patterns of human mobility has been taken from studies in US, UK and China. It is not clear (due to lack of literature), whether the same patterns hold good in developing countries such as India, where the current study focuses on. Mobility patterns are closely linked to transportation networks and traffic density. Inclusion of these factors, along with population density can give us more specific insights into how mobility affects spread of vector borne diseases in cities. We hope that the present work will motivate researchers to take up such studies in the future.

Acknowledgment

The authors would like to thank Vinod Reddy, Richard Koblenu, and Profs. Malavika Subramnyam, Bireswar Das, and Ravindra Amritkar for insightful discussions and suggestions. We specially thank Dr. V K Kohli, Assistant Entomologist Ahmedabad Municipal Corporation for providing us with data on Dengue incidences and mosquitoes in Ahmedabad city.

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