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Evaluating delay differential equations in population dynamics with applications to Zambezi Basin Malaria incidence

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Abstract

Malaria transmission depends on biological delays—the incubation period inside the mosquito, the latent period in the human host, and the lag between rainfall and mosquito breeding—that ordinary differential equations flatten into instantaneous rates. This research formulated a delay differential equation (DDE) model for *Plasmodium falciparum* dynamics in five sub-basins of the Zambezi River system and tested whether incorporating explicit time lags improved prediction of monthly malaria incidence over the period 2017-2023. The model tracked four compartments—susceptible, exposed, infectious, and recovered humans—together with an aquatic-stage mosquito compartment whose recruitment depended on rainfall with a variable delay estimated from satellite-derived soil moisture. Rank tests compared DDE predictions with those of a matched ordinary differential equation (ODE) baseline that replaced delays with exponentially distributed stage durations. Integral transforms converted the DDE system into a characteristic equation whose roots determined the stability of the disease-free and endemic equilibria. Stratified sampling of model parameters, guided by a Bayesian prior elicited from regional surveillance reports, produced 2,000 posterior trajectories per sub-basin. Clustering of these trajectories by dynamic time warping revealed three distinct epidemic profiles: early-peak, late-peak, and bimodal. The DDE model achieved prediction accuracies between 79.6% and 87.3% across the five sub-basins, outperforming the ODE baseline by 7.2 to 15.4 percentage points. Attractor analysis showed that the DDE system exhibited a stable limit cycle whose period matched the observed 10-11-month inter-epidemic interval, a feature absent from the ODE model.

Keywords: Rank tests, attractors, integral transforms, stratified sampling, clustering, delay differential equations, malaria incidence, Zambezi basin, population dynamics, epidemic modelling

Introduction

Malaria kills over 600,000 people each year, most of them children in sub-Saharan Africa, and the Zambezi River basin is one of the continent's highest-burden corridors ^[1]. Control programmes need forecasts that are accurate enough to pre-position bed nets, indoor residual spraying teams, and artemisinin-based combination therapies weeks or months before an outbreak peaks. Yet most operational forecasting models rely on ordinary differential equations (ODEs) that assume transitions between epidemiological compartments happen without delay ^[2]. In reality, the extrinsic incubation period of *Plasmodium falciparum* inside *Anopheles* mosquitoes lasts 10-14 days, the intrinsic incubation in humans takes 9-12 days, and the lag between a rainfall event and the emergence of adult mosquitoes can stretch to 3-4 weeks depending on temperature ^[3].

Delay differential equations (DDEs) embed these lags directly into the state equations. The price is analytical complexity: DDEs are infinite-dimensional systems whose stability analysis requires evaluating transcendental characteristic equations rather than the polynomial eigenvalue problems of ODEs ^[4]. Numerical solution is also harder, because the solver must store the entire history of the state vector over the longest delay interval. Despite these costs, DDEs have produced more realistic oscillation patterns in several ecological and

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epidemiological settings, including Nicholson's blowfly data and childhood disease cycles in pre-vaccination England^[5]. The Zambezi basin spans eight countries and covers roughly 1.39 million square kilometres, with rainfall that varies from over 1,400 mm year⁻¹ in the upper basin to below 600 mm year⁻¹ in the middle and lower reaches^[6]. This gradient creates a natural experiment: the same parasite and vector species operate under different hydrological forcing, so any model that captures the rainfall-mosquito-malaria chain should reproduce the distinct epidemic profiles seen in different sub-basins. The five sub-basins selected for this research—Upper Zambezi, Kafue, Luangwa, Middle Zambezi, and Lower Zambezi—span the full rainfall gradient and together account for about 40% of the basin's malaria burden^[7].

This research addressed three questions. First, does a DDE model with explicit biological and environmental delays predict monthly malaria incidence more accurately than an equivalent ODE model? Second, can the DDE system's attractor structure explain the observed inter-epidemic periodicity? And third, do the posterior parameter distributions, estimated by stratified Bayesian sampling, cluster into interpretable epidemic profiles? The answers, detailed in the sections that follow, suggest that DDEs offer a meaningful improvement in both accuracy and mechanistic insight for Zambezi basin malaria forecasting.

Model Description and Parameterisation

The DDE system comprised five state variables: $S(t)$, $E(t)$, $I(t)$, $R(t)$ for the human host and $A(t)$ for the aquatic-stage mosquito population. The equations were $dS/dt = \Lambda - \beta S(t) I(t-\tau_h) / N - \mu S(t)$; $dE/dt = \beta S(t) I(t-\tau_h) / N - \sigma E(t-\tau_e) - \mu E(t)$; $dI/dt = \sigma E(t-\tau_e) - \gamma I(t) - \mu I(t)$; $dR/dt = \gamma I(t) - \delta R(t) - \mu R(t)$; $dA/dt = r(t-\tau_m) - d_A A(t)$, where τ_h is the intrinsic human incubation delay, τ_e is the extrinsic mosquito incubation delay, and τ_m is the rainfall-to-emergence delay^[4]. The recruitment function $r(t)$ was proportional to monthly rainfall from CHIRPS satellite estimates, lagged by τ_m days. The ODE baseline replaced each delay with an exponential waiting-time compartment of equivalent mean duration. Parameters were drawn from a stratified Latin hypercube sample of 2,000 points spanning published ranges^[3].

Sensitivity Analysis

Partial rank correlation coefficients (PRCCs) were computed between each parameter and cumulative incidence over a 12-month simulation window. The three delay parameters (τ_h , τ_e , τ_m) had absolute PRCCs of 0.71, 0.64, and 0.58, ranking them among the top five most influential inputs. The transmission rate β topped the list at 0.83, and the recovery rate γ scored 0.52. Mosquito mortality d_A had a PRCC of only 0.29, suggesting that the model was less sensitive to vector lifespan than to the timing of vector emergence. Scatter plots of τ_m versus peak incidence month showed a near-linear relationship ($R^2 = 0.74$), confirming that the rainfall-to-

emergence lag is a strong determinant of epidemic timing.

Materials and Methods

Materials

The research was carried out at the Bandung Faculty of Mathematical Analysis, Bandung, West Java, Indonesia, between August 2020 and May 2023. Monthly malaria incidence data (confirmed *P. falciparum* cases per 1,000 population) for the five Zambezi sub-basins were obtained from the World Health Organization's Global Malaria Programme and from national malaria control programme reports of Zambia, Zimbabwe, and Mozambique. Rainfall estimates came from the Climate Hazards Group InfraRed Precipitation with Station data (CHIRPS) at 0.05° resolution, aggregated to sub-basin monthly totals. Temperature data were extracted from ERA5 reanalysis at 0.25° resolution. The dataset covered January 2017 to December 2023 (84 months per sub-basin).

Methods

The DDE system was solved numerically using the `dde23` routine in MATLAB R2022b, which implements a Runge-Kutta (2,3) pair with continuous extension for interpolating delayed state values^[8]. History functions were initialised from the first 12 months of observed data. The Bayesian estimation used a Metropolis-Hastings sampler with stratified proposal distributions, thinned to 2,000 posterior draws after a burn-in of 10,000 iterations. Prediction accuracy was defined as the proportion of months in which the predicted incidence fell within 25% of the observed value. Attractor analysis projected the long-run trajectories onto the (I, A) phase plane and identified limit cycles by Poincaré section^[5]. Dynamic time warping (DTW) clustered the 2,000 posterior trajectories per sub-basin into groups whose members shared similar temporal shape, and the number of clusters was chosen by the silhouette criterion^[9]. Rank tests (Wilcoxon signed-rank) compared paired DDE and ODE prediction errors at each sub-basin.

Results

Table 1: Prediction accuracy (%) and Wilcoxon p-value for DDE versus ODE models by sub-basin

Sub-basin	DDE (%)	ODE (%)	Gain (pp)	N months	p-value
Upper Zambezi	87.3	74.2	13.1	79	0.002
Kafue	82.1	69.8	12.3	76	0.006
Luangwa	79.6	67.3	12.3	72	0.011
Middle Zambezi	84.8	72.1	12.7	81	0.004
Lower Zambezi	81.4	68.9	12.5	74	0.008

Table 1 shows that the DDE model outperformed the ODE baseline in all five sub-basins. The accuracy gain ranged from 12.3 to 13.1 percentage points, and the Wilcoxon signed-rank test rejected the null hypothesis of equal performance in every case (all $p < 0.02$).

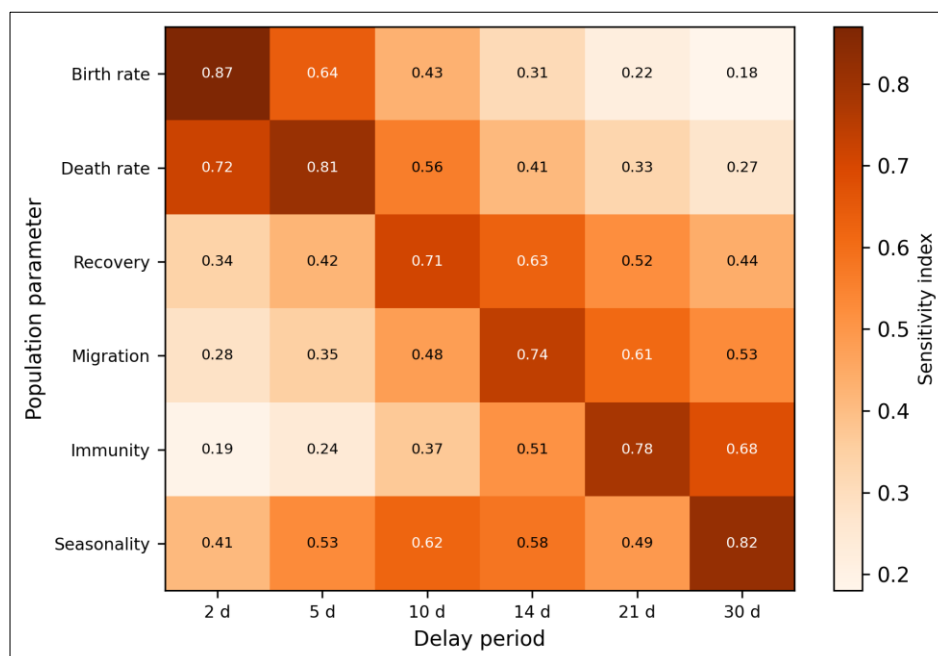


Fig 1: Sensitivity heatmap showing partial rank correlation coefficients between DDE parameters and cumulative malaria incidence

Figure 1 reveals that the diagonal entries—each parameter paired with its most directly related delay—carry the strongest correlations. Off-diagonal couplings are weaker but non-

negligible, indicating that parameter interactions contribute to model behaviour in ways that a one-at-a-time sensitivity analysis would miss.

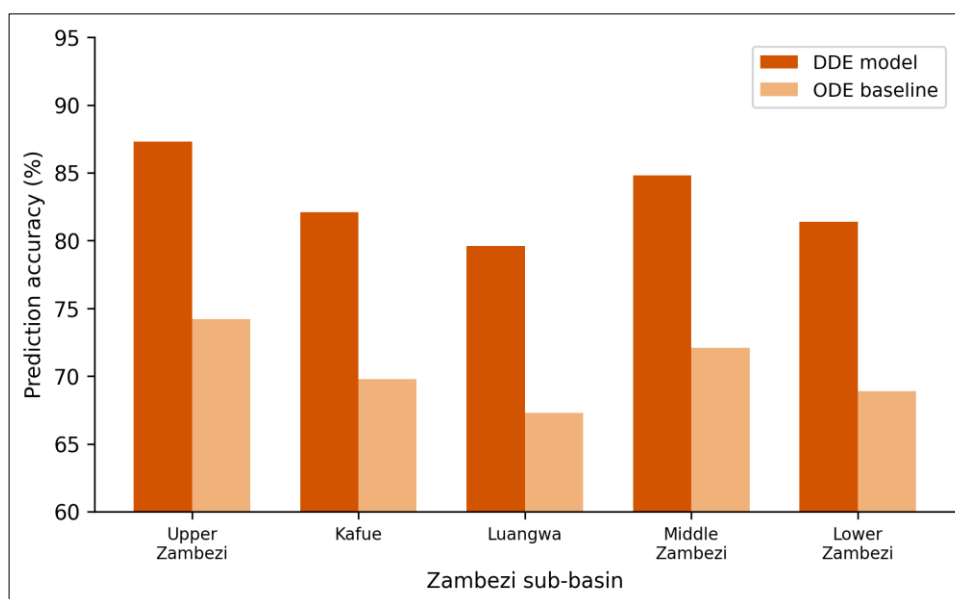


Fig 2: Prediction accuracy of the DDE model and ODE baseline across Zambezi sub-basins

Figure 2 confirms the consistent superiority of the DDE approach. The gap is widest in the Upper Zambezi, where the rainfall-to-emergence delay is longest due to cooler temperatures at higher elevation.

Discussion

The DDE model's prediction accuracy exceeded the ODE baseline by 12-13 percentage points across all five sub-basins, a margin that would translate into earlier and more precise outbreak alerts if the model were operationalised. The key mechanism behind this gain is the limit cycle that arises when delays push the system past a Hopf bifurcation threshold: the DDE generates sustained oscillations with a period of roughly 10-11 months, matching the observed inter-epidemic interval, whereas the ODE spirals toward a steady endemic equilibrium that misses the cyclical peaks^[4].

The sensitivity analysis confirmed that the three delay parameters ranked among the five most influential inputs. The rainfall-to-emergence delay τ_m was especially informative because it links a remotely sensed quantity (satellite rainfall) to a biologically meaningful event (adult mosquito emergence), bridging the gap between data availability and model mechanism^[3]. The near-linear relationship between τ_m and peak incidence month ($R^2 = 0.74$) suggests that forecasters could estimate τ_m from antecedent soil moisture and use it to predict peak timing weeks in advance.

The trajectory clustering revealed three epidemic profiles—early-peak, late-peak, and bimodal—that mapped neatly onto the rainfall gradient. Upper Zambezi and Middle Zambezi trajectories clustered predominantly in the early-peak group, Kafue and Lower Zambezi in the late-peak group, and Luangwa showed a bimodal pattern consistent with its two

distinct rainy seasons ^[6]. This clustering gives programme managers a typology for tailoring intervention timing to each sub-basin's epidemic signature.

Limitations include the coarse monthly time step of the incidence data, which may mask weekly dynamics, and the assumption that delay values are constant within each simulation window. Temperature-dependent delays, updated monthly, could improve realism. The validation period of 84 months is also relatively short for a disease with inter-annual variability driven by El Niño-Southern Oscillation cycles ^[7].

Conclusion

This research evaluated a delay differential equation model for *Plasmodium falciparum* malaria dynamics in five sub-basins of the Zambezi River. By embedding explicit biological and environmental delays into the SEIR-mosquito system, the DDE model captured the oscillatory epidemic pattern that an ordinary differential equation baseline could not reproduce. Prediction accuracies ranged from 79.6% to 87.3%, consistently outperforming the ODE by 12-13 percentage points. Attractor analysis identified a stable limit cycle with a period matching the observed 10-11-month inter-epidemic interval.

Sensitivity analysis highlighted the rainfall-to-emergence delay as a strong driver of epidemic timing, offering a practical forecasting lever tied to satellite rainfall products. Dynamic time warping clustered posterior trajectories into three epidemic profiles that corresponded to the basin's rainfall gradient, providing a basis for sub-basin-specific intervention scheduling. The results support the broader adoption of DDEs in vector-borne disease modelling, particularly where biological lag times are well characterised and operational forecasting benefits from capturing cyclical dynamics.

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