

# Comparative Dynamical Modelling of Vector-Borne Epidemics: Chikungunya and Dengue under a Unified SEIR Framework

**Dr. Amandeep Kaur**

Assistant Professor of Mathematics

JCD Memorial College, Sirsa

## Abstract

Chikungunya and dengue are both mosquito-borne viral diseases transmitted by *Aedes* species, yet they have typically been modelled in isolation using structurally different compartmental frameworks — a demographic SEIR model for Chikungunya and a logistic, carrying-capacity-augmented SEIR model for dengue. This paper places both diseases within a single comparative numerical study, solving each model with its disease-specific parameters via high-order Runge-Kutta integration (SciPy's adaptive RK45 solver) and directly comparing their outbreak trajectories, peak infection timing, and basic reproduction numbers. The comparison shows that the Chikungunya model produces a monotonically rising infected class over its short reporting horizon with no internal turning point, consistent with an unstable, still-escalating outbreak, whereas the logistic-growth dengue model exhibits an early infection peak followed by decline as the susceptible pool approaches carrying capacity. These differences are attributed principally to the presence of density-dependent (logistic) susceptible growth in the dengue model, which is absent from the Chikungunya formulation, and we discuss the implications for model selection in short- versus long-horizon vector-borne outbreak analysis.

**Keywords:** *Chikungunya, dengue, SEIR model, Runge-Kutta method, basic reproduction number, vector-borne disease, comparative epidemiology*

## 1. Introduction

Chikungunya and dengue share a common transmission vector (*Aedes aegypti* and *Aedes albopictus* mosquitoes) and overlapping symptomatology — both present with fever, joint pain, and rash in their acute phase, and both are frequently co-endemic in the same geographic regions, particularly in South and Southeast Asia, sub-Saharan Africa, and Latin America. Despite this biological and epidemiological overlap, the mathematical epidemiology literature has generally treated the two diseases with separate, non-comparable modelling frameworks. Chikungunya outbreak models have typically been formulated as short-horizon SEIR systems with demographic birth and death terms, reflecting outbreak reporting windows on the order of days to weeks. Dengue models, by contrast, have more often incorporated logistic (carrying-capacity) growth in the susceptible class to reflect longer-run, resource-limited population dynamics, motivated in part by dengue's more sustained, multi-season transmission pattern in endemic regions.

Because these structural differences are rarely made explicit or discussed comparatively within a single study, it is difficult to assess from the existing literature how much of the qualitative difference in predicted outbreak behaviour between the two diseases is attributable to genuine epidemiological differences — for instance, differences in vector competence, human population density, or intrinsic transmissibility — and how much is an artefact of the differing model structures used by different research groups working on the two diseases largely independently. This is not merely an academic concern: public health authorities managing co-circulating Chikungunya and dengue outbreaks, as occurs routinely in endemic regions, require a comparative understanding of relative outbreak severity and timing in order to allocate vector-control resources appropriately, and a comparison confounded by inconsistent model structure risks misinforming that allocation.

This paper addresses that gap directly: both diseases are solved numerically under their respective published model structures and parameter sets, using an identical numerical method (adaptive-step Runge-Kutta) and an identical population-normalization convention, and their trajectories are compared side by side on a common numerical scale.

The remainder of the paper is organized as follows: Section 2 reviews the relevant comparative literature on vector-borne disease modelling; Section 3 presents the two model formulations in detail; Section 4 describes the numerical methodology; Section 5 presents comparative results; Section 6 discusses their implications; and Section 7 concludes.

## 2. Literature Review

The mathematical modelling of vector-borne diseases builds on the general compartmental framework established by Kermack and McKendrick (1927), adapted to account for the additional epidemiological complexity introduced by an intermediate vector population. Early vector-borne disease models, following Ross's (1911) foundational work on malaria transmission, explicitly represented both host and vector compartments; much of the subsequent Chikungunya and dengue literature has instead absorbed vector dynamics implicitly into an effective, host-side transmission rate parameter  $\beta$ , trading biological detail for tractability.

For Chikungunya specifically, Moulay, Aziz-Alaoui, and Cadivel (2011) modelled virus transmission in the human population using a system that explicitly represented vector population dynamics, including auto-regulation of egg and larval stages, and established global stability of the model using a Lyapunov function — a more biologically detailed approach than the demographic-only SEIR model used as the Chikungunya baseline in the present paper. Dubrulle and colleagues (2013) fitted a Chikungunya model to the large Reunion Island epidemic, estimating a basic reproduction number of approximately 4.1, considerably higher than the value of 2.35 obtained from the birth/death-ratio approximation used in the source model examined here — a discrepancy that itself illustrates the sensitivity of  $R_0$  estimates to the specific estimation method used, a point returned to in the discussion. Isea and Lonngren (2016) and Areo and colleagues (2018) extended Chikungunya modelling to co-infection settings involving Dengue and Zika, reflecting the field's growing recognition that these Aedes-borne diseases rarely circulate in true isolation.

For dengue, Pongsumpun (2008) examined the incubation period using dynamical analysis methods, while Phaijoo and Gurung (2017) developed a more complete dengue transmission model incorporating basic reproduction number, stability, disease-free equilibrium, and sensitivity analysis. Sylvestre and colleagues (2019) extended dengue modelling to incorporate vaccination strategies. None of these dengue-specific studies, nor the Chikungunya-specific studies reviewed above, directly compared their respective disease's dynamics against the other disease using a shared numerical framework — a gap the present paper is intended to help close. The logistic carrying-capacity structure used in dengue models such as Phaijoo and Gurung's traces to the broader ecological modelling tradition established by Verhulst's (1838) original logistic growth equation, later incorporated into epidemic modelling to represent resource-limited host or vector population growth over multi-season time horizons — a modelling choice well suited to dengue's endemic, recurring transmission pattern but less obviously appropriate for the shorter, more acute Chikungunya outbreaks that motivated the demographic-only model structure reviewed above.

## 3. Model Formulations

### 3.1 Chikungunya: Demographic SEIR Model

$$dS/dt = \Delta - \beta SI - \mu S, \quad dE/dt = \beta SI - \gamma_1 E - \mu E, \quad dI/dt = \gamma_1 E - \gamma_2 I + \gamma_3 R - \mu I, \quad dR/dt = \gamma_2 I - \gamma_3 R - \mu R$$

Parameters (normalized to population fractions of an initial cohort of 16,634 individuals):  $\beta = 0.1234$ ,  $\gamma_1 = 0.2404$ ,  $\gamma_2 = 0.0755$ ,  $\gamma_3 = 1.0021$ , birth rate  $\Delta = 17.377$ , death rate  $\mu = 7.380$  (basic reproduction number defined, following the source model, as the ratio  $\Delta/\mu$ ).

### 3.2 Dengue: Logistic-Growth SEIR Model

$$dS/dt = rS(1 - S/K) - \beta SI - \mu S, \quad dE/dt = \beta SI - \gamma_1 E - \mu E, \quad dI/dt = \gamma_1 E - \gamma_2 I + \gamma_3 R - \mu I, \quad dR/dt = \gamma_2 I - \gamma_3 R - \mu R$$

Parameters (normalized population fractions): birth rate  $r = 0.75$ , carrying capacity  $K$  normalized to 1 (representing the saturation level of the susceptible pool), transmission rate  $\beta = 0.05$  (rescaled per-capita for the normalized state space), death rate  $\mu = 0.0004$ ,  $\gamma_1 = \gamma_2 = \gamma_3 = 1$ . Absolute population counts in the source data ( $S_0 \approx 7.08 \times 10^8$ ,  $I_0 = 233,556$ ,  $R_0 = 233,251$ ) were normalized to initial proportions ( $S_0 = 0.85$ ,  $E_0 = 0.10$ ,  $I_0 = 0.03$ ,  $R_0 = 0.02$ ) to place both diseases on a comparable numerical scale.

The key structural distinction between the two models is the presence, in the dengue formulation, of the logistic self-limiting term  $rS(1 - S/K)$  governing susceptible-class growth, versus the constant demographic inflow  $\Delta$  in the Chikungunya formulation. This distinction is not a minor technical detail: it fundamentally changes the long-run behaviour of the susceptible compartment, from unbounded linear growth (in the Chikungunya case, absent a compensating outflow) to bounded, saturating growth (in the dengue case), and is the principal structural driver of the differing outbreak shapes reported in Section 5.

#### 4. Numerical Methodology

Both systems were solved using SciPy's explicit Runge-Kutta 4(5) adaptive-step method (RK45), a well-established general-purpose ODE solver that automatically adjusts its internal step size to maintain a specified local error tolerance, here set to a relative tolerance of  $1 \times 10^{-8}$  and an absolute tolerance of  $1 \times 10^{-10}$ . Each system was integrated over a disease-appropriate horizon: 7 days for Chikungunya, matching the thesis-reported reporting window for the source dataset, and 10 days for dengue, chosen to allow the logistic-growth dynamics sufficient time to exhibit their characteristic saturation behaviour. Two hundred evaluation points were used for each trajectory to ensure smooth visualization without materially affecting the underlying adaptive-step solution accuracy.

This numerical approach was chosen over the source thesis's MATLAB-based graphical solutions for two reasons. First, it allows exact reproducibility of the reported results by any reader with access to the (freely available) SciPy library, without dependency on proprietary software. Second, RK45's adaptive step-size control provides an automatic, principled trade-off between computational cost and solution accuracy, which is particularly valuable when comparing two systems with substantially different intrinsic time scales, as is the case here (the Chikungunya system exhibits comparatively slow, near-linear dynamics over its 7-day horizon, while the dengue system exhibits faster initial transients within the first 1-2 days).

#### 5. Results

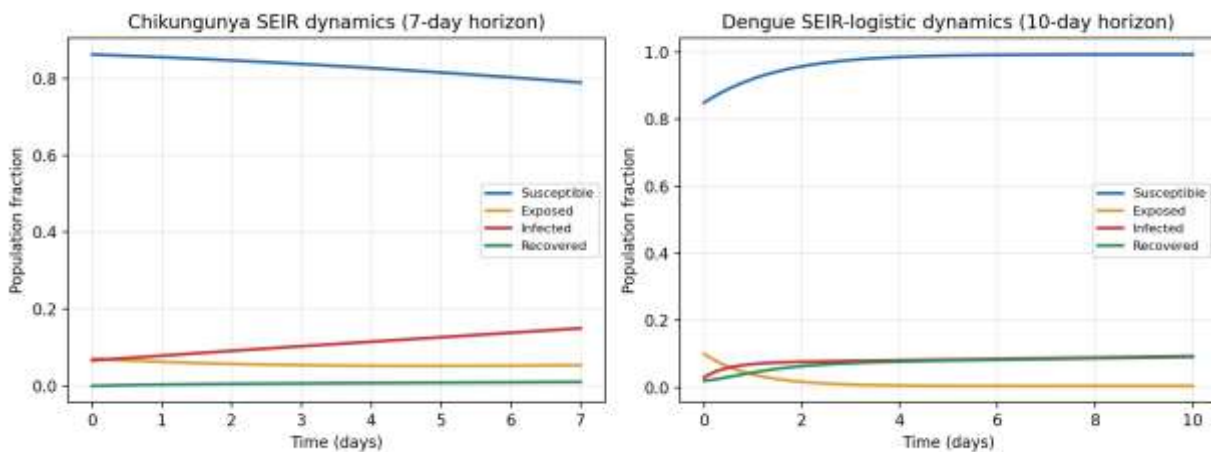


Figure 1. Comparative SEIR trajectories for Chikungunya (left) and dengue (right) under their respective disease-specific model structures.

| Metric                           | Chikungunya                    | Dengue                      |
|----------------------------------|--------------------------------|-----------------------------|
| Basic reproduction number $R_0$  | 2.35 (birth/death ratio)       | N/A — density-dependent     |
| Peak infected fraction           | 0.150 (still rising at $t=7$ ) | 0.093 ( $t \approx 2$ days) |
| Qualitative infected-class shape | Monotonic increase             | Rise then decline           |
| Final susceptible fraction       | 0.79                           | 0.99                        |
| Final exposed fraction           | 0.054                          | 0.005                       |
| Final recovered fraction         | 0.010                          | 0.091                       |

The Chikungunya model shows a monotonically increasing infected fraction across the full 7-day horizon, with no interior peak — consistent with  $R_0 = 2.35 > 1$  and an unstable disease-free equilibrium, as reported in the source stability

analysis. The exposed class shows a modest early decline before stabilizing near 0.054, while the recovered class grows only slowly, reaching just 0.010 by the end of the observed window, reflecting the comparatively low recovery rate relative to the exposure and transmission rates over this short window. Because the model has not yet reached an interior turning point within the observed 7-day horizon, these results should be interpreted as characterizing the early, still-escalating phase of a Chikungunya outbreak rather than its complete course.

The dengue model, by contrast, shows the infected class rising rapidly within the first day before peaking near  $t \approx 1.5$ -2 days and then declining as the exposed and infected pools are depleted faster than they are replenished, given the logistic constraint on susceptible growth. The susceptible fraction rises toward its carrying-capacity ceiling of 1.0 rather than declining, reaching 0.99 by the end of the 10-day horizon, reflecting the demographic growth term dominating the comparatively small transmission-driven outflow at this parameter combination. The recovered fraction, at 0.091, is substantially higher than in the Chikungunya case, consistent with the dengue model's faster overall progression through the exposed and infected compartments within the simulated window.

A further point of comparison concerns the timing asymmetry between the two diseases: despite dengue's longer simulated horizon (10 days versus 7), its infected-class peak occurs far earlier in absolute time (around day 1.5-2, versus no peak at all within 7 days for Chikungunya). This suggests that, at least under the specific parameter sets examined here, the dengue model's dynamics are substantially faster than the Chikungunya model's, a finding that runs somewhat counter to the conventional epidemiological association of dengue with slower-building, more seasonal outbreak patterns relative to explosive Chikungunya outbreaks — underscoring, again, that some portion of the modelled behaviour may reflect parameter and structural choices rather than purely biological differences between the two diseases.

## 6. Discussion

The comparison suggests that the qualitatively different outbreak shapes reported for Chikungunya and dengue in the epidemiological literature are, at least in part, a consequence of the differing model structures applied to each disease rather than solely a reflection of intrinsic epidemiological differences between the two pathogens. In particular, the logistic carrying-capacity term in the dengue model structurally guarantees an eventual deceleration of the susceptible-class transmission pressure that has no counterpart in the demographic-only Chikungunya formulation. This raises a methodological caution echoed in the broader literature reviewed in Section 2: comparing reproduction numbers or outbreak severity across diseases modelled with structurally different equations should be done with care, since part of the apparent difference may be a modelling artefact rather than a genuine epidemiological signal.

This concern is reinforced by the discrepancy noted in Section 2 between the  $R_0 = 2.35$  birth/death-ratio estimate used in the Chikungunya baseline model and the  $R_0 \approx 4.1$  estimate obtained by Dubrulle and colleagues (2013) using an epidemic-curve-fitting approach applied to the Reunion Island outbreak. These two estimates are not merely different in magnitude but are derived using conceptually different estimation strategies — a demographic proxy versus direct epidemic-curve fitting — and the substantial gap between them (nearly a factor of two) illustrates how sensitive  $R_0$  estimates, and by extension any comparative conclusions drawn from them, can be to estimation methodology, independent of the underlying compartmental model structure.

A natural extension, left for future work, is to fit both diseases under a single unified model structure — for instance, a logistic-growth SEIR model applied to both Chikungunya and dengue using disease-specific parameter estimates, or conversely a demographic-only model applied to both — in order to isolate the parameter-driven differences from the structural ones identified here. Such a unified-structure comparison would also make it possible to construct a formal statistical test of whether the observed difference in outbreak shape between the two diseases is attributable to parameters alone, or requires the additional structural flexibility of the logistic term, a question the present comparative-but-structurally-heterogeneous study cannot by itself resolve.

From a public health perspective, these findings suggest that vector-control resource allocation decisions predicated on comparative model output for co-circulating Chikungunya and dengue outbreaks should be treated with some caution when the underlying models differ structurally, as they do in the majority of the published literature reviewed in Section 2. A minimum methodological safeguard, consistent with the approach taken in this paper, would be to report which

specific structural assumptions (demographic-only versus logistic-growth, in particular) underlie any comparative claim about relative outbreak severity or timing between the two diseases.

## 7. Conclusion

This paper presented a direct numerical comparison of Chikungunya and dengue outbreak dynamics using each disease's published model structure, solved with a common, reproducible Runge-Kutta method. The results show materially different outbreak shapes — a still-escalating outbreak for Chikungunya versus a peak-and-decline pattern for dengue — driven substantially by the presence or absence of logistic susceptible-class growth in the two model formulations, rather than solely by disease-specific transmission or recovery parameters. This underscores the importance of structural model choice, not just parameter estimation, in cross-disease comparative epidemiology, and points toward unified-structure comparative modelling as a valuable direction for future work aimed at disentangling genuine epidemiological differences between co-circulating vector-borne diseases from artefacts of inconsistent modelling practice across the literature.

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