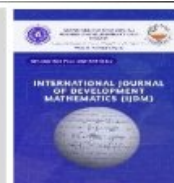




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Threshold Dynamics, Stability and Sensitivity Analysis of a Multi-host West Nile Virus Model Incorporating Lateral Transmission in Hosts and Vertical Transmission in the Vector Population

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Abstract

This paper formulates and analyses a twenty-two compartment deterministic model with constant control that divides the avian hosts into corvid and non-corvid classes, resolves the immature vector stages, carries an environmentally persistent viral compartment, and admits predatory and non-predatory bird-to-bird transmission, transfusion and transplant transmission in humans, and vertical transmission in mosquitoes. Positivity and boundedness of solutions are established and a biologically feasible invariant region is identified. Two disease-free equilibria are obtained, and the non-trivial one is shown to exist only when a demographic vector reproduction ratio exceeds unity. The basic reproduction number is derived by the next generation matrix method and is shown to decompose into four sub-reproduction numbers associated with the mosquito-bird cycle, the bird-to-bird and environmental pathway, vertical transmission, and human spillover, the human component being structurally decoupled from the enzootic cycle. The disease-free equilibrium is proved to be locally asymptotically stable by the Routh–Hurwitz criterion and globally asymptotically stable by the Castillo-Chavez framework, while a logarithmic Lyapunov function together with LaSalle’s invariance principle establishes global asymptotic stability of the endemic equilibrium. Sensitivity analysis identifies vector survival, biting and transmission intensity and environmental viral persistence as the dominant drivers of transmission, and numerical simulations confirm the analytical findings.

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1 Introduction

West Nile virus (WNV), a vector-borne arbovirus of the flavivirus genus, is the causative agent of the resurgent zoonotic disease known as West Nile disease. The virus was first isolated in a woman in the West Nile district of Uganda in 1937, from which it takes its name (Smithburn et al., 1940). It is transmitted principally by mosquitoes of the *Culex* genus, which act as the primary vector, and it is the most widely distributed neuroinvasive arboviral illness observed worldwide (Ciota & Kramer, 2013). Severe cases may be life-threatening, particularly in immuno-compromised, younger and elderly individuals (Kramer et al., 2007), as infection can result in inflammation of the brain and spinal cord in birds, humans and other mammals. The disease has spread from Africa to Europe, the Americas, the Middle East and West Asia (WHO, 2017), and is becoming endemic in several regions of the world (Sule et al., 2018; Habarugira et al., 2020). Between 1999 and 2023, 86,758 confirmed human cases were reported, a figure widely believed to understate the true burden because of under-reporting (CDC, 2024c). There are increasing calls to designate West Nile disease formally as a neglected tropical disease, since it affects vulnerable populations in areas where mosquito-borne diseases are common while receiving comparatively little attention from researchers, clinicians and policy makers (Ronka et al., 2021).

The principal transmission cycle of WNV is the bird-mosquito-bird cycle. Birds are the primary reservoir of the virus, with more than three hundred species found to carry it (CDC, 2024e), and their migration together with the commercial transportation of infected birds and mosquitoes is a key factor in the rapid global spread of the disease (Chatterjee et al., 2008; Powell & Tabachnick, 2013). The avian population has been categorised into two groups, one of which is more susceptible to the disease, and therefore suffers greater morbidity and mortality and is more competent in transmission than the other (WOAH, 2024; CWHL, 2024). Modelling attention has not, however, been given to lateral transmission within the avian population. Predatory interaction between birds of prey or scavengers, which may be susceptible, and other infected or dead infected birds does occur and causes viral transmission (Komar et al., 2003; Nemeth & Kunkel, 2024; NPS, 2025). Experimental evidence further shows that birds can be infected through virus shed orally and cloacally when they are kept in close quarters, which indicates that mosquitoes, though the primary vectors, are not the sole cause of transmission within the avian population.

Female mosquitoes such as *Culex pipiens*, *Culex univittatus* and *Culex antennatus* transmit the disease to susceptible humans and birds after ingesting the virus during a blood meal taken on infected birds (Hayes et al., 2005a). Not all of the vector population, however, requires contact with infected birds in order to become infected, since vertical transmission of the virus within the mosquito population has been recorded repeatedly (Miller et al., 2000; Dohm et al., 2002; Anderson et al., 2014; Nelms et al., 2013). Given a short life cycle that is significantly affected by seasonality, mosquitoes are able to sustain the virus by laying infected eggs which survive the winter and hatch infected progeny once ambient temperatures become favourable. Temperature and season are therefore essential determinants of the spread of mosquito-borne disease (Garcia-Carrasco et al., 2022; ECDPC, 2025), since they govern mosquito development from egg-hatching to maturation as well as transmission viability (Ciota et al., 2014).

Humans and other mammals are infected incidentally through the bite of an infected mosquito but do not form part of the transmission cycle, because they maintain a viral load in the blood stream that is insufficient to infect a blood-feeding mosquito (Clark & Schaefer, 2023). This does not, however, preclude humans from perpetuating the disease within the human population, since the virus is transmitted horizontally through organ transplantation (CDC, 2024d; Kusne & Smilack, 2005), blood transfusion (Jimenez et al., 2021) and, rarely, through breast feeding by infected mothers (Hinckley et al., 2007). Roughly one in five infected persons develops West Nile fever, and about one in every hundred and fifty develops a severe and occasionally fatal neuroinvasive illness (Kramer et al., 2007; Barret, 2018b; CDC, 2024a).

Mathematical modelling has become an essential tool for analysing the behaviour of the disease and predicting future trends. Numerous studies have employed compartmental models for this purpose. Wonham et al. (2004) carried out an invasion analysis of a single-season mosquito-bird model, and Cruz-Pacheco et al. (2005) analysed the dynamics of the mosquito-bird cycle and quantified the threshold governing invasion. Bowman et al. (2005)

assessed control strategies against the virus, while Liu et al. (2005) examined spatial spread in a patchy environment and showed that the long-range dispersal of birds is responsible for the observed discontinuous spatial spread. Abdelrazec et al. (2014) modelled the mosquito-bird cycle for a single season with the avian population separated into corvid and non-corvid groups, established the reproduction number, and detected a backward bifurcation in which a stable endemic equilibrium coexists with the stable disease-free equilibrium, so that the ability of the virus to spread may depend on the initial sizes of the subpopulations. Bergsman et al. (2016) built a model with resident and migratory birds and found by forward sensitivity analysis that the average number of bites by an infectious mosquito is the most crucial component of the reproduction number, and that a small reduction in the average lifespan of a mosquito has a substantial effect on that number. Blayneh et al. (2010) and Abdelrazec et al. (2015) examined backward bifurcation and optimal control in the transmission dynamics of the virus with seasonality, while Moschini et al. (2017) modelled the interaction of the virus with seasonal effects in *Culex pipiens* mosquitoes, and Fesce et al. (2023) quantified the parameters most critical for predicting infection dynamics. Agosto et al. (2022) constructed a model that included the mosquito life stages together with larviciding and adulticiding, and Zhou et al. (2019) proposed a two-threshold policy to interrupt transmission to birds. Bhowmick et al. (2020, 2023) developed weather-driven models of the spread of the virus in Germany and reported strong correlations between secondary infections in humans and in birds.

Notwithstanding this substantial body of work, some aspects of the transmission dynamics remain unexplored. In the model of Bhowmick et al. (2023), which the present work modifies, the interacting bird, mosquito and human populations exhibit only a single transmission pathway, namely cross-transmission. Important as that pathway is, the model does not take account of the fact that WNV can also be transmitted through lateral routes, that is, within the bird population when a susceptible bird interacts carnivorously with an infected bird, and within the human population through blood transfusion or organ transplantation, even though these routes do not operate at the same magnitude as cross-transmission. The same deficiency characterises other existing models such as Abdelrazec et al. (2014), and this constitutes the gap which the present work addresses. Accordingly, this paper proposes and analyses a WNV model which incorporates lateral transmission in both the bird and human populations, vertical transmission in the vector population, an environmentally persistent viral compartment, and constant control. The objectives are to establish the positivity and boundedness of solutions, to obtain the disease-free and endemic states, to derive the basic reproduction number and its transmission pathways, to carry out the local and global stability analyses of both equilibria, to identify by sensitivity analysis the parameters that drive transmission, and to illustrate the analytical results by numerical simulation.

The remainder of the paper is organised as follows. Section 2 formulates the model and states its variables and parameters. Section 3 presents the analysis, comprising positivity, boundedness, the equilibrium states, the basic reproduction number and the local and global stability results. Section 4 reports the sensitivity analysis, Section 5 presents the numerical simulations, Section 6 discusses the findings and Section 7 concludes.

2 Model formulation

2.1 Description of the model

A deterministic compartmental model consisting of a system of non-linear ordinary differential equations is proposed. The system comprises the vector and host populations together with the viral population in the environment, and the compartments record the disease status of all populations at time t . The mosquito population, that of *Culex pipiens*, is given by

$$N_M(t) = S_E(t) + I_E(t) + S_L(t) + I_L(t) + S_M^F(t) + E_M^F(t) + I_M^F(t), \quad (1)$$

the reservoir host (bird) population by

$$N_B(t) = S_B^C(t) + S_B^N(t) + E_B^C(t) + E_B^N(t) + I_B^C(t) + I_B^N(t) + R_B^C(t) + R_B^N(t), \quad (2)$$

and the incidental host (human) population by

$$N_H(t) = S_H(t) + E_H(t) + I_H^A(t) + I_H^N(t) + I_H^I(t) + R_H(t), \quad (3)$$

while $D(t)$ denotes the population of pathogens in the environment. All populations except those of dead birds and of pathogens are replenished by births and depleted either by natural or by induced deaths, whereas the environmental viral load increases through shedding by live infectious birds and through the carrion of infected birds, and decreases by viral decay.

The mosquito population is compartmentalised so as to display the developmental stages through which the virus is transmitted vertically. The susceptible egg class S_E is replenished by $\varpi\Phi[S_M^F + E_M^F + (1 - \tau)I_M^F]$ and depleted by decay μ_E and by hatching νS_E ; the infected egg class I_E follows the same course, being replenished by $\varpi\Phi\tau I_M^F$ and depleted by $\mu_E I_E$. Eggs hatch into susceptible and infected larvae, S_L and I_L , at the hatching rate ν , and larvae mature into the corresponding adult classes at the maturation rate m_L , subject to the density-dependent limitation

$$\Phi = \max \left\{ 0, 1 - \frac{N_L}{K_L} \right\}, \quad (4)$$

in which N_L is the current larval density and K_L the larval carrying capacity. Susceptible adult female mosquitoes progress to the exposed class at the force of infection $\bar{\lambda}_M^F S_M^F$, where

$$\bar{\lambda}_M^F = \frac{(\beta_{1M} I_B^C + \beta_{2M} I_B^N) \beta \theta_M}{\varphi_F N_B + N_H}, \quad (5)$$

the denominator representing the total blood meal available to adult female mosquitoes on the assumption that mosquitoes take blood only from birds and humans. Here $\beta_{1M} > \beta_{2M}$, on account of the higher viraemia of corvid relative to non-corvid birds (CWHL, 2024). Exposed mosquitoes become infectious after the incubation period η_M , and, because infected larvae mature into infected adults at the rate $abm_L I_L$, the transovarial route feeds the infectious vector class directly.

The avian population is recruited at the rates $p\Lambda_B$ and $(1 - p)\Lambda_B$ into the susceptible corvid and non-corvid classes respectively, with $\Lambda_B = \mu_B N_B$. In each category, susceptible birds move to the exposed class either at the vector-borne force of infection

$$\bar{\lambda}_B^i = \frac{\beta_{Bi} \beta \theta_M \varphi_F I_M^F}{\varphi_F N_B + N_H}, \quad i = C, N, \quad (6)$$

or at the consumption-dependent force of infection $\lambda_B^i = \lambda_i^c + \lambda_i^p$, in which

$$\lambda_i^c = \frac{\beta_{Bi} \pi_{ij} D}{N_B}, \quad \lambda_i^p = \frac{I_B^C}{1 + I_B^C/K_{sat}} d_1 \beta_B^C \vartheta_1 + \frac{I_B^N}{1 + I_B^N/K_{sat}} d_2 \beta_B^N \vartheta_2, \quad (7)$$

λ_i^c being the non-predatory consumption force of infection, scaled by the reservoir population N_B , and λ_i^p the predatory consumption force of infection. Exposed birds progress to the infectious class at the incubation rate η_B^i and remain infectious for an average duration $1/\gamma_B^i$. A fraction q_i of recovered birds returns to the susceptible class at the immunity loss rate ρ_B^i , the infected classes are depleted by disease-induced and natural mortality, and live infectious birds shed virus into the environment at the rates ε_i .

Humans are recruited into the susceptible class at the rate $\Lambda_H = \mu_H N_H$. Susceptible humans move to the exposed class either at the vector-borne force of infection $\bar{\lambda}_H S_H$ or at the transfusion and transplant dependent force of infection $\lambda_H S_H$, where

$$\bar{\lambda}_H = \frac{\beta_H \beta \theta_M I_M^F}{\varphi_F N_B + N_H}, \quad \lambda_H = (p_1 a_{H1} + p_2 a_{H2}) \frac{I_H^A}{N_H}. \quad (8)$$

A proportion κ of exposed individuals moves to the asymptomatic class I_H^A after the incubation period η_H , and the complementary fraction $(1 - \kappa)$ moves to the non-neuroinvasive class I_H^N at the same rate. Asymptomatic

individuals either recover at the rate γ_H or progress to the non-neuroinvasive class at the rate ε , according to differences in immune response. Non-neuroinvasive individuals either progress to the neuroinvasive class at the rate σ_H or recover under the saturated supportive treatment function

$$\gamma_H^N(\varsigma) = \frac{\xi_1 I_H^N}{1 + v_1 I_H^N}, \quad \gamma_H^I(\varsigma) = \frac{\xi_2 I_H^I}{1 + v_2 I_H^I}, \quad (9)$$

in which ξ_1 and ξ_2 are the cure rates and v_1 and v_2 measure the delay in receiving supportive treatment arising from lack of awareness, reluctance to seek medical attention or improper diagnosis. Here $\xi_1 \geq \xi_2$, because the disease is more difficult to manage at an advanced stage. A proportion h of recovered individuals returns to the susceptible class through waning immunity at the rate ζ . Finally, the vector, reservoir and incidental host abundances are linked by the vector-host ratios

$$\alpha_B = \frac{N_M^0}{N_B^0}, \quad \alpha_H = \frac{N_M^0}{N_H^0}, \quad (10)$$

so that $N_M^0 = \alpha_B N_B^0$, with α_H a derived quantity; these ratios fix the abundances and hence the blood-meal index $\varphi_F N_B^0 + N_H^0$.

Figure 1 presents the schematic flow of the model with corvid and non-corvid birds, vertical transmission in the mosquito population and lateral transmission within the bird and human populations, under constant control. The state variables together with the initial values used in the numerical simulation are listed in Table 1, and the parameters, with their baseline values, ranges and sources, are listed in Table 2.

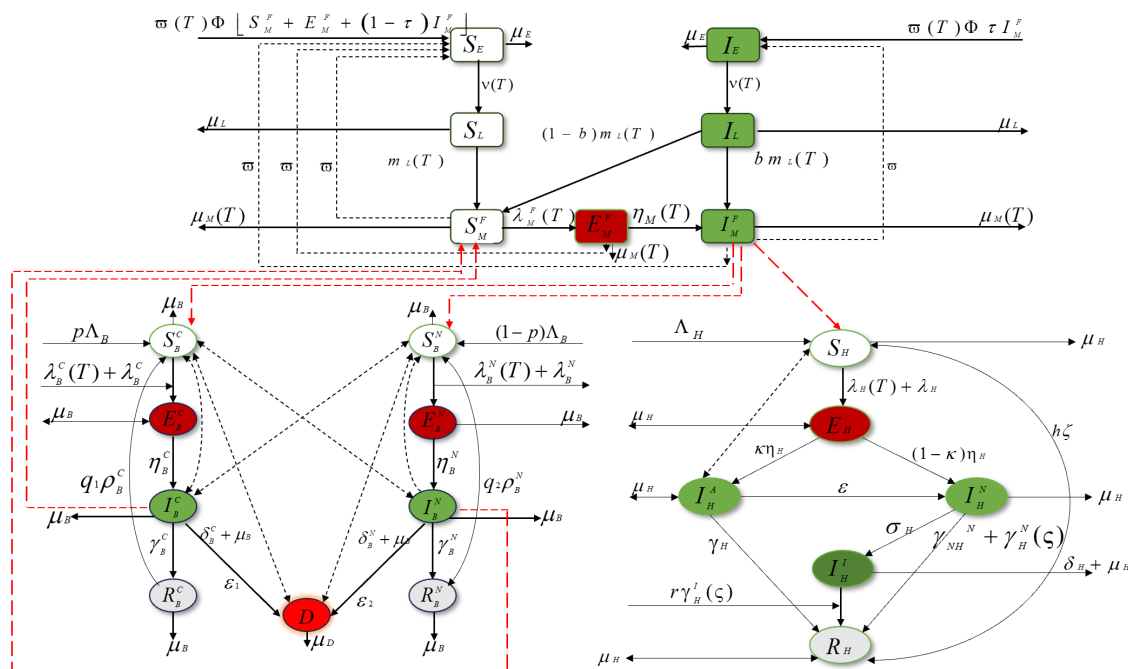


Figure 1. Schematic of the WNV model with corvid and non-corvid birds, lateral transmission in hosts and vertical transmission in the vector population, with constant control.

Table 1. Model variables, their descriptions and the initial values used for the numerical simulation.

Var.	Description	Value	Source	Var.	Description	Value	Source
$S_E(t)$	Susceptible mosquito egg	8.20×10^7	Assumed	$S_B^N(t)$	Susceptible non-corvid bird	35000	Estimated
$I_E(t)$	Infected mosquito egg	300	Assumed	$E_B^N(t)$	Exposed non-corvid bird	90000	Assumed
$S_L(t)$	Susceptible mosquito larva	2×10^7	Estimated	$I_B^N(t)$	Infected non-corvid bird	50000	Assumed

Continued on next page

Table 1 continued from previous page

Var.	Description	Value	Source	Var.	Description	Value	Source
$I_L(t)$	Infected mosquito larva	200	Assumed	$R_B^N(t)$	Recovered non-corvid bird	250000	Assumed
$S_M^F(t)$	Susceptible mosquito	7.80×10^8	Calculated	$S_H(t)$	Susceptible human	3.00×10^7	Nat. Bureau of Statistics (2026)
$E_M^F(t)$	Exposed mosquito	25000	Assumed	$E_H(t)$	Exposed human	30	Assumed
$I_M^F(t)$	Infected mosquito	120000	Assumed	$I_H^A(t)$	Asymptomatic infected human	10	Assumed
$S_B^C(t)$	Susceptible corvid bird	7.70×10^7	Calculated	$I_H^N(t)$	Non-neuroinvasive human	5	Assumed
$E_B^C(t)$	Exposed corvid bird	50	Assumed	$I_H^I(t)$	Neuroinvasive human	2	Assumed
$I_B^C(t)$	Infected corvid bird	80	Assumed	$R_H(t)$	Recovered human	100	Assumed
$R_B^C(t)$	Recovered corvid bird	200	Assumed	$D(t)$	WNv contaminated environment	2×10^7	Assumed

Table 2. Model parameters, descriptions, baseline values, ranges and sources.

Parameter	Description	Baseline	Range	Source
Λ_H	Recruitment rate for human	1072	–	Calculated
Λ_B	Bird recruitment rate	$0.000365N_B$	–	Assumed
ϖ	Oviposition rate	33.8	Varies	Marini et al. (2020)
μ_H	Human natural death rate	3.91×10^{-5}	–	Sweilam et al. (2019)
μ_E	Egg natural decay rate	0.125	0.0625–0.1875	Assumed
μ_L	Larva natural mortality rate	0.1041	0.071–0.25	Okuneye & Gumel (2016)
μ_M	Adult mosquito natural mortality rate	0.0595	Varies	Okuneye & Gumel (2016)
μ_B	Bird natural mortality rate	0.000365	–	Calculated from Mbaoma et al. (2024)
μ_D	WNv decay rate	0.55	0.1–1.0	Assumed
β_{1M}	Transmission probability from corvid bird to mosquito	0.70	0–1	Bhowmick et al. (2020)
β_{2M}	Transmission probability from non-corvid bird to mosquito	0.50	0–1	Bhowmick et al. (2020)
β_H	Transmission probability from mosquito to human	0.01	–	Abdelrazec et al. (2014)
β_{BC}	Transmission probability from mosquito to corvid bird	0.5601	–	Laine (2014)
β_{BN}	Transmission probability from mosquito to non-corvid bird	0.4	0.088–0.9	Abdelrazec et al. (2015)
θ_M	Non-diapausing mosquitoes	$f(D_M)$	–	Bhowmick et al. (2023)
τ	Vertical transmission rate	0.5	0–1.0	Nelms et al. (2013)
a	Fraction of larvae maturing into adult female mosquitoes	0.9	0.8–1.0	Assumed
b	Proportion of infected larvae maturing into infected adults	0.1	0.05–0.15	Baqar et al. (1993)
p_1	Probability of transmission via blood transfusion	0.05	0–0.1	Assumed
p_2	Probability of transmission via organ transplant	0.1	0–0.25	Assumed
η_H	Disease incubation rate in human	0.10	–	Abdelrazec et al. (2014)
η_M	Disease incubation rate in mosquito	0.0469	Varies	Laine (2014)
η_B^C	Disease incubation rate in corvid bird	0.33	–	Laine (2014)
η_B^N	Disease incubation rate in non-corvid bird	0.25	–	Laine (2014)
κ	Fraction of asymptomatic infected individuals	0.75	–	West & Chellamuthu (2020); CDC (2019)

Continued on next page

Table 2 continued from previous page

Parameter	Description	Baseline	Range	Source
ε	Rate of becoming symptomatic from asymptomatic state	0.1	0.05–0.15	Assumed
δ_H	Disease-induced mortality for human	0.015	–	Abdelrazec et al. (2015)
δ_B^C	Disease-induced mortality for corvids	0.50	0–0.5	Moschini et al. (2017)
δ_B^N	Disease-induced mortality for non-corvids	0.3	0–0.5	Moschini et al. (2017); Abdelrazec et al. (2015)
ε_1	Corvid bird viral shedding coefficient	1	–	Calculated from CDC (2003)
ε_2	Non-corvid bird viral shedding coefficient	0.63	–	Calculated from CDC (2003)
ξ_1	Recovery rate coefficient, non-neuroinvasive case	0.225	–	Assumed
ξ_2	Recovery rate coefficient, neuroinvasive case	0.0675	–	Assumed
v_1	Saturation (treatment delay) parameter, non-neuroinvasive	0.25	–	Assumed
v_2	Saturation (treatment delay) parameter, neuroinvasive	0.081	–	Assumed
γ_H	Natural recovery rate for asymptomatic human	0.00002	–	Assumed
γ_{NH}^N	Natural recovery rate, non-neuroinvasive case	0.0714	–	West & Chellamuthu (2020)
$\gamma_H^N(\varsigma)$	Recovery rate due to supportive treatment, non-neuroinvasive	0.5	Variable	Calculated
$\gamma_H^I(\varsigma)$	Recovery rate due to supportive treatment, neuroinvasive	0.1	Variable	Calculated
γ_B^C	Corvid bird recovery rate	0.05	0–0.01	Abdelrazec et al. (2014)
γ_B^N	Non-corvid bird recovery rate	0.1	0–0.2	Abdelrazec et al. (2014)
σ_H	Rate of developing neuroinvasive disease	0.004	–	Assumed
r	Fraction of neuroinvasive cases recovering due to treatment	0.80	–	Assumed
ν	Mosquito egg hatching rate	$2.2 \times 10^{-3} T^{1.77}$	Varies	Marini et al. (2020)
m_L	Larval maturation rate	0.343	Varies	Okuneye & Gumel (2016)
β	Mosquito biting rate	0.122	Varies	Mordecai et al. (2019)
φ_F	Mosquito feeding index	5	–	Bhowmick et al. (2023)
h	Waning immunity rate in human	0.1	–	Assumed
ζ	Recovery to susceptible	0.002	–	Assumed
p	Fraction of corvids recruited into susceptible class	0.20	–	Assumed
q_i	Fraction of recovered birds losing immunity	Varies	–	West & Chellamuthu (2020)
ρ_B^C	Immunity loss rate for corvids	0.07	–	West & Chellamuthu (2020)
ρ_B^N	Immunity loss rate for non-corvids	0.03	–	West & Chellamuthu (2020)
a_{H1}	Proportion of humans undergoing blood transfusion	0.00004	–	Estimated from WHO (2026)
a_{H2}	Proportion of humans undergoing organ transplant	0.00216	0.00185–0.00247	Estimated from WHO (2024)
d_1	Proportion of predator corvid birds	0.85	–	Assumed
d_2	Proportion of predator non-corvid birds	0.35	–	Assumed
ϑ_i	Bird predation rate	1×10^{-4}	0–0.5	Assumed
α_B	Ratio of mosquito to bird	20	10–30	Bhowmick et al. (2020)
α_H	Ratio of mosquito to human	0.3	–	Ruktanonchai et al. (2016)
π_{ij}	Bird food and water consumption rate	0.52	0.04–1	Calculated from Cornell-Lab (n.d.)

Continued on next page

Table 2 continued from previous page

Parameter	Description	Baseline	Range	Source
K_L	Environmental carrying capacity for larvae	1.2175×10^8	Varies	Calibrated from $\langle S_M^{F0} \rangle = N_M^0$
K_{sat}	Half-saturation constant for predation-mediated transmission	$0.05N_B^0$	$(0.01-0.50)N_B^0$	Assumed, saturating form
N_H	Total human population	3.0636×10^7	–	Estimated from National Bureau of Statistics (2026)

2.2 Model equations

The autonomous model system is given by the following first order ODE:

$$\left. \begin{aligned}
 \frac{dS_B^C}{dt} &= p\Lambda_B + q_1\rho_B^C R_B^C - (\bar{\lambda}_B^C + \lambda_B^C + \mu_B)S_B^C \\
 \frac{dE_B^C}{dt} &= (\bar{\lambda}_B^C + \lambda_B^C)S_B^C - (\eta_B^C + \mu_B)E_B^C \\
 \frac{dI_B^C}{dt} &= \eta_B^C E_B^C - (\gamma_B^C + \delta_B^C + \mu_B)I_B^C \\
 \frac{dR_B^C}{dt} &= \gamma_B^C I_B^C - (q_1\rho_B^C + \mu_B)R_B^C \\
 \frac{dS_B^N}{dt} &= (1-p)\Lambda_B + q_2\rho_B^N R_B^N - (\bar{\lambda}_B^N + \lambda_B^N + \mu_B)S_B^N \\
 \frac{dE_B^N}{dt} &= (\bar{\lambda}_B^N + \lambda_B^N)S_B^N - (\eta_B^N + \mu_B)E_B^N \\
 \frac{dI_B^N}{dt} &= \eta_B^N E_B^N - (\gamma_B^N + \delta_B^N + \mu_B)I_B^N \\
 \frac{dR_B^N}{dt} &= \gamma_B^N I_B^N - (q_2\rho_B^N + \mu_B)R_B^N \\
 \frac{dS_E}{dt} &= \varpi\Phi[S_M^F + E_M^F + (1-\tau)I_M^F] - (\nu + \mu_E)S_E \\
 \frac{dI_E}{dt} &= \varpi\Phi\tau I_M^F - (\nu + \mu_E)I_E \\
 \frac{dS_L}{dt} &= \nu S_E - m_L S_L - \mu_L S_L \\
 \frac{dI_L}{dt} &= \nu I_E - (1-b)m_L I_L - bm_L I_L - \mu_L I_L \\
 \frac{dS_M^F}{dt} &= am_L S_L + (1-b)am_L I_L - (\bar{\lambda}_M^F + \mu_M)S_M^F \\
 \frac{dE_M^F}{dt} &= \bar{\lambda}_M^F S_M^F - (\eta_M + \mu_M)E_M^F \\
 \frac{dI_M^F}{dt} &= abm_L I_L + \eta_M E_M^F - \mu_M I_M^F \\
 \frac{dS_H}{dt} &= \Lambda_H + h\zeta R_H - (\bar{\lambda}_H + \lambda_H + \mu_H)S_H \\
 \frac{dE_H}{dt} &= (\bar{\lambda}_H + \lambda_H)S_H - (\kappa\eta_H + (1-\kappa)\eta_H + \mu_H)E_H \\
 \frac{dI_H^A}{dt} &= \kappa\eta_H E_H - (\varepsilon + \gamma_H + \mu_H)I_H^A \\
 \frac{dI_H^N}{dt} &= (1-\kappa)\eta_H E_H + \varepsilon I_H^A - (\sigma_H + \gamma_{NH}^N + \gamma_H^N(\varsigma) + \mu_H)I_H^N \\
 \frac{dI_H^I}{dt} &= \sigma_H I_H^N - (r\gamma_H^I(\varsigma) + \delta_H + \mu_H)I_H^I \\
 \frac{dR_H}{dt} &= \gamma_H I_H^A + (\gamma_{NH}^N + \gamma_H^N(\varsigma))I_H^N + r\gamma_H^I(\varsigma)I_H^I - (h\zeta + \mu_H)R_H \\
 \frac{dD}{dt} &= (\varepsilon_1 + \delta_B^C + \mu_B)I_B^C + (\varepsilon_2 + \delta_B^N + \mu_B)I_B^N - \mu_D D
 \end{aligned} \right\} \quad (11)$$

where $\bar{\lambda}_B^C = \lambda_B^C(T)$, $\bar{\lambda}_B^N = \lambda_B^N(T)$, $\bar{\lambda}_H = \lambda_H(T)$, $\varpi(T) = \varpi$, $m_L(T) = m_L$, $\nu(T) = \nu$, $\eta_M = \eta_M(T)$, $\mu_M = \mu_M(T)$ and $\psi_i(t) = 0$, subject to the initial conditions

$$\left. \begin{aligned} S_B^C(0) > 0, E_B^C(0) \geq 0, I_B^C(0) \geq 0, R_B^C(0) \geq 0, S_B^N(0) > 0, E_B^N(0) \geq 0, I_B^N(0) \geq 0, R_B^N(0) \geq 0, \\ S_E(0) > 0, I_E(0) \geq 0, S_L(0) > 0, I_L(0) \geq 0, S_M^F(0) > 0, E_M^F(0) \geq 0, I_M^F(0) \geq 0, \\ S_H(0) > 0, E_H(0) \geq 0, I_H^A(0) \geq 0, I_H^N(0) \geq 0, I_H^I(0) \geq 0, R_H(0) \geq 0, D(0) \geq 0. \end{aligned} \right\} \quad (12)$$

3 Model analysis

This section presents the basic properties of the model and their analysis: positivity and boundedness of solutions; the disease-free and endemic states; the basic reproduction number; and the local and global stability of the two equilibria.

3.1 Positivity of solutions

Since the model describes real populations, all compartments must remain non-negative.

Theorem 1. *Let the initial solution sets*

$$\begin{aligned} \{S_B^C > 0, E_B^C \geq 0, I_B^C \geq 0, R_B^C \geq 0, S_B^N > 0, E_B^N \geq 0, I_B^N \geq 0, R_B^N \geq 0\} &\in \mathbb{R}_+^8, \\ \{S_E > 0, I_E \geq 0, S_L > 0, I_L \geq 0, S_M^F > 0, E_M^F \geq 0, I_M^F \geq 0\} &\in \mathbb{R}_+^7, \\ \{S_H > 0, E_H \geq 0, I_H^A \geq 0, I_H^N \geq 0, I_H^I \geq 0, R_H \geq 0, D \geq 0\} &\in \mathbb{R}_+^7. \end{aligned} \quad (13)$$

Then the solution set $\{S_B^C(t), E_B^C(t), \dots, R_H(t), D(t)\}$ of the model (11) is positive for all $t \geq 0$.

Proof. From the first equation of (11),

$$\frac{dS_B^C}{dt} = p\Lambda_B + q_1\rho_B^C R_B^C - (\bar{\lambda}_B^C + \lambda_B^C + \mu_B)S_B^C \geq -(\bar{\lambda}_B^C + \lambda_B^C + \mu_B)S_B^C. \quad (14)$$

Separating the variables and integrating gives

$$\int \frac{dS_B^C}{S_B^C} \geq \int -(\bar{\lambda}_B^C + \lambda_B^C + \mu_B) dt, \quad (15)$$

$$\ln S_B^C(t) \geq \int -(\bar{\lambda}_B^C + \lambda_B^C + \mu_B) dt, \quad (16)$$

$$S_B^C(t) \geq e^{\int -(\bar{\lambda}_B^C + \lambda_B^C + \mu_B) dt} \cdot e^C. \quad (17)$$

Setting $C_1 = e^C$ yields $S_B^C(t) \geq C_1 e^{\int -(\bar{\lambda}_B^C + \lambda_B^C + \mu_B) dt}$, and at $t = 0$ we obtain $S_B^C(0) \geq C_1$, so that

$$S_B^C(t) \geq S_B^C(0) e^{\int -(\bar{\lambda}_B^C + \lambda_B^C + \mu_B) dt} > 0. \quad (18)$$

Applying the same argument to each of the compartments proves that

$$E_B^C(t), I_B^C(t), R_B^C(t), S_B^N(t), E_B^N(t), I_B^N(t), R_B^N(t), S_E(t), I_E(t), S_L(t), I_L(t), \quad (19)$$

$$S_M^F(t), E_M^F(t), I_M^F(t), S_H(t), E_H(t), I_H^A(t), I_H^N(t), I_H^I(t), R_H(t) \text{ and } D(t) > 0 \quad (20)$$

remains non-negative for all time $t > 0$.

3.2 Invariant region

The boundedness of the model (11) is necessary if the dynamics of WNV are to be studied in a biologically feasible region.

Theorem 2. *The region*

$$\Omega = \Omega_B \times \Omega_M \times \Omega_H \times \Omega_D = \left\{ \begin{array}{l} (S_B^C, E_B^C, I_B^C, R_B^C, S_B^N, E_B^N, I_B^N, R_B^N, S_E, I_E, S_L, I_L, S_M^F, E_M^F, I_M^F, \\ S_H, E_H, I_H^A, I_H^N, I_H^I, R_H, D) \in \mathbb{R}_+^{22} : N_B(t) \leq \frac{\Lambda_B}{\mu_B}; \\ N_M(t) \leq N_E^{max} + K_L + N_M^{Fmax}; N_H(t) \leq \frac{\Lambda_H}{\mu_H}; D(t) \leq \frac{M}{\mu_D} \end{array} \right\} \quad (21)$$

is invariant and attracting with respect to the system (11).

Proof. Let $N_B(t) = N_B^C + N_B^N$, so that

$$\frac{dN_B}{dt} = \left(\frac{dS_B^C}{dt} + \frac{dE_B^C}{dt} + \frac{dI_B^C}{dt} + \frac{dR_B^C}{dt} \right) + \left(\frac{dS_B^N}{dt} + \frac{dE_B^N}{dt} + \frac{dI_B^N}{dt} + \frac{dR_B^N}{dt} \right). \quad (22)$$

Summing the corresponding equations of (11) and simplifying gives

$$\frac{dN_B}{dt} = \Lambda_B - \mu_B N_B - (\delta_B^C I_B^C + \delta_B^N I_B^N). \quad (23)$$

Since $\delta_B^C + \delta_B^N > 0$, the comparison theorem yields

$$\frac{dN_B}{dt} \leq \Lambda_B - \mu_B N_B, \quad (24)$$

Using separation of variables on (24) and integrating, we get

$$N_B(t) \leq \frac{\Lambda_B}{\mu_B} + \left(N_B(0) - \frac{\Lambda_B}{\mu_B} \right) e^{-\mu_B t}, \quad (25)$$

Therefore, as $t \rightarrow \infty$, $N_B(t) \leq \Lambda_B/\mu_B$.

A similar approach taken for the rest of the sub-populations yields

$$\limsup_{t \rightarrow \infty} N_M(t) \leq K_L + \frac{am_L K_L}{\mu_M} + \frac{\varpi am_L K_L}{\mu_M(\nu + \mu_E)}, \quad (26)$$

where

$$\limsup_{t \rightarrow \infty} N_M^F(t) \leq \frac{am_L K_L}{\mu_M}, \quad \limsup_{t \rightarrow \infty} N_E(t) \leq \frac{\varpi am_L K_L}{\mu_M(\nu + \mu_E)}. \quad (27)$$

$$N_H(t) \leq \frac{\Lambda_H}{\mu_H} + \left(N_H(0) - \frac{\Lambda_H}{\mu_H} \right) e^{-\mu_H t} \rightarrow N_H(t) \leq \frac{\Lambda_H}{\mu_H} \text{ as } t \rightarrow \infty \quad (28)$$

$$D(t) \leq \frac{M}{\mu_D} + D(0)e^{-\mu_D t} - \frac{M}{\mu_D} e^{-\mu_D t} \rightarrow 0 < D(t) \leq \frac{M}{\mu_D} \text{ as } t \rightarrow \infty. \quad (29)$$

Hence Ω is positively invariant and globally attracting, and the model (11) is mathematically well posed and biologically meaningful in Ω .

3.3 Disease-free equilibrium

The model (11) has two disease-free equilibria, namely a trivial disease-free equilibrium and a non-trivial one. Both equilibria are determined by the mosquito demographic reproduction ratio $\mathcal{R}_M$, where

$$\mathcal{R}_M = \frac{\nu \varpi am_L}{(am_L + \mu_L)(\nu + \mu_E)\mu_M} \quad (30)$$

so that:

(i) If $\mathcal{R}_M \leq 1$, then equation (11) has a trivial disease-free equilibrium (E_T^0) given by

$$\begin{aligned} E_{AT}^0 &= (S_B^{C0}, E_B^{C0}, I_B^{C0}, R_B^{C0}, S_B^{N0}, E_B^{N0}, I_B^{N0}, R_B^{N0}, S_E^0, I_E^0, S_L^0, I_L^0, S_M^{F0}, E_M^{F0}, I_M^{F0}, S_H^0, E_H^0, I_H^{A0}, I_H^{N0}, I_H^{I0}, R_H^0, D^0) \\ &= \left(\frac{p\Lambda_B}{\mu_B}, 0, 0, 0, \frac{(1-p)\Lambda_B}{\mu_B}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, \frac{\Lambda_H}{\mu_H}, 0, 0, 0, 0, 0, 0 \right), \end{aligned} \quad (31)$$

- (ii) If $\mathcal{R}_M > 1$, the equation (11) has a non-trivial or mosquito-presence disease-free equilibrium (E_N^0) given by

$$\begin{aligned} E_{N,0} = & (S_{B,0}^C, E_{B,0}^C, I_{B,0}^C, R_{B,0}^C, S_{B,0}^N, E_{B,0}^N, I_{B,0}^N, R_{B,0}^N, S_{E,0}, I_{E,0}, S_{L,0}, I_{L,0}, S_{M,0}^F, E_{M,0}^F, I_{M,0}^F, \\ & S_{H,0}, E_{H,0}, I_{H,0}^A, I_{H,0}^N, I_{H,0}^I, R_{H,0}, D_0) \\ = & \left(\frac{p\Lambda_B}{\mu_B}, 0, 0, 0, \frac{(1-p)\Lambda_B}{\mu_B}, 0, 0, 0, S_{E,0}, 0, S_{L,0}, 0, S_{M,0}^F, 0, 0, \frac{\Lambda_H}{\mu_H}, 0, 0, 0, 0, 0, 0 \right), \end{aligned} \quad (32)$$

where

$$S_E^0 = \frac{\varpi \Phi S_M^{F0}}{\nu + \mu_E}, \quad S_L^0 = \frac{\nu S_E^0}{m_L + \mu_L}, \quad S_M^{F0} = \frac{am_L S_L^0}{\mu_M}. \quad (33)$$

3.4 Endemic equilibrium point

For the West Nile virus presence equilibrium, we let $(E_B^C, E_B^N, I_B^C, I_B^N, R_B^C, R_B^N, I_E, I_L, E_M^F, I_M^F, E_H, I_H^A, I_H^N, I_H^I, R_H, D) \neq 0$, and system (11) is solved at the equilibrium state. Writing $\lambda_1^* = \bar{\lambda}_B^{C*} + \lambda_B^{C*}$ and $\lambda_2^* = \bar{\lambda}_B^{N*} + \lambda_B^{N*}$ for the total forces of infection acting on the corvid and non-corvid susceptible classes, solving system (11) at equilibrium

gives:

$$\left. \begin{aligned}
 S_B^{C*} &= \frac{\gamma_B^C \eta_B^C \lambda_1^* p \Lambda_B (p \Lambda_B + q_1 \rho_B^C)}{(\lambda_1^* + \mu_B) [(\eta_B^C + \mu_B)(q_1 \rho_B^C + \mu_B)(\gamma_B^C + \delta_B^C + \mu_B)(\lambda_1^* + \mu_B) - \gamma_B^C \eta_B^C \lambda_1^* q_1 \rho_B^C]} \\
 S_B^{N*} &= \frac{\gamma_B^N \eta_B^N \lambda_2^* (1-p) \Lambda_B [(1-p) \Lambda_B + q_2 \rho_B^N]}{(\lambda_2^* + \mu_B) [(\eta_B^N + \mu_B)(q_2 \rho_B^N + \mu_B)(\gamma_B^N + \delta_B^N + \mu_B)(\lambda_2^* + \mu_B) - \gamma_B^N \eta_B^N \lambda_2^* q_2 \rho_B^N]} \\
 E_B^{C*} &= \frac{p \Lambda_B \eta_B^C \gamma_B^C (p \Lambda_B + q_1 \rho_B^C)(\lambda_1^*)^2}{(\eta_B^C + \mu_B)(\lambda_1^* + \mu_B) [(\eta_B^C + \mu_B)(q_1 \rho_B^C + \mu_B)(\gamma_B^C + \delta_B^C + \mu_B)(\lambda_1^* + \mu_B) - \gamma_B^C q_1 \rho_B^C \lambda_1^*]} \\
 E_B^{N*} &= \frac{(1-p) \Lambda_B \eta_B^N \gamma_B^N [(1-p) \Lambda_B + q_2 \rho_B^N](\lambda_2^*)^2}{(\eta_B^N + \mu_B)(\lambda_2^* + \mu_B) [(\eta_B^N + \mu_B)(q_2 \rho_B^N + \mu_B)(\gamma_B^N + \delta_B^N + \mu_B)(\lambda_2^* + \mu_B) - \gamma_B^N q_2 \rho_B^N \lambda_2^*]} \\
 I_B^{C*} &= \frac{\gamma_B^C (\eta_B^C)^2 p b_B N_B^* (p b_B N_B^* + q_1 \rho_B^C)(\lambda_1^*)^2}{(\eta_B^C + \mu_B)(\lambda_1^* + \mu_B)(\gamma_B^C + \delta_B^C + \mu_B) [(\eta_B^C + \mu_B)(q_1 \rho_B^C + \mu_B)(\gamma_B^C + \delta_B^C + \mu_B)(\lambda_1^* + \mu_B) - q_1 \rho_B^C \gamma_B^C \lambda_1^*]} \\
 I_B^{N*} &= \frac{\gamma_B^N (\eta_B^N)^2 (1-p) b_B N_B^* [(1-p) \Lambda_B + q_2 \rho_B^N](\lambda_2^*)^2}{(\eta_B^N + \mu_B)(\lambda_2^* + \mu_B)(\gamma_B^N + \delta_B^N + \mu_B) [(\eta_B^N + \mu_B)(q_2 \rho_B^N + \mu_B)(\gamma_B^N + \delta_B^N + \mu_B)(\lambda_2^* + \mu_B) - q_2 \rho_B^N \gamma_B^N \lambda_2^*]} \\
 R_B^{C*} &= \frac{\gamma_B^C \eta_B^C \lambda_1^* p \Lambda_B q_1 \rho_B^C}{(\eta_B^C + \mu_B)(q_1 \rho_B^C + \mu_B)(\gamma_B^C + \delta_B^C + \mu_B)(\lambda_1^* + \mu_B) - q_1 \rho_B^C \gamma_B^C \eta_B^C \lambda_1^*} \\
 R_B^{N*} &= \frac{\gamma_B^N \eta_B^N \lambda_2^* (1-p) \Lambda_B q_2 \rho_B^N}{(\eta_B^N + \mu_B)(q_2 \rho_B^N + \mu_B)(\gamma_B^N + \delta_B^N + \mu_B)(\lambda_2^* + \mu_B) - q_2 \rho_B^N \gamma_B^N \eta_B^N \lambda_2^*} \\
 S_E^* &= \frac{\varpi \Phi^* [S_M^{F*} + E_M^{F*} + (1-\tau) I_M^{F*}]}{\nu + \mu_E} \\
 I_E^* &= \frac{\varpi \Phi^* \tau I_M^{F*}}{\nu + \mu_E} \\
 S_L^* &= \frac{\nu S_E^*}{am_L - \mu_L} \\
 I_L^* &= \frac{\nu I_E^* - bm_L}{(1-b)m_L + \mu_L} \\
 S_M^{F*} &= \frac{m_L [a S_L^* + (1-b) I_L^*]}{\bar{\lambda}_M^{F*} + \mu_M} \\
 E_M^{F*} &= \frac{\bar{\lambda}_M^{F*} S_M^{F*}}{\eta_M + \mu_M} \\
 I_M^{F*} &= \frac{1}{\mu_M} (bm_L I_L^* + \eta_M E_M^{F*}) \\
 S_H^* &= \frac{\Lambda + h \zeta R_H^*}{\bar{\lambda}_H^* + \lambda_H^* + \mu_H} \\
 E_H^* &= \frac{(\bar{\lambda}_H^* + \lambda_H^*) S_H^*}{\kappa \eta_H + (1-\kappa) \eta_H + \mu_H} \\
 I_H^{A*} &= \frac{\kappa \eta_H E_H^*}{\varepsilon + \gamma_H + \mu_H} \\
 I_H^{N*} &= \frac{(1-\kappa) \eta_H E_H^* + \varepsilon I_H^{A*}}{\sigma_H + \gamma_{NH}^N + \gamma_H^N(\varsigma) + \mu_H} \\
 I_H^{I*} &= \frac{\sigma_H I_H^{N*}}{r \gamma_H^I(\varsigma) + \delta_H + \mu_H} \\
 R_H^* &= \frac{\gamma_H I_H^{A*} + (\gamma_{NH}^N + \gamma_H^N(\varsigma)) I_H^{N*} + r \gamma_H^I(\varsigma) I_H^{I*}}{h \zeta + \mu_H} \\
 D^* &= \frac{1}{\mu_B} [(\varepsilon_1 + \delta_B^C + \mu_B) I_B^{C*} + (\varepsilon_2 + \delta_B^N + \mu_B) I_B^{N*}]
 \end{aligned} \right\} \quad (34)$$

3.5 Basic reproduction number

The next generation matrix method of Van den Driessche and Watmough (2002) is applied to determine $R_0 = \rho(FV^{-1})$, by setting $\dot{X}(t) = F(X) - V(X)$, where $F(X)$ denotes the vector of new infection terms and $V(X)$ the vector of transfer terms, and by evaluating $F = [\partial F_i(X^0)/\partial X_j]$ and $V = [\partial V_i(X^0)/\partial X_j]$ at the disease-free equilibrium (32). Taking the infected state vector as

$$X = (E_B^C, I_B^C, E_B^N, I_B^N, I_E, I_L, E_M^F, I_M^F, E_H, I_H^A, I_H^N, I_H^I, D)^T, \quad (35)$$

we have, linearising and evaluating at the disease-free equilibrium,

$$F = \frac{\partial F_i}{\partial X_j} = \begin{pmatrix} 0 & a_1 S_B^{C0} & 0 & a_2 S_B^{C0} & 0 & 0 & 0 & a_3 S_B^{C0} & 0 & 0 & 0 & 0 & a_4 S_B^{C0} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & a_1 S_B^{N0} & 0 & a_2 S_B^{N0} & 0 & 0 & 0 & a_5 S_B^{N0} & 0 & 0 & 0 & 0 & a_6 S_B^{N0} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_7 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & a_8 S_M^{F0} & 0 & a_9 S_M^{F0} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{10} S_H^0 & 0 & a_{11} S_H^0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (36)$$

where

$$\left. \begin{aligned} a_1 &= d_1 \beta_1 \vartheta_1, & a_2 &= d_2 \beta_2 \vartheta_2, & a_3 &= \frac{\beta_1 \beta \theta_M \varphi_F}{\varphi_F N_B^0 + N_H^0}, & a_4 &= \frac{\beta_1 \pi_{1j}}{N_B^0}, \\ a_5 &= \frac{\beta_2 \beta \theta_M \varphi_F}{\varphi_F N_B^0 + N_H^0}, & a_6 &= \frac{\beta_2 \pi_{2j}}{N_B^0}, & a_7 &= \varpi \Phi^* \tau, & (\Phi^* &= 1/\mathcal{R}_M), & a_8 &= \frac{\beta_{1M} \beta \theta_M \varphi_F}{\varphi_F N_B^0 + N_H^0}, \\ a_9 &= \frac{\beta_{2M} \beta \theta_M \varphi_F}{\varphi_F N_B^0 + N_H^0}, & a_{10} &= \frac{\beta_H \beta \theta_M}{\varphi_F N_B^0 + N_H^0}, & a_{11} S_H^0 &= p_1 a_{H1} + p_2 a_{H2}, \end{aligned} \right\} \quad (37)$$

and

$$V = \frac{\partial V_i}{\partial X_j} = \begin{pmatrix} b_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ b_{14} & b_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & b_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & b_{15} & b_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & b_5 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & b_{16} & b_6 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & b_7 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & b_{17} & b_{18} & b_8 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & b_9 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & b_{19} & b_{10} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & b_{20} & b_{21} & b_{11} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & b_{22} & b_{12} & 0 \\ 0 & b_{23} & 0 & b_{24} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & b_{13} \end{pmatrix} \quad (38)$$

with

$$\begin{aligned} b_1 &= \eta_B^C + \mu_B, & b_2 &= \gamma_B^C + \delta_B^C + \mu_B, & b_3 &= \eta_B^N + \mu_B, & b_4 &= \gamma_B^N + \delta_B^N + \mu_B, & b_5 &= \nu + \mu_E, \\ b_6 &= m_L + \mu_L, & b_7 &= \eta_M + \mu_M, & b_8 &= \mu_M, & b_9 &= \eta_H + \mu_H, & b_{10} &= \varepsilon + \gamma_H + \mu_H, \\ b_{11} &= \sigma_H + \gamma_{NH}^N + \mu_H, & b_{12} &= \delta_H + \mu_H, & b_{13} &= \mu_D, & b_{14} &= -\eta_B^C, & b_{15} &= -\eta_B^N, & b_{16} &= -\nu, \\ b_{17} &= -abm_L, & b_{18} &= -\eta_M, & b_{19} &= -\kappa\eta_H, & b_{20} &= -(1-\kappa)\eta_H, & b_{21} &= -\varepsilon, & b_{22} &= -\sigma_H, \\ b_{23} &= -(\varepsilon_1 + \delta_B^C + \mu_B), & b_{24} &= -(\varepsilon_2 + \delta_B^N + \mu_B). \end{aligned} \quad (39)$$

Inverting V term by term gives the auxiliary quantities

$$\begin{aligned} B_{14} &= -\frac{b_{14}}{b_1 b_2}, & B_{15} &= -\frac{b_{15}}{b_3 b_4}, & B_{16} &= -\frac{b_{16}}{b_5 b_6}, & B_{17} &= \frac{b_{16} b_{17}}{b_5 b_6 b_8}, & B_{18} &= -\frac{b_{17}}{b_6 b_8}, & B_{19} &= -\frac{b_{18}}{b_7 b_8}, \\ B_{20} &= -\frac{b_{19}}{b_9 b_{10}}, & B_{21} &= -\frac{b_{10} b_{20} - b_{19} b_{21}}{b_9 b_{10} b_{11}}, & B_{22} &= -\frac{b_{21}}{b_{10} b_{11}}, & B_{23} &= \frac{b_{10} b_{20} b_{22} - b_{19} b_{21} b_{22}}{b_9 b_{10} b_{11} b_{12}}, \\ B_{24} &= \frac{b_{21} b_{22}}{b_{10} b_{11} b_{12}}, & B_{25} &= -\frac{b_{22}}{b_{11} b_{12}}, & B_{26} &= \frac{b_{14} b_{23}}{b_1 b_2 b_{13}}, & B_{27} &= -\frac{b_{23}}{b_2 b_{13}}, & B_{28} &= \frac{b_{15} b_{24}}{b_3 b_4 b_{13}}, & B_{29} &= -\frac{b_{24}}{b_4 b_{13}}. \end{aligned} \quad (40)$$

Forming the product and writing $A_1 = a_1 S_B^{C0}$, $A_2 = a_2 S_B^{C0}$, $A_3 = a_3 S_B^{C0}$, $A_4 = a_4 S_B^{C0}$, $A_5 = a_5 S_B^{N0}$, $A_6 = a_6 S_B^{N0}$, $A_7 = a_7$, $A_8 = a_8 S_M^{F0}$, $A_9 = a_9 S_M^{F0}$, $A_{10} = a_{10} S_H^0$, $A_{11} = a_{11} S_H^0$, $A_{12} = a_1 S_B^{N0}$ and $A_{13} = a_2 S_B^{N0}$, the next generation matrix becomes

$$FV^{-1} = \begin{pmatrix} \phi_1 & \phi_{26} & \phi_2 & \phi_3 & \phi_{32} & \phi_{19} & \phi_4 & \phi_5 & 0 & 0 & 0 & 0 & \phi_6 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \phi_{27} & \phi_{28} & \phi_{29} & \phi_{30} & \phi_7 & \phi_{20} & \phi_{21} & \phi_8 & 0 & 0 & 0 & 0 & \phi_9 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \phi_{10} & \phi_{22} & \phi_{23} & \phi_{11} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \phi_{12} & \phi_{31} & \phi_{13} & \phi_{14} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \phi_{15} & \phi_{24} & \phi_{25} & \phi_{16} & \phi_{17} & \phi_{18} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (41)$$

where

$$\begin{aligned}
 \phi_1 &= A_1 B_{14} + A_4 B_{26}, & \phi_2 &= A_2 B_{15} + A_4 B_{28}, & \phi_3 &= A_2 B_4 + A_4 B_{29}, & \phi_4 &= A_3 B_{19}, & \phi_5 &= A_3 B_8, \\
 \phi_6 &= A_4 B_{13}, & \phi_7 &= A_5 B_{17}, & \phi_8 &= A_5 B_8, & \phi_9 &= A_6 B_{13}, & \phi_{10} &= A_7 B_{17}, & \phi_{11} &= A_7 B_8, & \phi_{12} &= A_8 B_{14}, \\
 \phi_{13} &= A_9 B_{15}, & \phi_{14} &= B_4 A_9, & \phi_{15} &= A_{10} B_{17}, & \phi_{16} &= B_8 A_{10}, & \phi_{17} &= A_{11} B_{20}, & \phi_{18} &= A_{11} B_{10}, & \phi_{19} &= A_3 B_{18}, \\
 \phi_{20} &= A_5 B_{18}, & \phi_{21} &= A_5 B_{19}, & \phi_{22} &= A_7 B_{18}, & \phi_{23} &= A_7 B_{19}, & \phi_{24} &= A_{10} B_{18}, & \phi_{25} &= A_{10} B_{19}, \\
 \phi_{26} &= A_1 B_2 + A_4 B_{27}, & \phi_{27} &= A_{12} B_{14} + A_6 B_{26}, & \phi_{28} &= A_{12} B_2 + A_6 B_{27}, & \phi_{29} &= A_{13} B_{15} + A_6 B_{28}, \\
 \phi_{30} &= A_{13} B_4 + A_6 B_{29}, & \phi_{31} &= A_8 B_2, & \phi_{32} &= A_3 B_{17}.
 \end{aligned} \tag{42}$$

Because rows 2, 4, 6, 8, 10, 11, 12 and 13 of (41) vanish identically, the corresponding columns cancel in the evaluation of the cofactors, and

$$\det(FV^{-1} - \lambda I) = (-\lambda)^8 \det(Z_5 - \lambda I_5), \tag{43}$$

where

$$|Z_{5 \times 5} - \lambda I_5| = \begin{vmatrix} \phi_1 - \lambda & \phi_2 & \phi_{32} & \phi_4 & 0 \\ \phi_{27} & \phi_{29} - \lambda & \phi_7 & \phi_{21} & 0 \\ 0 & 0 & \phi_{10} - \lambda & \phi_{23} & 0 \\ \phi_{12} & \phi_{13} & 0 & 0 - \lambda & 0 \\ 0 & 0 & \phi_{15} & \phi_{25} & \phi_{17} - \lambda \end{vmatrix} = 0. \tag{44}$$

The fifth column has a single non-zero entry, so that expanding along it gives

$$\det(Z_5 - \lambda I_5) = (\phi_{17} - \lambda) \det(M_4 - \lambda I_4), \tag{45}$$

whence

$$\lambda = \phi_{17} = A_{11} B_{20} = R_{0,H}, \tag{46}$$

which shows that the human component is structurally decoupled from the vector-avian cycle. With (41) now reduced to $Z_{4 \times 4}$,

$$|Z_4 - \lambda I_4| = \begin{vmatrix} \phi_1 - \lambda & \phi_2 & \phi_{32} & \phi_4 \\ \phi_{27} & \phi_{29} - \lambda & \phi_7 & \phi_{21} \\ 0 & 0 & \phi_{10} - \lambda & \phi_{23} \\ \phi_{12} & \phi_{13} & 0 & -\lambda \end{vmatrix} = 0, \tag{47}$$

from which the characteristic polynomial

$$\det(Z_4 - \lambda I) = \lambda^4 - w_1 \lambda^3 + w_2 \lambda^2 - w_3 \lambda + w_4 = 0 \tag{48}$$

is obtained, w_k being the sum of the $k \times k$ principal minors.

$$\lambda^3 - (T_B + R_V) \lambda^2 + (D_B + R_V T_B - R_C) \lambda - (R_V D_B - E) = 0, \tag{49}$$

where

$$R_V = \phi_{10}, \quad T_B = \phi_1 + \phi_{29}, \quad D_B = \phi_1 \phi_{29} - \phi_2 \phi_{27}, \tag{50}$$

$$R_C = \phi_4 \phi_{12} + \phi_{21} \phi_{13}, \quad E = \phi_4 (\phi_{12} \phi_{29} - \phi_{13} \phi_{27}) + \phi_{21} (\phi_1 \phi_{13} - \phi_2 \phi_{12}). \tag{51}$$

Expanding (49), collecting the terms free of R_V , D_B and E , and factorising,

$$\lambda(\lambda^2 - T_B \lambda - R_C) = R_V(\lambda^2 - T_B \lambda + D_B) - D_B \lambda - E. \tag{52}$$

Setting the right-hand side to zero, since these are small quantities, discards R_V , D_B and E ; D_B and E vanish

for the structural reason given above and, at the baseline parameterisation, $R_V = 0.0405$, leaving the part driven by the vector cycle. Thus

$$\lambda(\lambda^2 - T_B\lambda - R_C) = 0 \implies \lambda^2 - T_B\lambda - R_C = 0, \quad (53)$$

so that dropping D_B and E costs nothing, as we put to numerical verification.

$$R_0 = \frac{(T_B + R_V) + \sqrt{(T_B + R_V)^2 + 4(R_C - R_V T_B)}}{2}. \quad (54)$$

The transmission pathways are accordingly

$$R_0^{(M \leftrightarrow B)} = \sqrt{R_C} \text{ (mosquito-bird cycle),} \quad R_0^{(B \leftrightarrow B)} = T_B \text{ (bird-to-bird transmission),} \quad (55)$$

$$R_0^{(vert.)} = R_V \text{ (vertical transmission),} \quad R_{0,H} = \phi_{17} \text{ (human transmission),} \quad (56)$$

At the baseline parameterisation of Table 2, evaluated at $T = 25^\circ\text{C}$, the basic reproduction number is $R_0 = 1.844546$.

3.6 Local stability of the disease-free equilibrium

Theorem 3. *The disease-free equilibrium of the WNV model (11) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. The Jacobian matrix of system (11) at the West Nile virus free equilibrium is a 22×22 matrix which, after row and column reordering to isolate the corvid, non-corvid, mosquito and human blocks, decouples into four sub-blocks whose characteristic polynomials multiply to give the full characteristic polynomial. Writing

$$\begin{aligned} f_1 &= \mu_B, & f_2 &= \eta_B^C + \mu_B, & f_3 &= \gamma_B^C + \delta_B^C + \mu_B, & f_4 &= q_1 \rho_B^C + \mu_B, & f_5 &= \mu_B, & f_6 &= \eta_B^N + \mu_B, \\ f_7 &= q_2 \rho_B^N + \mu_B, & f_8 &= \nu + \mu_E, & f_9 &= a m_L + \mu_L, & f_{10} &= m_L + \mu_L, & f_{11} &= \mu_M, & f_{12} &= \eta_M + \mu_M, \\ f_{13} &= \mu_H, & f_{14} &= \eta_H + \mu_H, & f_{15} &= \varepsilon + \gamma_H + \mu_H, & f_{16} &= 1 + \mu_H, & f_{17} &= \delta_H + \mu_H, & f_{18} &= h\zeta + \mu_H, \\ f_{19} &= \mu_D, & f_{20} &= d_1 \beta_1 \vartheta_1 S_B^{C0}, & f_{21} &= d_1 \beta_1 \vartheta_1 S_B^{N0}, & f_{22} &= q_1 \rho_B^C, & f_{23} &= d_2 \beta_2 \vartheta_2 S_B^{C0}, \\ f_{24} &= d_2 \beta_2 \vartheta_2 S_B^{N0}, & f_{25} &= \frac{\beta_1 \beta \theta_M \varphi_F}{\varphi_F N_B + N_H} S_B^{C0}, & f_{26} &= q_2 \rho_B^N, & f_{27} &= \frac{\beta_2 \beta \theta_M \varphi_F}{\varphi_F N_B + N_H} S_B^{N0}, \\ f_{28} &= (1 - \tau) \varpi \Phi, & f_{29} &= \varpi \Phi \tau, & f_{30} &= \frac{\beta_1 M \beta \theta_M \varphi_F}{\varphi_F N_B + N_H} S_M^{F0}, & f_{31} &= \frac{\beta_2 M \beta \theta_M \varphi_F}{\varphi_F N_B + N_H} S_M^{F0}, \\ f_{32} &= a m_L, & f_{33} &= (1 - b) m_L, & f_{34} &= b m_L, & f_{35} &= \frac{\beta_H \beta}{\theta_H N_B + N_H} S_H^0, & f_{36} &= \lambda_H = p_1 a_{H1} + p_2 a_{H2}, \\ f_{37} &= h\zeta, & f_{38} &= \kappa \eta_H, & f_{39} &= (1 - \kappa) \eta_H, & f_{40} &= 1, & f_{41} &= \varepsilon_1 + \delta_B^C + \mu_B, & f_{42} &= \varepsilon_2 + \delta_B^N + \mu_B, \end{aligned} \quad (57)$$

the characteristic polynomial of the Jacobian factorises as

$$\begin{aligned} & \left(g_1 \lambda^6 + g_2 \lambda^5 + g_3 \lambda^4 + g_4 \lambda^3 + g_5 \lambda^2 + g_6 \lambda + g_0 \right) \left(h_1 \lambda^6 + h_2 \lambda^5 + h_3 \lambda^4 + h_4 \lambda^3 + h_5 \lambda^2 + h_6 \lambda + h_0 \right) \\ & \times \left(\lambda^5 + m_1 \lambda^4 + m_2 \lambda^3 + m_3 \lambda^2 + m_4 \lambda + m_5 \right) \left(\lambda^5 + n_1 \lambda^4 + n_2 \lambda^3 + n_3 \lambda^2 + n_4 \lambda + n_5 \right) = 0, \end{aligned} \quad (58)$$

with coefficients

$$\left. \begin{aligned} g_1 &= 1, \\ g_2 &= (2f_1 + f_2 + f_3 + f_4 + f_5), \\ g_3 &= f_3(f_1 + f_2) + f_5(2f_1 + f_2 + f_3 + f_4) + f_1f_2 + f_4(f_1 + f_2 + f_3) - \eta_B^C f_{20} + f_1(f_1 + f_2 + f_3 + f_4), \\ g_4 &= f_4[f_3(f_1 + f_2) + f_1f_2 - \eta_B^C f_{20}] + f_1[f_3(f_1 + f_2) + f_1f_2 + f_4(f_1 + f_2 + f_3) - \eta_B^C f_{20}] \\ &\quad + f_5[f_3(f_1 + f_2) + f_1f_2 + f_4(f_1 + f_2 + f_3) - \eta_B^C f_{20} + f_1(f_1 + f_2 + f_3 + f_4)] \\ &\quad - \eta_B^C f_{20}(f_1 + f_2) + \eta_B^C f_2f_{20} + f_1f_2f_3, \\ g_5 &= f_4[\eta_B^C f_2f_{20} - \eta_B^C f_{20}(f_1 + f_2) + f_1f_2f_3] \\ &\quad + f_5\left\{f_4[f_3(f_1 + f_2) + f_1f_2 - \eta_B^C f_{20}] + f_1[f_3(f_1 + f_2) + f_1f_2 + f_4(f_1 + f_2 + f_3) - \eta_B^C f_{20}] \right. \\ &\quad \left. - \eta_B^C f_{20}(f_1 + f_2) + \eta_B^C f_2f_{20} + f_1f_2f_3\right\} \\ &\quad + f_1f_4[f_3(f_1 + f_2) + f_1f_2 - \eta_B^C f_{20}] - \eta_B^C f_{20}(f_1 + f_2) + \eta_B^C f_2f_{20} + f_1f_2f_3, \\ g_6 &= f_5f_4[\eta_B^C f_2f_{20} - \eta_B^C f_{20}(f_1 + f_2) + f_1f_2f_3] + f_1f_4[f_3(f_1 + f_2) + f_1f_2 - \eta_B^C f_{20}] \\ &\quad - \eta_B^C f_{20}(f_1 + f_2) + \eta_B^C f_2f_{20} + f_1f_2f_3 + f_1f_4[\eta_B^C f_2f_{20} - \eta_B^C f_{20}(f_1 + f_2) + f_1f_2f_3], \\ g_0 &= f_1f_4f_5[\eta_B^C f_2f_{20} - \eta_B^C f_{20}(f_1 + f_2) + f_1f_2f_3], \end{aligned} \right\} \quad (59)$$

$$\left. \begin{aligned} h_1 &= 1 \\ h_2 &= (f_6 + f_7 + 2f_8 + f_9 + f_{10}) \\ h_3 &= (f_9(f_6 + f_7 + 2f_8) + f_8(f_6 + f_7) + f_{10}(f_6 + f_7 + 2f_8 + f_9) + f_6f_7 + f_8(f_6 + f_7 + f_8)) \\ h_4 &= (f_{10}[f_9(f_6 + f_7 + 2f_8) + f_8(f_6 + f_7) + f_6f_7 + f_8(f_6 + f_7 + f_8)] + f_8[f_8(f_6 + f_7) + f_6f_7] \\ &\quad + f_9[f_8(f_6 + f_7) + f_6f_7 + f_8(f_6 + f_7 + f_8)] + f_6f_7f_8) \\ h_5 &= (f_9[f_8(f_8(f_6 + f_7) + f_6f_7) + f_6f_7f_8] + f_{10}[f_8(f_8(f_6 + f_7) + f_6f_7) \\ &\quad + f_9(f_8(f_6 + f_7) + f_6f_7 + f_8(f_6 + f_7 + f_8)) + f_6f_7f_8 + f_6f_7f_8^2]) \\ h_6 &= (f_{10}f_9[f_8(f_8(f_6 + f_7) + f_6f_7) + f_6f_7f_8] + f_6f_7f_8^2) + f_6f_7f_8^2f_9 \\ h_0 &= f_6f_7f_8^2f_9f_{10}, \end{aligned} \right\} \quad (60)$$

$$\left. \begin{aligned} m_1 &= 1 \\ m_2 &= (2f_{11} + f_{12} + f_{13} + f_{14}) \\ m_3 &= (f_{14}(2f_{11} + f_{12} + f_{13}) + f_{11}(f_{11} + f_{12}) + f_{11}f_{12} + f_{13}(2f_{11} + f_{12})) \\ m_4 &= (f_{11}^2f_{12} + f_{13}[f_{11}(f_{11} + f_{12}) + f_{11}f_{12}] + f_{14}[f_{11}(f_{11} + f_{12}) + f_{11}f_{12} + f_{13}(2f_{11} + f_{12})]) \\ m_5 &= (f_{14}f_{11}^2f_{12} + f_{13}[f_{11}(f_{11} + f_{12}) + f_{11}f_{12}]) + f_{11}^2f_{12}f_{13} \\ m_0 &= f_{11}^2f_{12}f_{13}f_{14}, \end{aligned} \right\} \quad (61)$$

and

$$\left. \begin{aligned} n_1 &= 1, \quad n_2 = (f_{15} + f_{16} + f_{17} + f_{18} + f_{19}), \\ n_3 &= f_{17}(f_{15} + f_{16}) + f_{15}f_{16} + f_{18}(f_{15} + f_{16} + f_{17}) + f_{19}(f_{15} + f_{16} + f_{17} + f_{18}), \\ n_4 &= f_{18}[f_{17}(f_{15} + f_{16}) + f_{15}f_{16}] + f_{19}[f_{17}(f_{15} + f_{16}) + f_{15}f_{16} + f_{18}(f_{15} + f_{16} + f_{17})] + f_{15}f_{16}f_{17}, \\ n_5 &= f_{19}\{f_{18}[f_{17}(f_{15} + f_{16}) + f_{15}f_{16}] + f_{15}f_{16}f_{17}\} + f_{15}f_{16}f_{17}f_{18}, \quad n_0 = f_{15}f_{16}f_{17}f_{18}f_{19}, \end{aligned} \right\} \quad (62)$$

The Routh–Hurwitz criterion states that all roots of polynomial (58) have negative real parts if and only if the coefficients D_i , with $g_i, h_i, m_i, n_i \in D_i$, are positive and the principal minors $H_i > 0$ for $i = 1, 2, \dots, 22$. Since $\lambda_i < 0$ for all i whenever $R_0 < 1$, the disease-free equilibrium of the model (11) is locally asymptotically stable.

3.7 Global stability of the WNV-free equilibrium

The method of Castillo-Chavez, Feng and Huang (2008) is used to determine the global stability of the disease-free equilibrium, on the basis of the two conditions that guarantee it. The system is rewritten as

$$\frac{dX}{dt} = F(X, Z), \quad \frac{dZ}{dt} = G(X, Z), \quad G(X, 0) = 0, \quad (63)$$

where $X = (S_B^C, R_B^C, S_B^N, R_B^N, S_E, S_L, S_M^F, S_H, R_H) \in \mathbb{R}^9$ denotes the uninfected compartments and $Z = (E_B^C, I_B^C, E_B^N, I_B^N, I_E, I_L, E_M^F, I_M^F, E_H, I_H^A, I_H^N, I_H^I, D) \in \mathbb{R}^{13}$ the infected compartments, and the disease-free state is $E^0 = (X^0, 0)$.

Lemma 4. *The point $K_0 = (X^0, 0)$ is a globally asymptotically stable equilibrium point provided that $R_0 < 1$ and that the following two conditions hold:*

(H1) for $\frac{dX}{dt} = F(X, 0)$, X^0 is globally asymptotically stable;

(H2) $G(X, Z) = CZ - \hat{G}(X, Z)$ with $\hat{G}(X, Z) \geq 0$ for $(X, Z) \in \Omega$, where $C = D_Z G(X^0, 0)$ is an M-matrix and Ω is the biologically feasible region.

Theorem 5. *Let $R_0 < 1$. Then the disease-free equilibrium of the model (11) is globally asymptotically stable in Ω .*

Proof. With $X^0 = (S_B^{C0}, 0, S_B^{N0}, 0, S_E^0, S_L^0, S_M^{F0}, S_H^0, 0)$ as in (32), the uninfected subsystem evaluated at $Z = 0$ is

$$F(X, 0) = \begin{pmatrix} p\Lambda_B - \mu_B S_B^{C0} \\ (1-p)\Lambda_B - \mu_B S_B^{N0} \\ \varpi\Phi S_M^F - (\nu + \mu_E)S_E \\ \nu S_E - (am_L + \mu_L)S_L \\ am_L S_L - \mu_M S_M^F \\ \Lambda_H - \mu_H S_H \\ 0 \end{pmatrix}, \quad (64)$$

which is a linear cascade with strictly negative diagonal, so that $S_B^C \rightarrow S_B^{C0}$, $S_B^N \rightarrow S_B^{N0}$, $S_E \rightarrow S_E^0$, $S_L \rightarrow S_L^0$, $S_M^F \rightarrow S_M^{F0}$ and $S_H \rightarrow S_H^0$ as $t \rightarrow \infty$; hence X^0 is globally asymptotically stable and condition (H1) is satisfied. For the infected subsystem, the linearisation at the disease-free equilibrium gives $C = D_Z G(X^0, 0)$ with entries

$$\begin{aligned} C_1 &= \eta_B^C + \mu_B, & C_2 &= d_1 \beta_1 \vartheta_1, & C_3 &= d_2 \beta_2 \vartheta_2, & C_4 &= \frac{\beta_i \beta \theta_M \varphi_F}{\varphi_F N_B^0 + N_H^0}, & C_5 &= \beta_i \pi_{ij} / W_{ref}, \\ C_6 &= \gamma_B^C + \delta_B^C + \mu_B, & C_7 &= \eta_B^N + \mu_B, & C_8 &= \gamma_B^N + \delta_B^N + \mu_B, & C_9 &= \nu + \mu_E, & C_{10} &= \varpi \Phi \tau, \\ C_{11} &= m_L + \mu_L, & C_{12} &= \frac{\beta_{1M} \beta \theta_M \varphi_F}{\varphi_F N_B + N_H}, & C_{13} &= \frac{\beta_{2M} \beta \theta_M \varphi_F}{\varphi_F N_B + N_H}, & C_{14} &= b m_L, & C_{15} &= \frac{\beta_H \beta \theta_M \varphi_F}{\varphi_F N_B + N_H}, \\ C_{16} &= \kappa \eta_H + (1 - \kappa) \eta_H + \mu_H, & C_{17} &= p_1 a_{H1} + p_2 a_{H2}, & C_{18} &= \kappa \eta_H, & C_{19} &= \varepsilon + \gamma_H + \mu_H, \\ C_{20} &= (1 - \kappa) \eta_H, & C_{21} &= \sigma_H + \gamma_{NH}^N + \gamma_H^N(\varsigma) + \mu_H, & C_{22} &= r \gamma_H^I(\varsigma) + \delta_H + \mu_H, \\ C_{23} &= \varepsilon_1 + \delta_B^C + \mu_B, & C_{24} &= \varepsilon_2 + \delta_B^N + \mu_B, \end{aligned} \quad (65)$$

so that all the off-diagonal entries of C are non-negative and C is an M-matrix. Writing $G(X, Z) = CZ - \hat{G}(X, Z)$ and carrying out the subtraction gives

$$\hat{G}(X, Z) = \begin{pmatrix} (\bar{\lambda}_B^C + \lambda_B^C)(S_B^{C0} - S_B^C) \\ 0 \\ (\bar{\lambda}_B^N + \lambda_B^N)(S_B^{N0} - S_B^N) \\ 0 \\ 0 \\ 0 \\ 0 \\ \bar{\lambda}_M^F(S_M^{F0} - S_M^F) \\ 0 \\ (\bar{\lambda}_H + \lambda_H)(S_H^0 - S_H) \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}. \quad (66)$$

Since $S_B^C \leq S_B^{C0}$, $S_B^N \leq S_B^{N0}$, $S_M^F \leq S_M^{F0}$ and $S_H \leq S_H^0$ within Ω , we have $\hat{G}_i(X, Z) \geq 0$ for $i = 1, 2, \dots, 13$, so that condition (H2) is satisfied. Both hypotheses of Lemma 4 therefore hold, and the disease-free equilibrium is globally asymptotically stable whenever $R_0 < 1$.

3.8 Global stability of the endemic equilibrium

Following Cangiotti et al. (2023) and Korobeinikov and Maini (2004), the global stability of the endemic equilibrium is analysed by means of a logarithmic Lyapunov function. For any compartment X with equilibrium value X^* , define

$$g(X, X^*) = X - X^* - X^* \ln \frac{X}{X^*}. \quad (67)$$

Then $g(X^*, X^*) = 0$, $\partial g / \partial X = 1 - X^*/X$ vanishes at $X = X^*$, and $\partial^2 g / \partial X^2 = X^*/X^2 > 0$ for all $X > 0$, so that g is strictly convex. Expanding about X^* by Taylor's theorem and using $g(X^*) = g'(X^*) = 0$,

$$g(X, X^*) = \frac{1}{2} g''(\xi) (X - X^*)^2 > 0 \quad \text{for } X \neq X^*, \quad (68)$$

with ξ between X and X^* ; hence g is positive definite.

Theorem 6. *If $R_0 > 1$, the endemic equilibrium E^* of the system (11) is globally asymptotically stable in the interior of the feasible region Ω .*

Proof. Consider the Lyapunov function

$$V = V_M + V_B + V_H + V_D, \quad (69)$$

in which

$$\begin{aligned} V_M &= g(E_M^F, E_M^{F*}) + c_1 g(I_M^F, I_M^{F*}) + c_2 g(I_E, I_E^*) + c_3 g(I_L, I_L^*), \\ V_B &= \alpha_1 g(E_B^C, E_B^{C*}) + \alpha_2 g(I_B^C, I_B^{C*}) + \alpha_3 g(E_B^N, E_B^{N*}) + \alpha_4 g(I_B^N, I_B^{N*}), \\ V_H &= \beta_1 g(E_H, E_H^*) + \beta_2 g(I_H^A, I_H^{A*}) + \beta_3 g(I_H^N, I_H^{N*}) + \beta_4 g(I_H^I, I_H^{I*}), \\ V_D &= c_D g(D, D^*). \end{aligned} \quad (70)$$

The positive weights are chosen so that the cross terms generated by the flows between compartments cancel. Matching the flow $E_M^F \xrightarrow{\eta_M} I_M^F$ requires $c_1 \mu_M I_M^{F*} = \eta_M E_M^{F*}$, and using the equilibrium relation $\eta_M E_M^{F*} = \mu_M I_M^{F*} - b m_L I_L^*$ gives

$$c_1 = 1 - \frac{b m_L I_L^*}{\mu_M I_M^{F*}}, \quad \text{for which we take } c_1 = \frac{\eta_M}{\mu_M}. \quad (71)$$

Differentiating (69) along the solutions of (11), using $\frac{d}{dt} g(X, X^*) = (1 - X^*/X) \dot{X}$ together with the equilibrium relations, and rearranging each term into standard form, the components are

$$\begin{aligned} \frac{dV_M}{dt} &= -(\eta_M + \mu_M) E_M^{F*} \left[2 - \frac{E_M^F}{E_M^{F*}} - \frac{E_M^{F*}}{E_M^F} + \frac{S_M^F \bar{\lambda}_M^F}{S_M^{F*} \bar{\lambda}_M^{F*}} \right] \\ &\quad - c_1 \mu_M I_M^{F*} \left[2 - \frac{I_M^F}{I_M^{F*}} - \frac{I_M^{F*}}{I_M^F} + \frac{b m_L I_L}{b m_L I_L^*} + \frac{E_M^F}{E_M^{F*}} \right] \\ &\quad - c_2 (\nu + \mu_E) I_E^* \left[2 - \frac{I_E}{I_E^*} - \frac{I_E^*}{I_E} + \frac{I_M^F}{I_M^{F*}} \right] - c_3 (m_L + \mu_L) I_L^* \left[2 - \frac{I_L}{I_L^*} - \frac{I_L^*}{I_L} + \frac{I_E}{I_E^*} \right], \end{aligned} \quad (72)$$

$$\frac{dV_B^C}{dt} = -\alpha_1 (\eta_B^C + \mu_B) E_B^{C*} \left[2 - \frac{E_B^C}{E_B^{C*}} - \frac{E_B^{C*}}{E_B^C} + \frac{S_B^C}{S_B^{C*}} \cdot \frac{\bar{\lambda}_B^C + \lambda_B^C}{\bar{\lambda}_B^{C*} + \lambda_B^{C*}} \right] - \alpha_2 (\gamma_B^C + \delta_B^C + \mu_B) I_B^{C*} \left[2 - \frac{I_B^C}{I_B^{C*}} - \frac{I_B^{C*}}{I_B^C} + \frac{E_B^C}{E_B^{C*}} \right], \quad (73)$$

$$\frac{dV_B^N}{dt} = -\alpha_3 (\eta_B^N + \mu_B) E_B^{N*} \left[2 - \frac{E_B^N}{E_B^{N*}} - \frac{E_B^{N*}}{E_B^N} + \frac{S_B^N}{S_B^{N*}} \cdot \frac{\bar{\lambda}_B^N + \lambda_B^N}{\bar{\lambda}_B^{N*} + \lambda_B^{N*}} \right] - \alpha_4 (\gamma_B^N + \delta_B^N + \mu_B) I_B^{N*} \left[2 - \frac{I_B^N}{I_B^{N*}} - \frac{I_B^{N*}}{I_B^N} + \frac{E_B^N}{E_B^{N*}} \right], \quad (74)$$

$$\begin{aligned} \frac{dV_H}{dt} &= -\beta_1 \eta_H E_H^* \left[2 - \frac{E_H}{E_H^*} - \frac{E_H^*}{E_H} + \frac{S_H}{S_H^*} \cdot \frac{\bar{\lambda}_H + \lambda_H}{\bar{\lambda}_H^* + \lambda_H^*} \right] - \beta_2 (\varepsilon + \gamma_H + \mu_H) I_H^{A*} \left[2 - \frac{I_H^A}{I_H^{A*}} - \frac{I_H^{A*}}{I_H^A} + \kappa \frac{E_H}{E_H^*} \right] \\ &\quad - \beta_3 (\sigma_H + \gamma_{NH}^N + \gamma_H^N(s) + \mu_H) I_H^{N*} \left[2 - \frac{I_H^N}{I_H^{N*}} - \frac{I_H^{N*}}{I_H^N} + (1 - \kappa) \frac{E_H}{E_H^*} + \varepsilon \frac{I_H^A}{I_H^{A*}} \right] \\ &\quad - \beta_4 (r \gamma_H^I(s) + \delta_H + \mu_H) I_H^{I*} \left[2 - \frac{I_H^I}{I_H^{I*}} - \frac{I_H^{I*}}{I_H^I} + \sigma_H \frac{I_H^N}{I_H^{N*}} \right], \end{aligned} \quad (75)$$

$$\frac{dV_D}{dt} = -c_D \mu_D D^* \left[2 - \frac{D}{D^*} - \frac{D^*}{D} + \frac{I_B^C}{I_B^{C*}} + \frac{I_B^N}{I_B^{N*}} \right]. \quad (76)$$

Each bracketed expression has the form $[2 - X/X^* - X^*/X + (\text{coupling terms})]$, and by the arithmetic-geometric mean

inequality $a + 1/a \geq 2$ for $a > 0$, with equality if and only if $a = 1$, which follows from $(a - 1)^2 \geq 0$, we have

$$\frac{X}{X^*} + \frac{X^*}{X} \geq 2 \implies 2 - \frac{X}{X^*} - \frac{X^*}{X} \leq 0, \quad (77)$$

with equality if and only if $X = X^*$. Consequently

$$\frac{dV}{dt} = \frac{dV_M}{dt} + \frac{dV_B}{dt} + \frac{dV_H}{dt} + \frac{dV_D}{dt} \leq 0, \quad (78)$$

with $dV/dt = 0$ if and only if the system is at the endemic equilibrium E^* . Hence V is a Lyapunov function, $dV/dt \leq 0$ in the feasible region, and the largest invariant set on which $dV/dt = 0$ is the singleton $\{E^*\}$. By LaSalle's invariance principle the endemic equilibrium E^* is therefore globally asymptotically stable in the interior of the feasible region.

4 Sensitivity analysis

The input parameters of a transmission model must be considered carefully, because the rate of progression and the dynamics of the disease are governed by their uncertainty. Resolving this requires a sensitivity analysis, which assesses the robustness and sufficiency of the model and identifies those parameters that exert a significant influence on its output. Sensitivity analysis is therefore carried out here in order to determine the key transmission parameters that influence the basic reproduction number of WNV and each of its transmission pathways $R_0^{(M \leftrightarrow B)}$, $R_0^{(B \leftrightarrow B)}$, $R_{0,H}$ and $R_0^{(vert.)}$. The elasticity, or normalised forward sensitivity index, of R_0 with respect to a parameter ξ is defined by

$$\Upsilon_{\xi}^{R_0} = \frac{\partial R_0}{\partial \xi} \cdot \frac{\xi}{R_0}, \quad (79)$$

and (79) is applied by the direct differential method to the closed forms derived in Section 3.5, using the baseline parameter values at $T = 25^\circ\text{C}$ given in Table 2, with R_0 and each reproduction pathway taken in turn as the response function. The resulting indices are collected in Table 3, in which the parameters are ordered by the magnitude of their influence on R_0 , so that those with the greatest impact occupy the upper rows. The uncertainty range over which each parameter is allowed to vary is recorded alongside its indices.

Table 3. Normalised forward sensitivity indices of R_0 and of the four transmission pathways, evaluated at the baseline values of Table 2 with $T = 25^\circ\text{C}$. A dash indicates that the parameter does not enter the corresponding response function.

Parameter	Range	Υ_{R_0}	$\Upsilon_{R_0^{(M \leftrightarrow B)}}$	$\Upsilon_{R_0^{(B \leftrightarrow B)}}$	$\Upsilon_{R_{0,H}}$	$\Upsilon_{R_0^{(vert.)}}$
μ_M	0.025–0.25 [‡]	–0.77512	–1.25319	—	—	—
β	0.061–0.183 [‡]	+0.61852	+1.00000	—	—	—
θ_M	0.5–1.0 [‡]	+0.61852	+1.00000	—	—	—
β_{BN}	0.088–0.9	+0.45607	+0.33886	+0.64609	—	—
μ_D	0.1–1.0	–0.38146	—	–0.99995	—	—
a	0.8–1.0	+0.30943	+0.50028	—	—	+0.74782
m_L	0.333–1.0	+0.30943	+0.50028	—	—	–0.01935
K_L	6.09×10^7 – 1.83×10^8 [§]	+0.30926	+0.50000	—	—	—
N_H	1.53×10^7 – 4.60×10^7 [‡]	–0.30926	–0.50000	—	—	—
δ_B^N	0–0.5	–0.26226	–0.25392	–0.27580	—	—
π_2	0.04–1.0	+0.24646	—	+0.64607	—	—
β_{BC}	0.28–0.84 [‡]	+0.23468	+0.16114	+0.35391	—	—
β_{2M}	0–1.0	+0.20959	+0.33886	—	—	—
α_B	10–30	+0.20617	+0.33333	—	—	—
α_H	10–30 [‡]	–0.20617	–0.33333	—	—	—
δ_B^C	0–0.5	–0.16821	–0.14639	–0.20359	—	—
ε_2	0.315–0.945 [‡]	+0.16689	—	+0.43749	—	—
η_M	0.0235–0.0704 [‡]	+0.15591	+0.25206	—	—	—
π_1	0.04–1.0	+0.13500	—	+0.35389	—	—
γ_B^N	0–0.2	–0.11391	–0.08464	–0.16138	—	—
φ_F	2.5–7.5 [‡]	+0.10309	+0.16667	—	—	—
β_{1M}	0–1.0	+0.09967	+0.16114	—	—	—

Continued on next page

Table 3 continued from previous page

Parameter	Range	Υ^{R_0}	$\Upsilon^{R_0^{(M \leftrightarrow B)}}$	$\Upsilon^{R_0^{(B \leftrightarrow B)}}$	$\Upsilon^{R_{0,H}}$	$\Upsilon^{R_0^{(vert.)}}$
ε_1	0.5–1.5 [†]	+0.08998	—	+0.23587	—	—
p	0.125–0.375 [†]	+0.08265	+0.04818	+0.13854	—	—
γ_B^C	0–0.01	–0.02132	–0.01464	–0.03215	—	—
μ_B	1.8×10^{-4} – 5.5×10^{-4} [‡]	–0.00137	–0.00109	–0.00182	—	—
ϖ	1–500	$+6.9614 \times 10^{-4}$	+0.00113	—	—	—
η_B^N	0.125–0.375 [†]	$+6.6488 \times 10^{-4}$	$+4.9402 \times 10^{-4}$	$+9.4192 \times 10^{-4}$	—	—
η_B^C	0.165–0.495 [†]	$+2.5928 \times 10^{-4}$	$+1.7803 \times 10^{-4}$	$+3.9101 \times 10^{-4}$	—	—
μ_L	0.071–0.25	-1.7555×10^{-4}	-2.8383×10^{-4}	—	—	+0.01935
μ_E	0.0625–0.1875 [†]	-1.1145×10^{-4}	-1.8018×10^{-4}	—	—	—
ν	0.328–0.984 [†]	$+1.1145 \times 10^{-4}$	$+1.8018 \times 10^{-4}$	—	—	—
d_2	0.175–0.525 [†]	$+9.8068 \times 10^{-6}$	—	$+2.5707 \times 10^{-5}$	—	—
ϑ_2	0–0.5	$+9.8068 \times 10^{-6}$	—	$+2.5707 \times 10^{-5}$	—	—
d_1	0.425–1.0 [†]	$+8.0895 \times 10^{-6}$	—	$+2.1205 \times 10^{-5}$	—	—
ϑ_1	0–0.5	$+8.0895 \times 10^{-6}$	—	$+2.1205 \times 10^{-5}$	—	—
κ	0.375–1.0 [†]	—	—	—	+1.00000	—
ε	0.05–0.15	—	—	—	–0.99941	—
a_{H2}	0.00185–0.00247	—	—	—	+0.99083	—
p_2	0–0.2	—	—	—	+0.99083	—
a_{H1}	2×10^{-5} – 6×10^{-5} [‡]	—	—	—	+0.00917	—
p_1	0–0.1	—	—	—	+0.00917	—
μ_H	1.96×10^{-5} – 5.87×10^{-5} [‡]	—	—	—	-7.8162×10^{-4}	—
η_H	0.05–0.15 [†]	—	—	—	$+3.9085 \times 10^{-4}$	—
γ_H	1×10^{-5} – 3×10^{-5} [‡]	—	—	—	-1.9988×10^{-4}	—
b	0.05–0.15 [†]	—	—	—	—	+1.00000
τ	0–1.0	—	—	—	—	+1.00000

The ranges without a marker are those reported in Table 2. [†] No range is reported in Table 2; the range is taken as the baseline $\pm 50\%$, truncated at unity for proportions. [‡] The range has been widened to correspond to adult mosquito lifespans of 4–40 days, the tabled range spanning only a factor of 1.5. [§] K_L is fixed by the calibration $\langle S_M^F \rangle = N_M^0$ rather than taken from the table; the range is $\pm 50\%$ of the calibrated value.

The third column of Table 3 gives the indices with R_0 as the response function. The parameters with positive indices, such as β (mosquito biting rate), θ_M (non-diapausing mosquitoes), β_{BN} (transmission probability from mosquito to non-corvid bird), m_L (larval maturation rate) and α_B (mosquito-to-bird ratio), affect the reproduction number positively, so that a reduction in the value of any one of them, the rest being held constant, reduces R_0 and consequently the incidence of WNv. Conversely, the parameters carrying negative indices, such as μ_M (adult mosquito mortality), μ_D (environmental viral decay), N_H (total human population) and δ_B^N (disease-induced death rate of non-corvid birds), act inversely on R_0 , so that an increase in any one of them decreases the basic reproduction number and hence the spread of the disease. The ranking is dominated by the vector: μ_M carries -0.77512 while β and θ_M each carry $+0.61852$, so that a ten per cent change in any one of these three quantities moves R_0 by six to eight per cent.

The fourth column gives the indices with $R_0^{(M \leftrightarrow B)}$ as the response variable. Here β and θ_M each carry a unit elasticity and μ_M carries -1.25319 , the largest index recorded anywhere in the table, with m_L , a and K_L following at one half and N_H and α_H entering negatively. The mosquito-bird cycle is therefore governed almost entirely by vector demography and vector contact, so that measures raising μ_M , such as adulticide application, or lowering m_L and K_L , such as larviciding and breeding-site clearance, act on this pathway more than proportionately.

The fifth column presents the indices with respect to $R_0^{(B \leftrightarrow B)}$. The environmental viral decay rate is decisive, μ_D carrying -0.99995 , while β_{BN} and π_2 carry $+0.64609$ and $+0.64607$ and ε_2 carries $+0.43749$. The lateral avian pathway is thus controlled jointly by the persistence of virus in the environment and by the rate at which birds consume contaminated food and water, and is comparatively insensitive to the predation parameters d_i and ϑ_i , whose indices are of order 10^{-5} . Measures accelerating the inactivation of virus in food and water sources would therefore weaken this pathway substantially.

The sixth column shows the indices associated with $R_{0,H}$. The fraction of asymptomatic infections κ carries a unit elasticity, the transplant parameters a_{H2} and p_2 carry +0.99083 each, and the rate at which asymptomatic infections become recognised cases, ε , carries -0.99941 ; the transfusion parameters a_{H1} and p_1 are smaller by two orders of magnitude. To reduce the human sub-threshold, therefore, control should be directed at transmission through organ transplantation and at the early recognition of asymptomatic infection, since raising ε lowers $R_{0,H}$ almost one for one. The last column shows that the transovarial pathway is driven by the vertical transmission rate τ and the infected-larva maturation fraction b , each with unit elasticity, followed by a at +0.74782, every parameter carrying a positive index except m_L .

5 Numerical simulations

This section presents the results of the numerical simulation of the model (11), carried out in MATLAB R2015a over a horizon of ninety days with the initial data of Table 1 and the baseline parameter values of Table 2 evaluated at $T = 25^\circ\text{C}$, for which $R_0 = 1.844546 > 1$. The results are illustrated in Figures 2–8, which describe the time-dependent behaviour of the compartments of each interacting population and the response of the human classes to variation in the principal transmission parameters.

5.1 Behaviour of $S_H, I_H^A, E_H, I_H^N, I_H^I$ and R_H against time (days)

The six human compartments are displayed in Figure 2(a–f). The susceptible class begins at its calibrated demographic level of 3.0000×10^7 , falls to a minimum of about 2.9998×10^7 near the twenty-fifth day, and then recovers to approximately 3.0008×10^7 by the ninetieth, the recovery being driven by the recruitment Λ_H together with the return of recovered individuals at the waning rate $h\zeta$. The depletion amounts to roughly one part in fifteen thousand of the susceptible pool, which reflects the status of humans as incidental, dead-end hosts: the human force of infection is small beside the avian forces, and $R_{0,H}$ was shown in Section 3.5 to be structurally decoupled from the enzootic cycle. The exposed class peaks at about 590 individuals near the twentieth day and eases to some 510; the asymptomatic class peaks at about 445 near the twenty-fifth day and eases to some 390; and the non-neuroinvasive class peaks at about 97 near the twenty-second day and settles near 85. The near coincidence of these three maxima follows from the structure of the human block, in which E_H feeds I_H^A at the rate $\kappa\eta_H$ and I_H^N at the rate $(1 - \kappa)\eta_H$, so that all three track the infectious vector population with only the delay imposed by the incubation period $1/\eta_H$.

The neuroinvasive class behaves differently, rising monotonically to approximately 6.2×10^3 individuals without attaining a turning point, while the recovered class plateaus at about 8.3×10^3 by the seventieth day. This accumulation occurs because the neuroinvasive compartment is drained only by the saturated treatment function $r\gamma_H^I(\varsigma)$ and by the small mortalities δ_H and μ_H , so that severe cases accumulate for as long as I_H^N supplies them at the rate σ_H . The burden of acute infection therefore peaks and recedes within the first month, whereas the burden of severe disease, and hence of clinical care, continues to grow long afterwards.

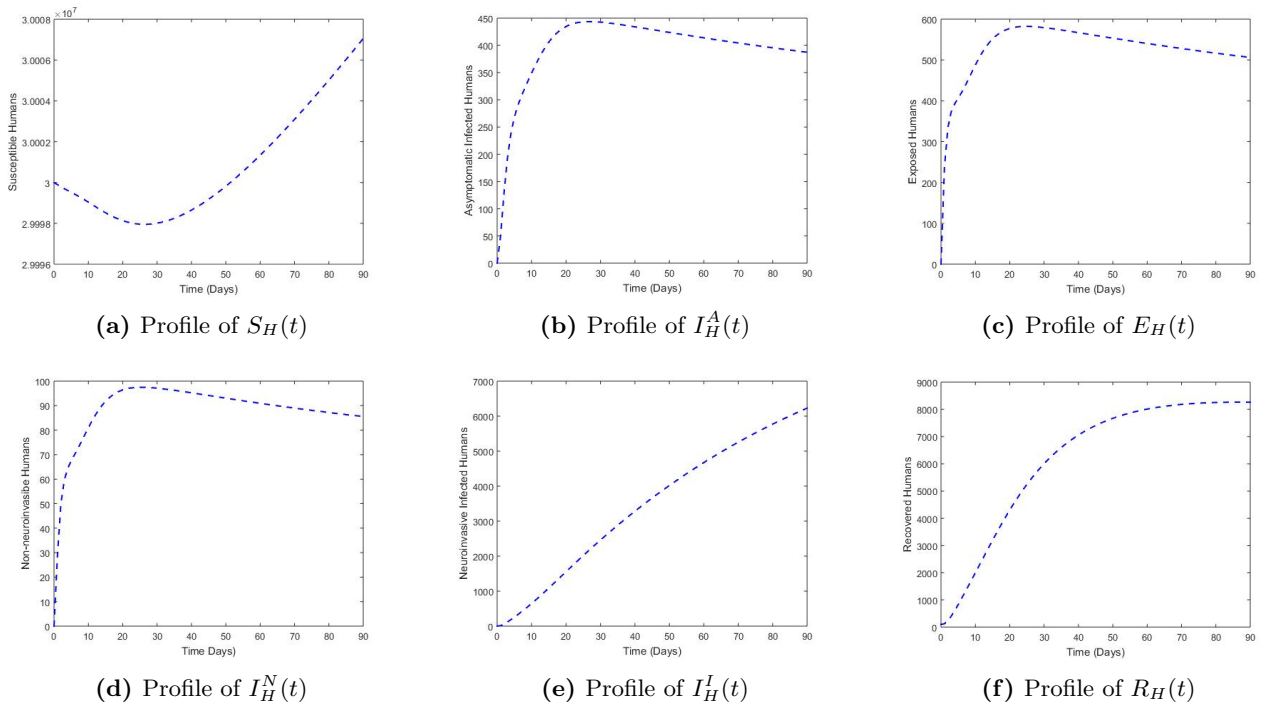


Figure 2. Dynamics of S_H , I_H^A , E_H , I_H^N , I_H^I and R_H populations against time (days).

5.2 Behaviour of S_B^C , E_B^C , I_B^C and R_B^C against time (days)

The corvid trajectories in Figure 3(a–d) describe an explosive epizootic. The susceptible class collapses from 7.70×10^7 birds to a negligible level within about eight days, the exposed class peaks at roughly 5.9×10^7 birds by the fourth day and is exhausted by the twenty-fifth, and the infected class peaks at about 1.95×10^7 birds near the sixth day before vanishing by the thirtieth. The recovered class peaks at about 4.1×10^7 birds around the sixteenth day and then decays to some 0.5×10^7 , under natural mortality together with the loss of immunity at the rate $q_1 \rho_B^C$.

The speed of the epizootic follows from the high transmission probability from mosquito to corvid bird, $\beta_{BC} = 0.5601$, together with the lateral routes carried by λ_B^C , so that the corvid class is exposed through three concurrent channels and the susceptible pool is consumed before recruitment can replenish it, while the high disease-induced mortality $\delta_B^C = 0.50$ removes infectious birds within an average of two days. These features account for the pattern in Table 3, where β_{BC} drives R_0 upward and δ_B^C downward: corvid competence is self-limiting, since the virulence that makes these birds efficient transmitters also removes them quickly from the infectious pool, as reported by Abdelrazec et al. (2014).

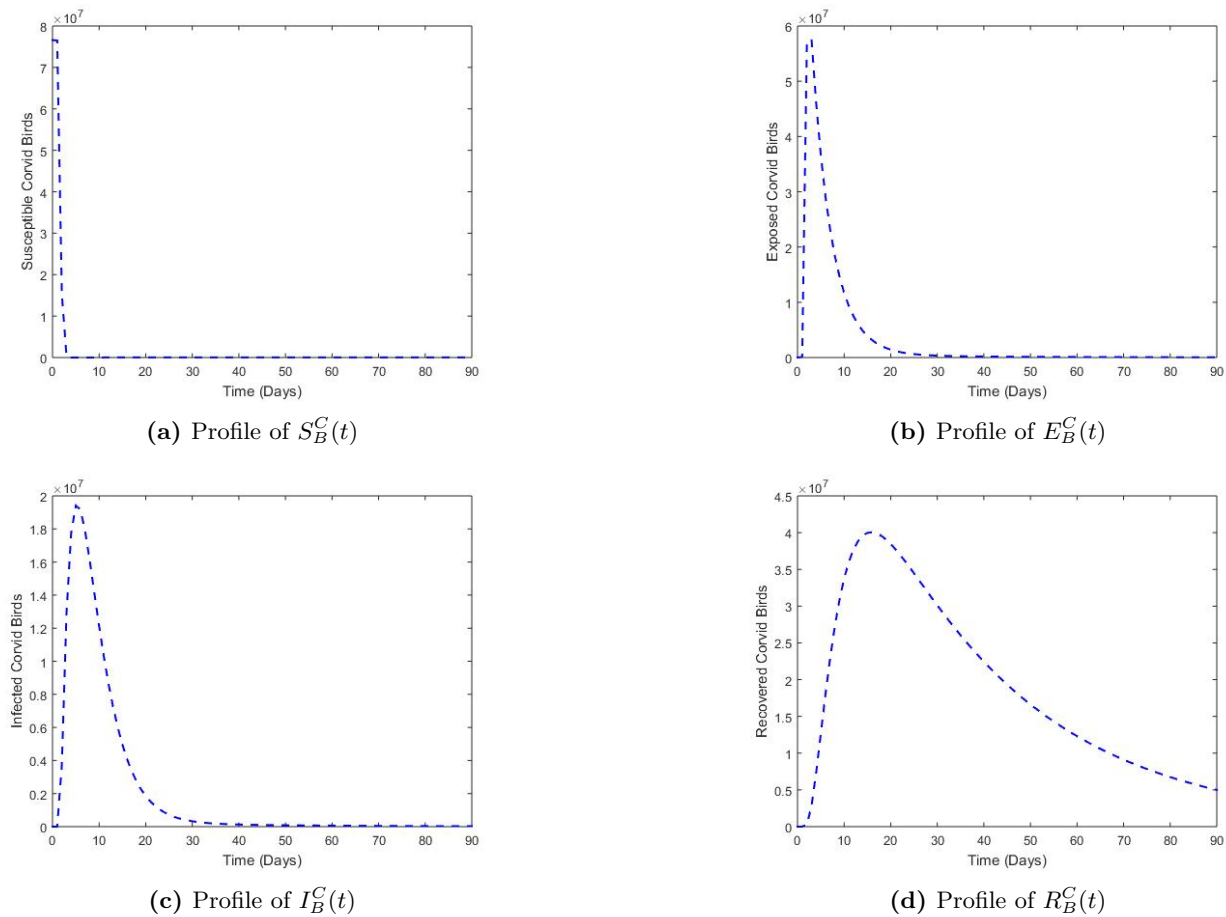


Figure 3. Dynamics of S_B^C , E_B^C , I_B^C and R_B^C populations against time (days).

5.3 Behaviour of S_B^N , E_B^N , I_B^N and R_B^N against time (days)

The non-corvid profiles in Figure 4(a–d) describe a slower and more protracted epizootic. The susceptible class rises sharply within two days to about 3.15×10^4 birds, falls to a minimum of roughly 4.5×10^3 between the fifteenth and twentieth day, and recovers to about 1.05×10^4 by the fiftieth. The exposed class peaks at approximately 8.7×10^4 birds near the tenth day, the infected class at about 4.85×10^4 birds near the fourteenth, and the recovered class at roughly 2.35×10^5 birds near the fortieth, after which all three decline gradually, the infected class remaining materially above zero throughout.

The non-corvid peaks occur some six to eight days later than the corvid ones and decay far more slowly, for two reasons: the lower disease-induced mortality $\delta_B^N = 0.3$ keeps infectious birds in circulation longer, and the recovery of the susceptible class after its minimum, sustained by the larger recruitment share $(1 - p)\Lambda_B$ and by the return of recovered birds at the rate $q_2\rho_B^N$, feeds a slow secondary transmission. The non-corvid group therefore acts as the persistent reservoir, as the additive structure of T_B anticipates, in accord with West and Chellamuthu (2020).

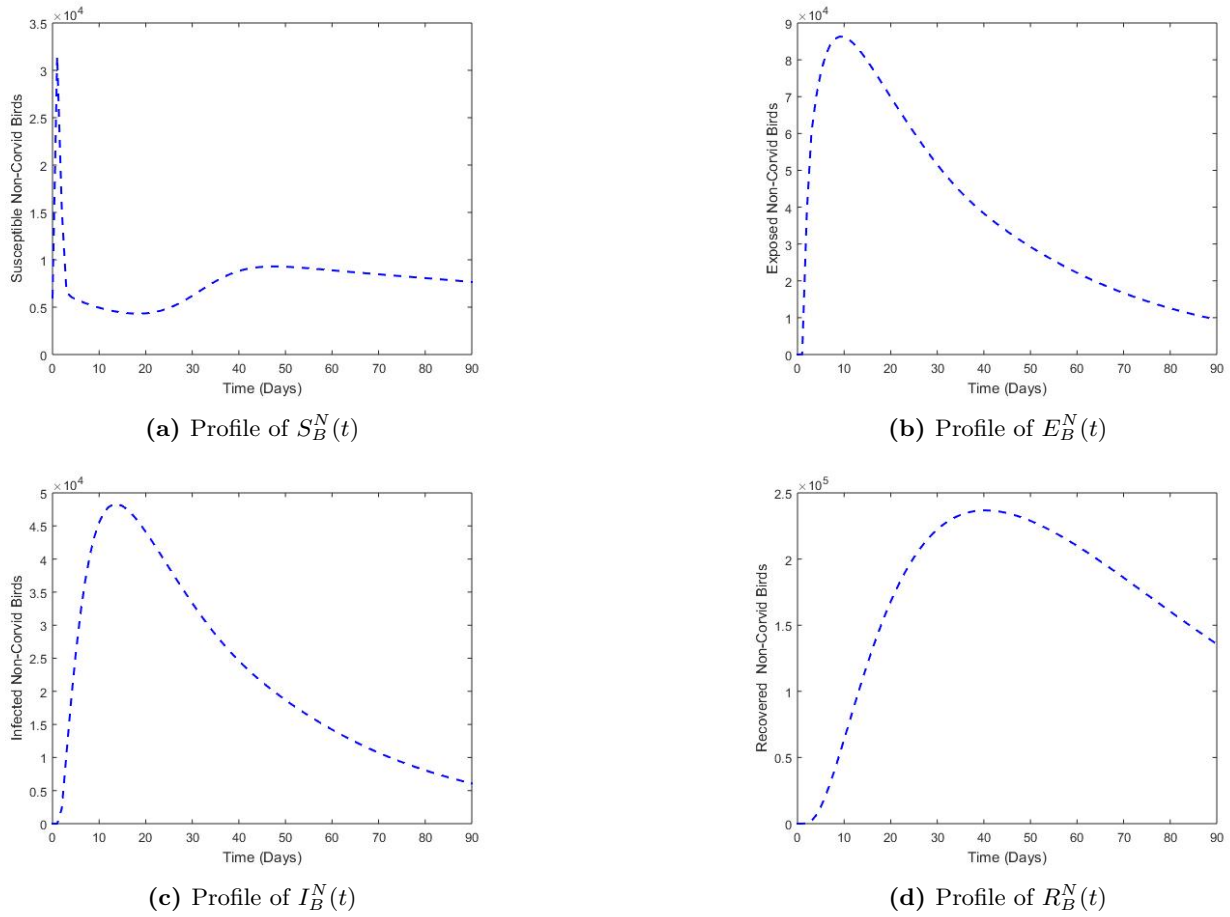


Figure 4. Dynamics of S_B^N , E_B^N , I_B^N and R_B^N populations against time (days).

5.4 Behaviour of S_M^F , E_M^F , I_M^F and S_L against time (days)

Figure 5(a–d) presents the adult vector and susceptible larval classes. The susceptible adult female class declines monotonically from 7.80×10^8 to approximately 3.2×10^8 mosquitoes, under infection at the force $\bar{\lambda}_M^F$ together with natural mortality μ_M . The exposed adult class spikes to about 2.15×10^4 mosquitoes near the seventh day and vanishes almost completely by the thirtieth, tracking the infectious avian classes with the delay imposed by the extrinsic incubation period. The infected adult class peaks at roughly 1.03×10^5 mosquitoes near the twenty-third day and then declines slowly, still standing at about 5.6×10^4 at the ninetieth day, while the susceptible larval class rises to a plateau of approximately 1.85×10^7 larvae imposed by Φ and the carrying capacity K_L .

The relationship between the exposed and infected adult classes is the clearest numerical signature of the transovarial route in the whole simulation. The exposed class is extinguished by the thirtieth day, since $\bar{\lambda}_M^F$ collapses once the avian infectious classes are depleted, and yet the infected adult class remains substantial for a further sixty days. Those infectious adults cannot have been recruited through $\eta_M E_M^F$; they are supplied by $abm_L I_L$, that is, by infected larvae maturing directly into infectious adults. Vertical transmission thus carries infection through the interval in which horizontal transmission from birds has ceased, the role assigned to $R_0^{(vert.)} = R_V$ in Section 3.5. Although $R_V = 0.0405$ is far too small to sustain transmission alone, its contribution is not negligible in time, which supports the experimental evidence of Nelms et al. (2013) and Baqar et al. (1993).

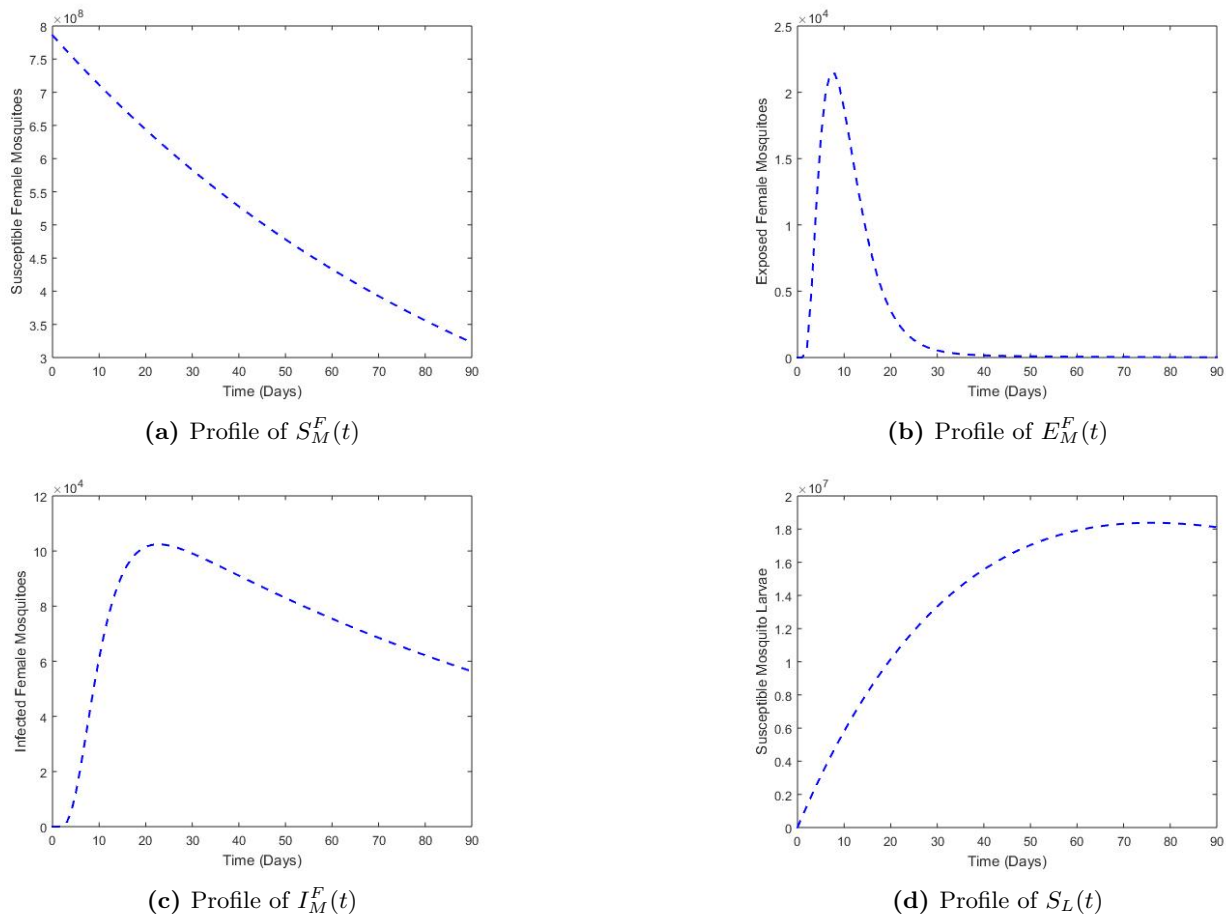


Figure 5. Dynamics of S_M^F , E_M^F , I_M^F and S_L populations against time (days).

5.5 Behaviour of S_E , I_E , I_L and D against time (days)

The immature vector classes and the environmental viral load are shown in Figure 6(a–d). The susceptible egg class declines from 8.20×10^7 to about 2.4×10^7 eggs, in step with the adult class that produces it, while the infected egg class rises sigmoidally to approximately 350 eggs and the infected larval class to about 7.9 larvae, both saturating near the eightieth day. That these classes saturate at low absolute levels, against an adult infectious population of order 10^5 , quantifies the limited but persistent character of the transovarial route: only the fraction τ of eggs laid by infectious females is infected, and only the fraction b of the resulting larvae matures into infectious adults.

The environmental compartment behaves quite differently, rising steeply to approximately 1.9×10^7 virus units by the twenty-fifth day and thereafter remaining almost constant. It saturates while the avian infected classes are at their peaks, driven by the shedding terms $(\varepsilon_1 + \delta_B^C + \mu_B)I_B^C$ and $(\varepsilon_2 + \delta_B^N + \mu_B)I_B^N$, and then persists because μ_D is slow relative to the rate at which carrion and excreta accumulate. It therefore outlives the epizootic that created it and continues to act as a source of infection through λ_i^c and λ_i^p long after the acute wave has passed. This is the mechanism behind the index of -0.99995 carried by μ_D against $R_0^{(B \leftrightarrow B)}$ in Table 3, and it identifies environmental viral persistence, alongside vector control, as a target of first importance.

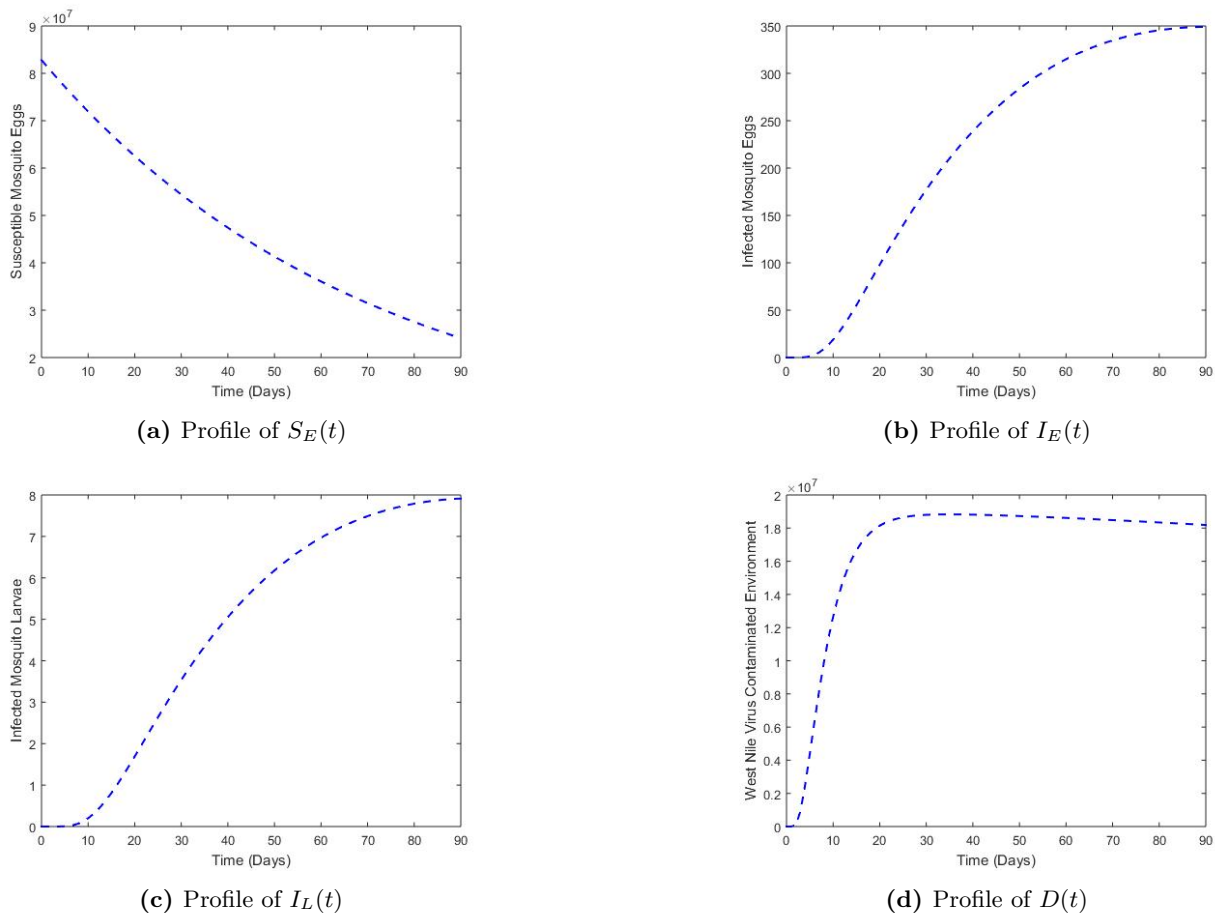


Figure 6. Dynamics of S_E , I_E , I_L and D populations against time (days).

5.6 Effect of variation in β_{1M} on the human classes

Figure 7(a–e) shows the effect on the human compartments of varying the transmission probability from corvid bird to mosquito over the values 0.25, 0.5, 0.75 and 1.0, all other parameters being held at their baseline values. The minimum of the susceptible class deepens from approximately 2.9998×10^7 to approximately 2.9992×10^7 and the trough moves later, from about the twenty-eighth day to about the fortieth. The peak of the asymptomatic class rises from roughly 415 to roughly 760 individuals, that of the exposed class from about 560 to about 1010, and that of the non-neuroinvasive class from about 95 to about 165, while the neuroinvasive class at the ninetieth day rises from approximately 6.0×10^3 to approximately 8.3×10^3 .

The ordering of the curves is monotone throughout, and a fourfold increase in the parameter almost doubles the peak human burden. The mechanism is indirect but decisive: β_{1M} does not appear anywhere in the human block, entering only $\bar{\lambda}_M^F$ and thence R_C , so that raising it enlarges the infectious vector population, which in turn raises $\bar{\lambda}_H$ and drives the human classes upward. The simulation thus confirms numerically what the decomposition of R_0 asserts analytically, that the human burden is a spillover from the enzootic cycle, and it shows that control directed at the vector-corvid interface yields proportionate benefit in humans.

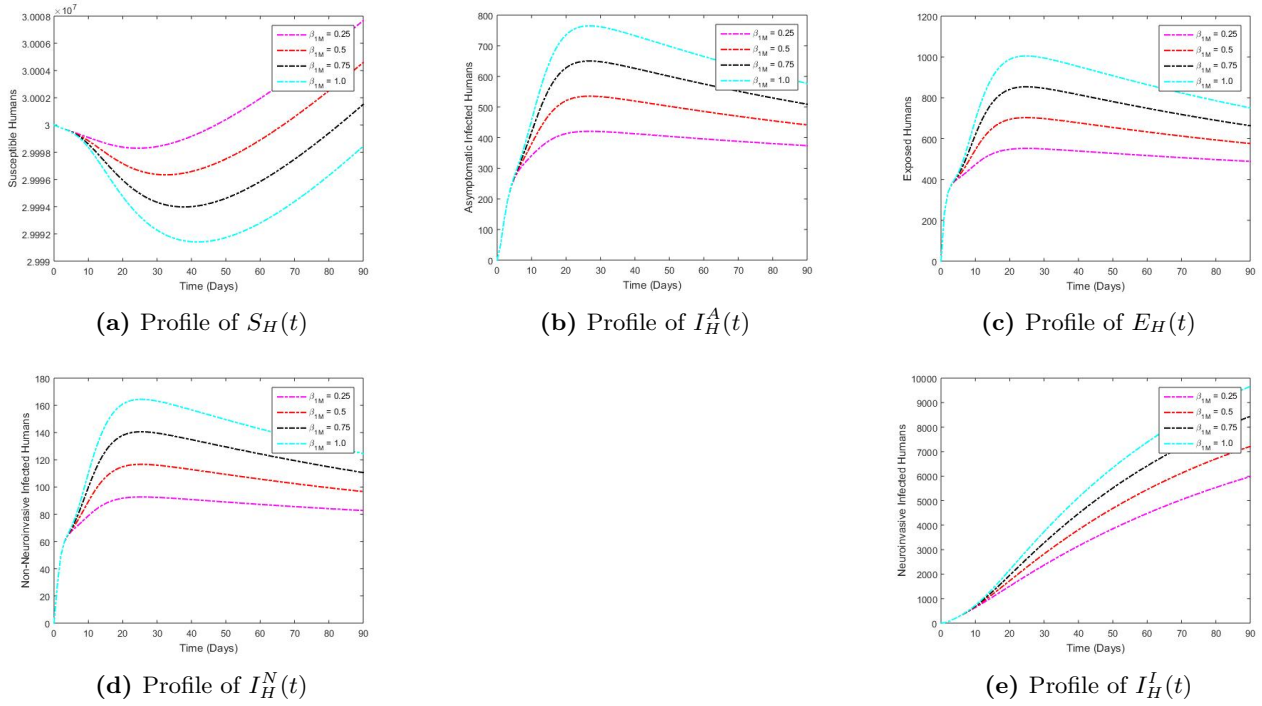


Figure 7. Effect of variation in β_{1M} on the S_H , I_H^A , E_H , I_H^N and I_H^I populations against time (days).

5.7 Effect of variation in β_H on the human classes

Figure 8(a–e) shows the corresponding response to variation in the transmission probability from mosquito to human over the same four values. The minimum of the susceptible class falls from about 2.9998×10^7 to about 2.99965×10^7 , the peak of the asymptomatic class rises from roughly 350 to roughly 500 individuals, that of the exposed class from about 470 to about 650, and that of the non-neuroinvasive class from about 350 to about 500, while the neuroinvasive class at the ninetieth day rises from approximately 4.8×10^3 to approximately 6.9×10^3 .

The response is monotone, as before, but appreciably weaker: the same fourfold increase raises the peak asymptomatic burden by about forty-three per cent against the eighty-three per cent produced by β_{1M} . The distinction is structural. Since humans are dead-end hosts, β_H appears only in $\bar{\lambda}_H$, scaling the rate at which infection is delivered to the human population without altering the enzootic cycle that generates the infectious vectors; it appears in none of R_C , T_B , R_V or $R_{0,H}$, and so does not alter the threshold R_0 at all. Variation in β_H therefore changes the size of the human epidemic but not the condition for its occurrence, whereas variation in β_{1M} changes both, so that personal protective measures mitigate the human burden without interrupting transmission and must be combined with measures acting on the enzootic cycle if elimination rather than mitigation is the objective. The same reasoning applies with greater force to the non-diapausing fraction θ_M , which multiplies every vector-borne force of infection and enters R_C quadratically, carrying indices of +0.61852 on R_0 and +1.00000 on $R_0^{(M \leftrightarrow B)}$ in Table 3: suppression of the actively host-seeking fraction of the vector population is the single most effective lever available within the model. This agrees with Bergsman et al. (2016) and Fesce et al. (2023), who identified vector-related quantities as the most critical for predicting WNV dynamics.

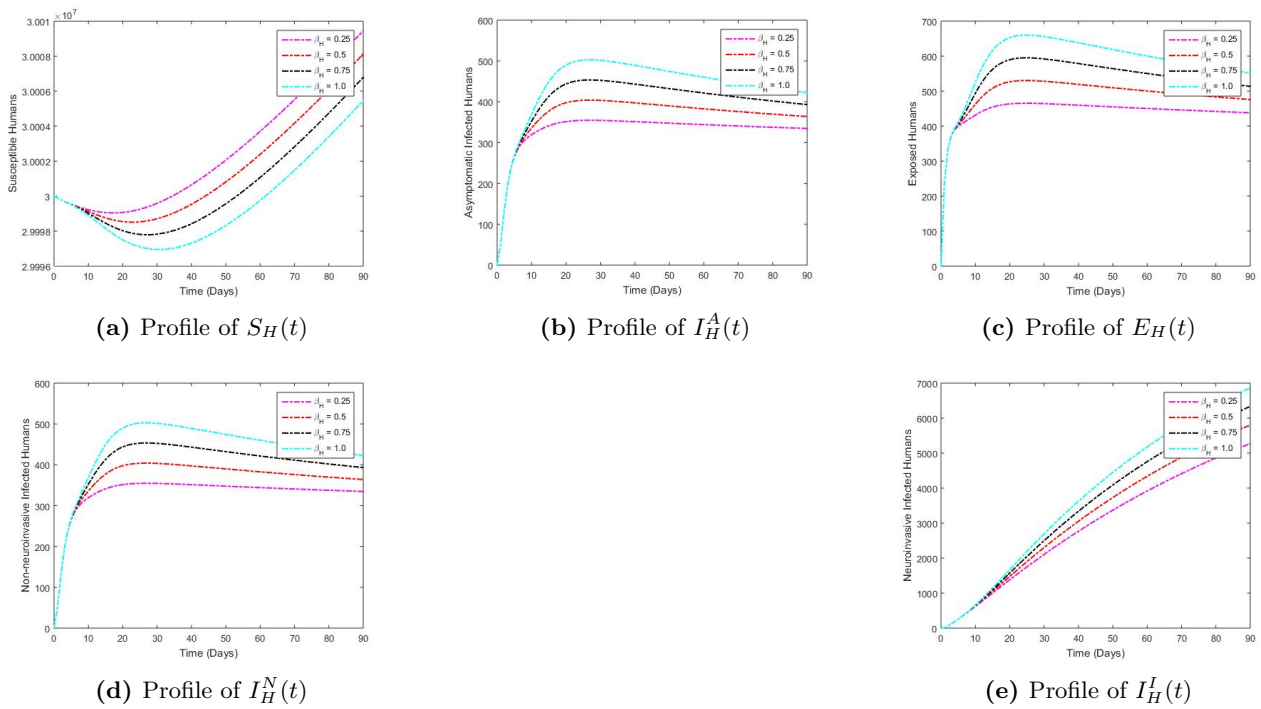


Figure 8. Effect of variation in β_H on the S_H , I_H^A , E_H , I_H^N and I_H^I populations against time (days).

6 Discussion

The analysis and the simulations together give a coherent account of how West Nile virus is maintained when the lateral and vertical routes are admitted alongside the classical cross-transmission route. Three findings deserve emphasis.

The first concerns the structure of the threshold. The next generation matrix does not yield a single indivisible reproduction number but a quantity $R_0 = (T_B + \sqrt{T_B^2 + 4R_C})/2$ in which the bird-to-bird pathway T_B and the mosquito-bird pathway R_C enter asymmetrically, the former linearly and the latter under a square root, together with two further quantities, R_V and $R_{0,H}$, that are separable from the enzootic cycle. The human component $R_{0,H} = \phi_{17}$ emerges as an isolated eigenvalue of the next generation matrix, which is the formal expression of the fact that humans are dead-end hosts: transfusion and transplantation can generate human infections from human infections, but they cannot feed back into the vector or avian populations, and the human sub-threshold therefore neither contributes to nor is contained in the enzootic threshold. Human-directed and cycle-directed interventions are therefore not substitutes, as the contrast between Figures 7 and 8 shows: variation in β_H rescales the human epidemic without moving R_0 , whereas variation in β_{1M} moves both.

The second concerns the standing of the two lateral routes that motivated the model. Neither is capable of sustaining transmission alone. The bird-to-bird pathway contributes T_B , which at the baseline parameterisation is of order unity but enters R_0 additively rather than multiplicatively, and the transovarial pathway contributes $R_V = 0.0405$, which is smaller by an order of magnitude than any other component. The simulations show why it would nonetheless be mistaken to neglect them. The environmental compartment $D(t)$ saturates within twenty-five days and then persists almost undiminished, so that the bird-to-bird route continues to deliver infection long after the acute epizootic has ended; and I_M^F remains substantial for sixty days after E_M^F has been extinguished, which is possible only because infected larvae mature directly into infectious adults. The lateral and vertical routes matter not for their magnitude at the threshold but for their persistence in time: they bridge the interval between successive amplification events. This is consistent with the dominant negative elasticity attaching to μ_D in Table 3 and with the experimental record of transovarial transmission in *Culex* reported by Miller et al. (2000), Dohm et al. (2002) and Nelms et al. (2013).

The third concerns the disaggregation of the avian host. The corvid and non-corvid classes do not merely differ in magnitude; they operate on different time scales, the corvids sustaining an intense epizootic spent

within a month and the non-corvids a slower one that persists throughout. The sensitivity analysis records the same asymmetry in the negative index carried by δ_B^C , which is paradoxical only until one observes that virulence is self-limiting: the corvid class amplifies efficiently because it is highly competent, and is transient for precisely the same reason. A model aggregating the avian population into a single class would reproduce neither behaviour, and would misstate both the height of the amplification peak and the duration of the reservoir.

7 Conclusion

This study has investigated the threshold dynamics, stability and sensitivity of a twenty-two compartment deterministic model for the transmission of West Nile virus which divided the avian host into corvid and non-corvid classes, resolves the immature stages of the vector, carries an environmentally persistent viral compartment, and admits lateral transmission within the avian and human populations together with vertical transmission within the vector population, under constant control. The positivity of solutions was established and a biologically feasible invariant region was identified, so that the model is mathematically well posed and biologically meaningful in Ω . Two disease-free equilibria were obtained, the trivial one corresponding to the absence of the vector population and the non-trivial one existing if and only if the demographic vector reproduction ratio $\mathcal{R}_M$ exceeds unity. The basic reproduction number was derived by the next generation matrix approach and was shown to decompose into four sub-reproduction numbers associated with the mosquito-bird cycle, the bird-to-bird and environmental pathway, vertical transmission and human spillover, the human component being structurally decoupled from the enzootic cycle. Stability analysis based on the Routh–Hurwitz criterion and the Castillo-Chavez framework demonstrated that the disease-free equilibrium is locally and globally asymptotically stable whenever $R_0 < 1$, while a logarithmic Lyapunov function together with LaSalle’s invariance principle established the global asymptotic stability of the endemic equilibrium whenever $R_0 > 1$, so that $R_0 = 1$ acts as a sharp threshold and no backward bifurcation arises. Sensitivity analysis identified vector survival, biting and transmission intensity and environmental viral persistence as the dominant drivers of transmission, and the numerical simulations illustrated the temporal behaviour of every compartment and showed that suppression of the actively host-seeking fraction of the vector population is the most effective single lever available within the model, personal protective measures reducing the human burden without altering the threshold. These findings provide a rigorous analytical basis and a practically actionable framework for West Nile virus surveillance and control in endemic settings.

Availability of data and material

Not applicable.

Competing interest

The authors declare no competing interest.

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