A Compartmental Model for Rabies Transmission with Behavioral Interventions: Vaccination and Public Awareness

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Abstract

Rabies remains one of the most lethal zoonotic diseases, causing tens of thousands of human deaths annually, the overwhelming majority transmitted through bites from infected domestic dogs. Conventional epidemic models of rabies treat vaccination coverage and human care-seeking behaviour as fixed parameters, yet in practice both are shaped dynamically by community awareness and campaign investment. In this paper we develop and analyse a nine-dimensional human–dog compartmental model in which dog vaccination uptake, human post-exposure prophylaxis (PEP) uptake, and bite-avoidance behaviour are all coupled to a time-evolving behavioural awareness variable $A(t)$. We establish positivity and boundedness of solutions, derive the disease-free equilibrium, and use the next-generation matrix approach to obtain a closed-form basic reproduction number R_0 that depends explicitly on the awareness-driven vaccination rate. Local stability of the disease-free equilibrium is established for $R_0 < 1$. A normalized forward sensitivity analysis identifies the dog-to-dog transmission rate and the disease-induced death rate in dogs as the most influential parameters, followed by the awareness-driven vaccination gain. Numerical simulations contrasting a low-awareness baseline against an active behavioural-intervention scenario show that sustained investment in public awareness campaigns can suppress R_0 from above unity to below unity, collapse the peak of infectious dogs, and reduce human rabies deaths by more than an order of magnitude. We conclude with policy recommendations for integrating behavioural-communication investment into dog-mediated rabies elimination programmes

Journal of Policy and Development Studies (JPDS)

Vol. 20 Issue 5 (2026)
ISSN(p) 1597-9385
ISSN (e) 2814-1091
Home page:
https://ajol.info/index.php/jpds

***ARTICLE INFO*:**
Keywords
Rabies, compartmental model, behavioural epidemiology, basic reproduction number, sensitivity analysis, vaccination, public awareness

Received: 9 April 2026
Revised: 20 June 2026
Accepted: 26 June 2026

DOI
https://dx.doi.org/10.4314/jpds.v20i5.13

1. Introduction

Rabies is vaccine-preventable, invariably fatal viral encephalitis that causes an estimated 59,000 human deaths every year, over 99% of which follow bites from rabid domestic dogs, predominantly in Africa and Asia (WHO, 2018). A recent Global Burden of Disease analysis confirms that this mortality remains disproportionately concentrated among children, older adults, and low-sociodemographic-index countries in Africa and South Asia, even as global incidence has declined markedly since 1990 (Yang et al., 2025). Because the disease circulates endemically in dog populations, the World Health Organization, the World Organisation for Animal Health, the Food and Agriculture Organization and the Global Alliance for Rabies Control have set a joint target of zero human deaths from dog-mediated rabies by 2030, built around two pillars: (i) mass dog vaccination to interrupt transmission at its animal reservoir, and (ii) accessible post-exposure prophylaxis (PEP) to prevent disease in already-exposed humans (WHO, 2018).

Both pillars are, in practice, behavioural interventions before they are biomedical ones. A vaccine only protects a dog if an owner brings it to a vaccination point; PEP only saves a bitten person if that person recognises the risk, seeks care promptly, and completes the regimen. Historically, mathematical models of rabies transmission have represented vaccination coverage and treatment-seeking as constant, exogenously fixed parameters (Hampson et al., 2009). This omits a first-order feature of real elimination campaigns: community awareness, generated by education programmes and amplified by the visible occurrence of cases, is what drives vaccination uptake and care-seeking in the first place, and it is itself something that public-health investment can shape.

This paper develops a human–dog compartmental model in which an explicit behavioural awareness variable $A(t)$ mediates three channels simultaneously: the rate at which dog owners vaccinate their animals, the rate at which bitten humans seek PEP, and the degree to which humans practice bite-avoidance behaviour that lowers their exposure risk. Awareness itself grows through sustained campaign investment and through a feedback response to observed disease prevalence, and decays through natural forgetting. This structure places our work within the broader behavioural-epidemiology literature that couples disease dynamics to information/behaviour dynamics (Funk et al., 2010; Manfredi & D'Onofrio, 2013), and extends it to the specific two-species structure of dog-mediated rabies. This literature has grown substantially in recent years, with awareness- and media-campaign-driven vaccination behaviour now modelled explicitly in general epidemic settings (Akdim et al., 2022; Akinwande et al., 2023; Verma et al., 2024) and with socially stratified risk perception shown to alter epidemic trajectories in ways that a single, population-averaged awareness variable cannot capture (Harris et al., 2023) — a simplification we adopt here for tractability but flag as a direction for extension.

Recent empirical work reinforces the premise that vaccination and care-seeking are behaviourally, not just biomedically, determined. In a cluster-randomised trial across the Mara region of Tanzania, community-based, continuously delivered dog vaccination sustained substantially higher and more stable coverage than conventional annual team-based campaigns, directly demonstrating that the delivery mechanism through which awareness and access translate into vaccination uptake shapes population-level coverage (Lankester et al., 2024). Complementing this, a study of participation in mass dog vaccination clinics in Tanzania found that community engagement strategies increased owner turnout, while decentralised delivery approaches such as the thermotolerant-vaccine "Zeepot" system were shown to improve both community awareness and sustained coverage between campaign pulses (Duamor et al., 2022; Lugelo et al., 2022). On the human side, recent Tanzanian studies confirm that PEP-seeking is far from automatic: cross-sectional evidence from the Maswa District found that compliance with the full PEP regimen was significantly associated with cost, distance, and awareness-related factors (Walwa et al., 2024), while comparable studies elsewhere in East Africa report that financial and logistical barriers delay PEP initiation beyond the clinically recommended 24-hour window for a substantial share of bite victims (Kisaka et al., 2022). Consistent with the behavioural mechanism modelled here, a phone text-message reminder intervention in rural Kenya significantly improved completion of the full PEP dosing schedule among bite victims (Chuchu et al., 2023), and community-level surveys in Kenya and Vietnam both identify household awareness and knowledge-attitude-practice profiles as strong predictors of proactive dog vaccination and bite-response behaviour (Ochieng et al., 2024; Tran et al., 2025). A recent review of One-Health strategies toward the 2030 elimination target likewise identifies uneven awareness, PEP access, and vaccination coverage — rather than any single biomedical constraint — as the principal bottlenecks to elimination (Zha et al., 2025). These findings collectively support the modelling choice, adopted here, of treating vaccination uptake and PEP-seeking as endogenous functions of awareness rather than fixed parameters.

The remainder of the paper is organised as follows. Section 2 formulates the model and presents the compartmental flow diagram. Section 3 gives the qualitative analysis: positivity, boundedness, the disease-free equilibrium, and the basic reproduction number R_0 obtained via the next-generation matrix method, together with a local stability result. Section 4 performs a normalized forward sensitivity analysis of R_0. Section 5 reports numerical simulations contrasting weak and strong behavioural-intervention scenarios, with interpretation. Section 6 concludes and offers policy recommendations.

2.1 Model Formulation

We partition the population into a dog subpopulation and a human subpopulation, coupled through a one-directional force of infection (dog bites cause human infection; there is no documented human-to-human or human-to-dog rabies transmission, so the human classes form an epidemiological dead end that does not feed back into the reproduction number). This compartmental structure builds on a growing body of recent dog-rabies modelling work that derives closed-form reproduction numbers via the next-generation matrix and incorporates vaccination explicitly into the dog subsystem (Hassan et al., 2024; Ali & Islam, 2026; Thichumpa et al., 2025).

2.1.1 Dog subpopulation

The dog population of size $N_d(t)$ is divided into susceptible dogs S_d, vaccinated dogs V_d, exposed (infected, not yet infectious) dogs E_d, and infectious dogs I_d, so that

$$N_d = S_d + V_d + E_d + I_d.$$

2.1.2 Human subpopulation

The human population of size $N_h(t)$ is divided into susceptible humans S_h, exposed/bitten humans E_h (incubating, not yet symptomatic), successfully treated humans V_h (received PEP and are protected), and symptomatic infectious humans I_h (untreated progression, which is essentially always fatal), so that

$$N_h = S_h + E_h + V_h + I_h.$$

2.1.3 Behavioural awareness

The scalar $A(t) \in [0,1]$ represents the community's level of rabies awareness/behavioural preparedness. It increases with sustained public-education campaign investment θ and with a feedback response of strength κ to observed disease prevalence in both species, and decays at a per-capita "forgetting" rate μ_A.

2.1.4 Behaviour-dependent rates

Two model rates are increasing functions of awareness (dog vaccination rate and human PEP-uptake rate), and the effective human exposure rate is scaled down by bite-avoidance behaviour:

$$\phi_d(A) = \phi_{d0} + \phi_{d1}A, \qquad \tau_h(A) = \tau_{h0} + \tau_{h1}A,$$

with $\varepsilon \in [0,1]$ the maximum attainable behavioural protection against bites.

2.1.5 Governing equations

With frequency-dependent (mass-action normalized by population size) transmission, the dog subsystem reads:

$$\begin{aligned}
\frac{dS_d}{dt} &= \Lambda_d - \beta_d \frac{I_d}{N_d} S_d - \phi_d(A)S_d + \omega_d V_d - \mu_d S_d, \\
\frac{dV_d}{dt} &= \phi_d(A)S_d - \omega_d V_d - \mu_d V_d, \\
\frac{dE_d}{dt} &= \beta_d \frac{I_d}{N_d} S_d - \sigma_d E_d - \mu_d E_d, \\
\frac{dI_d}{dt} &= \sigma_d E_d - (\delta_d + \mu_d) I_d,
\end{aligned}$$

and the human subsystem reads:

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \beta_h (1 - \varepsilon A) \frac{I_d}{N_d} S_h - \mu_h S_h, \\
\frac{dE_h}{dt} &= \beta_h (1 - \varepsilon A) \frac{I_d}{N_d} S_h - (\tau_h(A) + \sigma_h + \mu_h) E_h, \\
\frac{dV_h}{dt} &= \tau_h(A) E_h - \mu_h V_h, \\
\frac{dI_h}{dt} &= \sigma_h E_h - (\delta_h + \mu_h) I_h,
\end{aligned}$$

with the behavioural awareness equation:

$$\frac{dA}{dt} = \theta(1 - A) + \kappa \left(\frac{I_d}{N_d} + \frac{I_h}{N_h} \right)(1 - A) - \mu_A A,$$

subject to non-negative initial conditions. All parameters are strictly positive; their descriptions and illustrative baseline values (informed by the ranges reported in the rabies epidemiology literature; Hampson et al., 2009; WHO, 2018) are summarized in Table 1.

Table 1. Model parameters, description and baseline values used in simulation.

Symbol	Description	Unit	Baseline value	Reference
Λ_d	Dog recruitment rate	dogs/yr	500	Hampson et al. (2009)
μ_d	Natural death rate, dogs	/yr	0.40	Hampson et al. (2009)
β_d	Dog-to-dog effective transmission rate	/yr	115	Hampson et al. (2009)
σ_d	Progression rate $E_d \to I_d$ (1/incubation)	/yr	17.4	Hampson et al. (2009)
δ_d	Disease-induced death rate, dogs	/yr	73.0	Hampson et al. (2009)
ω_d	Vaccine waning rate	/yr	0.333	Lugelo et al. (2022)
ϕ_{d0}	Baseline (routine) dog vaccination rate	/yr	0.10	Lankester et al. (2024); Duamor et al. (2022)
ϕ_{d1}	Awareness-driven vaccination gain	/yr	0–1.50	Assumed
Λ_h	Human recruitment rate	people/yr	1000	Assumed (standard demographic)
μ_h	Natural death rate, humans	/yr	1/70	WHO (2018)
β_h	Dog-to-human effective exposure rate	/yr	0.55	Hampson et al. (2009)
σ_h	Progression rate $E_h \to I_h$ (1/incubation)	/yr	6.08	WHO (2018)
δ_h	Disease-induced death rate, humans	/yr	60.0	WHO (2018)
τ_{h0}	Baseline PEP-uptake rate	/yr	3.0	Chuchu et al. (2023); Kisaka et al. (2022)
τ_{h1}	Awareness-driven PEP-uptake gain	/yr	0–15.0	Assumed (illustrative — see Limitations)
ε	Maximum bite-avoidance protection	–	0–0.6	Assumed (illustrative — see Limitations)
θ	Awareness campaign investment rate	/yr	0.02–1.0	Assumed (illustrative — see Limitations)
κ	Case-driven awareness responsiveness	–	2.0	Assumed (illustrative — see Limitations)
μ_A	Awareness decay (forgetting) rate	/yr	0.5	Assumed (illustrative — see Limitations)

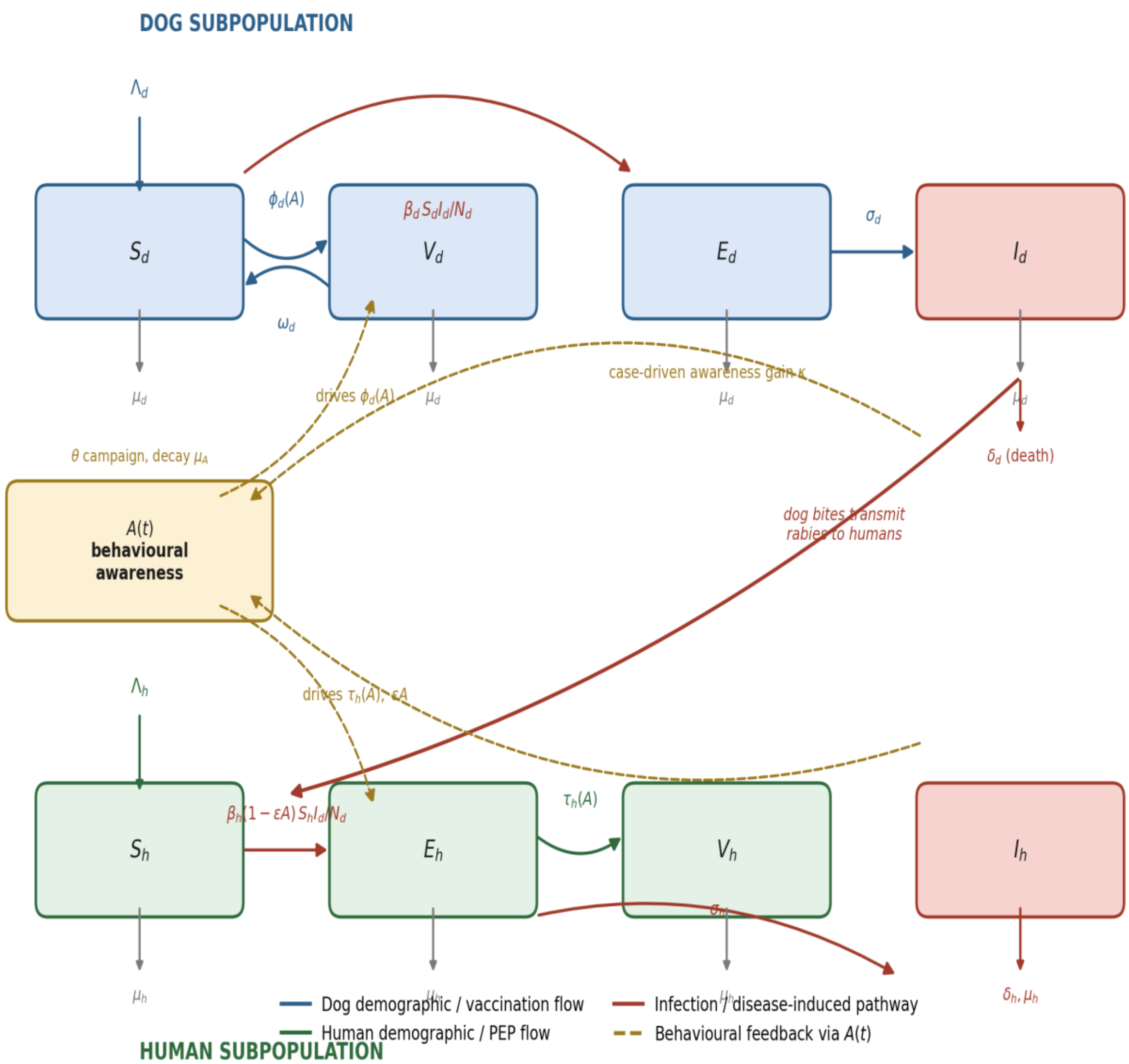

Figure 1: Compartmental flow diagram of the human–dog rabies model with behaviour-dependent vaccination, PEP-uptake and bite-avoidance, coupled through the awareness variable $A(t)$.

3.1 Model Analysis

3.1.1 Positivity and boundedness

Theorem 1. *Let* $\{S_d(0) \geq 0,\ V_d(0) \geq 0,\ E_d(0) \geq 0,\ I_d(0) \geq 0,\ S_h(0) \geq 0,\ E_h(0) \geq 0,\ V_h(0) \geq 0,\ I_h(0) \geq 0,\ A(0) \geq 0\} \in \mathbb{R}_+^9$ *be the set of initial solutions for the model system (1)–(9). Then the solution* $\big(S_d(t), V_d(t), E_d(t), I_d(t), S_h(t), E_h(t), V_h(t), I_h(t), A(t)\big)$ *of the model system (1)– (9) is positive for all* $t \geq 0$.

Proof. Considering the first equation in the model system (1)– (9), we have

$$\frac{dS_d}{dt} = \Lambda_d - \beta_d \frac{I_d}{N_d} S_d - \phi_d(A) S_d + \omega_d V_d - \mu_d S_d \ \geq\ -\left(\beta_d \frac{I_d}{N_d} + \phi_d(A) + \mu_d\right) S_d,$$

since $\Lambda_d > 0$ and $\omega_d V_d \geq 0$. Integrating and applying the initial condition yields

$$S_d(t) \;\geq\; S_d(0)\exp\left(-\int_0^t (\beta_d \frac{I_d(s)}{N_d(s)} + \phi_d\big(A(s)\big) + \mu_d)ds\right) \;\geq\; 0.$$

Using a similar approach, it can be shown that:

$$\begin{aligned}
\frac{dV_d}{dt} &= \phi_d(A)S_d - (\omega_d + \mu_d)V_d \;\geq\; -(\omega_d + \mu_d)V_d \\
&\implies V_d(t) \geq V_d(0)\exp(-(\omega_d + \mu_d)t) \geq 0, \\
\frac{dE_d}{dt} &= \beta_d \frac{I_d}{N_d} S_d - (\sigma_d + \mu_d)E_d \;\geq\; -(\sigma_d + \mu_d)E_d \\
&\implies E_d(t) \geq E_d(0)\exp(-(\sigma_d + \mu_d)t) \geq 0, \\
\frac{dI_d}{dt} &= \sigma_d E_d - (\delta_d + \mu_d)I_d \;\geq\; -(\delta_d + \mu_d)I_d \\
&\implies I_d(t) \geq I_d(0)\exp(-(\delta_d + \mu_d)t) \geq 0, \\
\frac{dS_h}{dt} &= \Lambda_h - \beta_h(1 - \varepsilon A)\frac{I_d}{N_d} S_h - \mu_h S_h \;\geq\; -\left(\beta_h(1 - \varepsilon A)\frac{I_d}{N_d} + \mu_h\right) S_h \\
&\implies S_h(t) \geq S_h(0)\exp\left(-\int_0^t (\beta_h\big(1 - \varepsilon A(s)\big)\frac{I_d(s)}{N_d(s)} + \mu_h)ds\right) \geq 0, \\
\frac{dE_h}{dt} &= \beta_h(1 - \varepsilon A)\frac{I_d}{N_d} S_h - (\tau_h(A) + \sigma_h + \mu_h)E_h \;\geq\; -(\tau_h(A) + \sigma_h + \mu_h)E_h \\
&\implies E_h(t) \geq E_h(0)\exp\left(-\int_0^t (\tau_h\big(A(s)\big) + \sigma_h + \mu_h)ds\right) \geq 0, \\
\frac{dV_h}{dt} &= \tau_h(A)E_h - \mu_h V_h \;\geq\; -\mu_h V_h \\
&\implies V_h(t) \geq V_h(0)\exp(-\mu_h t) \geq 0, \\
\frac{dI_h}{dt} &= \sigma_h E_h - (\delta_h + \mu_h)I_h \;\geq\; -(\delta_h + \mu_h)I_h \\
&\implies I_h(t) \geq I_h(0)\exp(-(\delta_h + \mu_h)t) \geq 0, \\
\frac{dA}{dt} &= \theta(1 - A) + \kappa\left(\frac{I_d}{N_d} + \frac{I_h}{N_h}\right)(1 - A) - \mu_A A \\
&\geq\; -\left[\theta + \kappa\left(\frac{I_d}{N_d} + \frac{I_h}{N_h}\right) + \mu_A\right] A,
\end{aligned}$$

since $\theta(1 - A) + \theta A = \theta \geq 0$ and likewise for the κ-term, so that

$$A(t) \;\geq\; A(0)\exp\left(-\int_0^t [\,\theta + \kappa(\frac{I_d(s)}{N_d(s)} + \frac{I_h(s)}{N_h(s)}) + \mu_A]ds\right) \;\geq\; 0.$$

Therefore, the solutions of the model system (1)– (9) are positive $\forall\, t \geq 0$. □

3.1.2 Boundedness of the model solutions

To show that the solution of model system (1)– (9) is bounded, we state and prove Theorem 2.

Theorem 2. *The solution set of the model system (1)– (9) defined in the region Ω is positively invariant, where* $\Omega = \left\{(S_d, V_d, E_d, I_d, S_h, E_h, V_h, I_h, A) \in \mathbb{R}_+^8 \times [0,1] :\ 0 \le N_d \le \frac{\Lambda_d}{\mu_d},\ \ 0 \le N_h \le \frac{\Lambda_h}{\mu_h},\ \ 0 \le A \le 1\right\}$.

Proof. Adding equations (1)– (4) for the dog subpopulation, the vaccination, waning and infection terms cancel in pairs, leaving

$$\frac{dN_d}{dt} = \Lambda_d - \delta_d I_d - \mu_d N_d \ \le\ \Lambda_d - \mu_d N_d,$$

since $\delta_d, I_d \ge 0$. Using the integrating factor and applying the initial condition, we obtain

$$N_d(t) \ \le\ \frac{\Lambda_d}{\mu_d} + \left(N_d(0) - \frac{\Lambda_d}{\mu_d}\right) e^{-\mu_d t}.$$

Taking the limit as $t \to \infty$, it can be shown that

$$N_d(t) \ \le\ \frac{\Lambda_d}{\mu_d}.$$

Similarly, adding equations (5)– (8) for the human subpopulation, the exposure and progression terms cancel in pairs, leaving

$$\frac{dN_h}{dt} = \Lambda_h - \delta_h I_h - \mu_h N_h \ \le\ \Lambda_h - \mu_h N_h.$$

Using the integrating factor and applying the initial condition, we obtain

$$N_h(t) \ \le\ \frac{\Lambda_h}{\mu_h} + \left(N_h(0) - \frac{\Lambda_h}{\mu_h}\right) e^{-\mu_h t}, \qquad N_h(t) \ \le\ \frac{\Lambda_h}{\mu_h} \ \text{ as } t \to \infty.$$

Since $N_d(t) > 0$ and $N_h(t) > 0$ for all $t \ge 0$ (Theorem 1), the ratios I_d/N_d and I_h/N_h appearing in (9) are well defined, and both lie in $[0,1]$ because each is a non-negative sub-population divided by the total population that contains it. Consequently, from

$$\frac{dA}{dt} = \theta(1 - A) + \kappa\left(\frac{I_d}{N_d} + \frac{I_h}{N_h}\right)(1 - A) - \mu_A A,$$

we observe that at $A = 0$,

$$\frac{dA}{dt} = \theta + \kappa\left(\frac{I_d}{N_d} + \frac{I_h}{N_h}\right) \ \ge\ 0, \qquad \text{since } \theta, \kappa \ge 0,$$

and at $A = 1$,

$$\frac{dA}{dt} = -\mu_A \ <\ 0, \qquad \text{since } \mu_A > 0.$$

Thus, the vector field points inward (or is tangent) on both boundaries of $[0,1]$, so $A(t)$ cannot leave $[0,1]$ once it starts there; hence

$$0 \le A(t) \le 1 \qquad \text{for all } t \ge 0.$$

Therefore, the solution of system (1)– (9) is positive and bounded in the region

$$\Omega = \left\{(S_d, V_d, E_d, I_d, S_h, E_h, V_h, I_h, A) \in \mathbb{R}_+^8 \times [0,1] :\ 0 \le N_d \le \frac{\Lambda_d}{\mu_d},\ \ 0 \le N_h \le \frac{\Lambda_h}{\mu_h},\ \ 0 \le A \le 1\right\},$$

where $N_d = S_d + V_d + E_d + I_d$ and $N_h = S_h + E_h + V_h + I_h$. Thus, the model system (1)–(9) is biologically meaningful and well posed, and Ω is positively invariant with respect to system (1)–(9). Therefore, we can consider the flow generated by this model system for further analysis. □

3.1.3 Disease-free equilibrium

At the disease-free equilibrium (DFE), $E_d = I_d = E_h = I_h = 0$. Setting $I_d = I_h = 0$ in the awareness equation gives the awareness level sustained by campaign investment alone,

$$A^* = \frac{\theta}{\theta + \mu_A}.$$

Writing $\phi_d^* = \phi_{d0} + \phi_{d1} A^*$, the dog equations at the DFE yield

$$\Lambda_d - \phi_d^* S_d^* + \omega_d V_d^* - \mu_d S_d^* = 0, \qquad \phi_d^* S_d^* - \omega_d V_d^* - \mu_d V_d^* = 0,$$

so that the disease-free susceptible fraction of dogs is

$$\frac{S_d^*}{N_d^*} = \frac{\omega_d + \mu_d}{\phi_d^* + \omega_d + \mu_d}, \qquad N_d^* = \frac{\Lambda_d}{\mu_d}.$$

On the human side, since no exposure occurs at the DFE, $S_h^* = \Lambda_h / \mu_h$, and $V_h^* = E_h^* = I_h^* = 0$. Thus, the DFE is

$$E_0 = (S_d^*,\ V_d^*,\ 0,\ 0,\ S_h^*,\ 0,\ 0,\ 0,\ A^*).$$

This susceptible-fraction expression already reveals the central qualitative mechanism of the paper: any behavioural mechanism that raises ϕ_d^* (through higher campaign investment θ or a stronger awareness–vaccination link ϕ_{d1}) strictly lowers the disease-free susceptible fraction of dogs, and — as shown next — lowers R_0 proportionally.

3.1.4 Basic reproduction number

Because humans do not transmit rabies onward (to other humans or to dogs), only the dog exposed and infectious classes, (E_d, I_d), constitute the infection subsystem relevant to R_0; the human classes are a one-way "spillover" that does not re-enter the next-generation calculation. Linearizing the new-infection and transition vectors at the DFE gives the new-infections matrix F and the transition matrix V:

$$F = \begin{pmatrix} 0 & \beta_d S_d^*/N_d^* \\ 0 & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} \sigma_d + \mu_d & 0 \\ -\sigma_d & \delta_d + \mu_d \end{pmatrix}.$$

Since V is lower triangular, $V^{-1} = \frac{1}{(\sigma_d+\mu_d)(\delta_d+\mu_d)}\begin{pmatrix} \delta_d + \mu_d & 0 \\ \sigma_d & \sigma_d + \mu_d \end{pmatrix}$, and the next-generation matrix $K = FV^{-1}$ has spectral radius equal to its $(1,1)$ entry (its second row is identically zero), giving the closed-form basic reproduction number

$$R_0 = \beta_d \cdot \frac{\sigma_d}{\sigma_d + \mu_d} \cdot \frac{1}{\delta_d + \mu_d} \cdot \frac{S_d^*}{N_d^*} = \frac{\beta_d\, \sigma_d\, (\omega_d + \mu_d)}{(\sigma_d + \mu_d)(\delta_d + \mu_d)(\phi_d^* + \omega_d + \mu_d)}.$$

The three factors in R_0 have direct epidemiological readings: β_d is the effective dog-to-dog transmission rate; $\sigma_d/(\sigma_d + \mu_d)$ is the probability that an exposed dog survives the incubation period to become infectious; $1/(\delta_d + \mu_d)$ is the mean infectious period of a rabid dog; and $S_d^*/N_d^* \in (0,1)$ is the disease-free susceptible fraction, which is driven down by behaviourally-mediated vaccination coverage. Setting $\phi_{d1} = 0,\ \theta = 0$ recovers the reproduction number of a model with only routine (non-behavioural) vaccination, confirming that formula (10) generalises the classical rabies R_0.

Theorem 3. *The disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof sketch. By the general result of van den Driessche and Watmough (2002, Theorem 2), for a compartmental disease model satisfying the standard assumptions (A1)–(A5) — which the present model satisfies, since $F \geq 0$, V is a non-singular M-matrix, and the disease-free subsystem $E_d = I_d = 0$ is locally asymptotically stable for the remaining variables — the Jacobian of the full system at E_0, restricted to the infected subspace (E_d, I_d), is $F - V$, and all its eigenvalues have negative real part if and only if the spectral radius of FV^{-1} is less than one. Since $\rho(FV^{-1}) = R_0$ by (10), it follows that E_0 is locally asymptotically stable whenever $R_0 < 1$. Conversely, when $R_0 > 1$, $F - V$ has an eigenvalue with positive real part, so E_0 is unstable. The remaining eigenvalues of the full Jacobian at E_0 (associated with S_d, V_d, S_h, A and the disease-free human classes) are $-\mu_d$, $-(\omega_d + \mu_d + \phi_d^*)$, $-\mu_h$ and $-(\theta + \mu_A)$, all strictly negative, so local stability of the full system is governed entirely by the sign of $\rho(FV^{-1}) - 1 = R_0 - 1$. □

Remark 1. *Because I_h does not feed back into any other equation, human rabies incidence is, to leading order, a linear functional of the dog outbreak trajectory; suppressing R_0 below unity in the dog population is both necessary and sufficient to eliminate the human health burden at source, which is precisely the epidemiological logic underlying the "vaccinate dogs to protect humans" strategy endorsed by WHO (2018). This is verified numerically in Section 5.*

4.1 Sensitivity Analysis

To identify which parameters most strongly influence transmission potential, and hence which levers public-health policy should prioritise, we compute the normalized forward sensitivity index of R_0 with respect to each parameter p,

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \cdot \frac{p}{R_0}.$$

Differentiating the closed-form R_0 in (10) analytically yields closed forms for several parameters,

$$\Upsilon_{\beta_d}^{R_0} = 1, \qquad \Upsilon_{\delta_d}^{R_0} = -\frac{\delta_d}{\delta_d + \mu_d}, \qquad \Upsilon_{\sigma_d}^{R_0} = \frac{\mu_d}{\sigma_d + \mu_d}, \qquad \Upsilon_{\phi_{d1}}^{R_0} = -\frac{\phi_{d1} A^*}{\phi_d^* + \omega_d + \mu_d},$$

with the remaining indices (for $\mu_d, \omega_d, \theta, \mu_A$) obtained in closed form by the chain rule through $A^* = \theta/(\theta + \mu_A)$ and evaluated numerically at the baseline parameter set of Table 1 (Table 2, Figure 2).

Table 2: Normalized forward sensitivity indices of R_0 at baseline parameter values.

Parameter	Index $\Upsilon_p^{R_0}$	Interpretation
β_d	$+1.000$	1% increase in transmission ⇒ 1% increase in R_0
δ_d	-0.995	Faster disease death in dogs strongly lowers R_0
ϕ_{d1}	-0.546	Awareness–vaccination linkage strongly lowers R_0
μ_d	$+0.300$	Higher natural dog turnover raises R_0
ω_d	$+0.273$	Faster vaccine waning raises R_0
θ	-0.182	Campaign investment lowers R_0
μ_A	$+0.182$	Faster forgetting of awareness raises R_0
ϕ_{d0}	-0.055	Routine vaccination has a small marginal effect
σ_d	$+0.023$	Incubation-period length has minimal effect

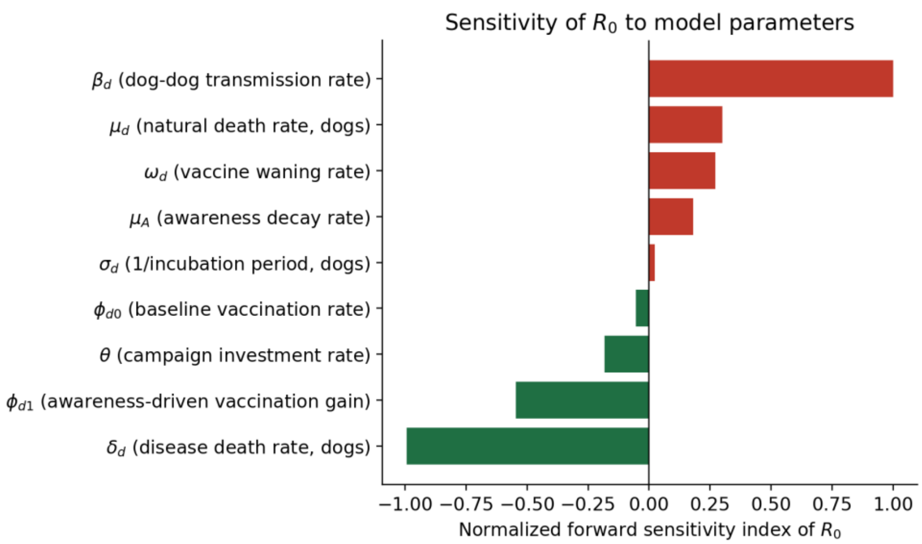

Figure 2: Normalized forward sensitivity indices of R_0. Bars extending right (red) indicate parameters that increase R_0; bars extending left (green) indicate parameters that decrease R_0.

Three findings stand out. First, as in classical rabies models, the dog-to-dog transmission rate β_d and the disease-induced death rate δ_d dominate R_0 by construction (a proportional, elasticity-one and near-unity relationship respectively); these are intrinsic viral/behavioural-contact parameters largely outside direct policy control. Second, and central to this paper, the behavioural parameters are collectively influential: ϕ_{d1} (the sensitivity of dog vaccination uptake to awareness) has the third-largest index in magnitude, exceedingly even the natural mortality and vaccine-waning rates, and θ (campaign investment) and μ_A (forgetting) form an equal-and-opposite pair confirming that sustaining — not merely launching — an awareness campaign is what matters epidemiologically. Third, routine (non-behavioural) vaccination ϕ_{d0} has a comparatively small marginal effect at baseline coverage, implying that incremental gains from routine, campaign-independent vaccination alone are limited unless coupled to awareness-responsiveness.

Figure 3 shows that this result holds up in practice, not just in theory: as we increase awareness-driven vaccination (ϕ_{d1}) and campaign investment (θ) across realistic values, R_0 drops below 1. This means that behavioural interventions alone can be enough to eliminate the disease. This qualitative pattern — vaccination coverage dominating projected elimination timelines relative to improvements in PEP access alone — mirrors recent national-scale decision-analytic rabies modelling, such as the STRATEGIC framework applied in China, in which scaling up dog vaccination to 70% coverage was found to be substantially more effective at averting deaths than expanding PEP access alone (Chen et al., 2023).

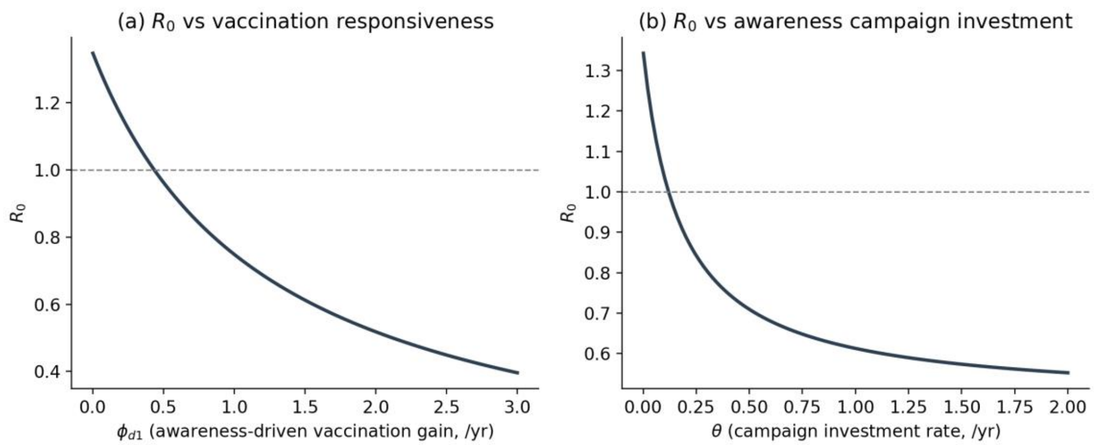

Figure 3: R_0 as a function of (a) the awareness-driven vaccination gain ϕ_{d1} and (b) the campaign investment rate θ, holding all other parameters at baseline. The dashed line marks the critical threshold $R_0 = 1$.

4.1 Numerical Simulation and Interpretation

The governing system was solved numerically (implicit Radau method, absolute/relative tolerance $10^{-10}/10^{-8}$) over a ten-year horizon from initial condition $(S_d, V_d, E_d, I_d) = (4800,200,0,20)$, $(S_h, E_h, V_h, I_h) = (9700,0,0,0)$, $A(0) = 0.02$, representing a small town with a nascent rabies outbreak in the dog population and negligible baseline awareness. Two scenarios are contrasted using the parameters of Table 1:

4.1.1 Without behavioural intervention: low, static campaign investment ($\theta = 0.02$), no awareness-driven vaccination gain ($\phi_{d1} = 0$), no awareness-driven PEP-uptake gain ($\tau_{h1} = 0$), and no bite-avoidance behaviour ($\varepsilon = 0$). This reproduces a conventional model in which vaccination and care-seeking are fixed at their routine baseline levels.

4.1.2 With behavioural intervention: sustained high campaign investment ($\theta = 1.0$), strong awareness–vaccination linkage ($\phi_{d1} = 1.5$), strong awareness–PEP linkage ($\tau_{h1} = 15$), and meaningful bite-avoidance behaviour ($\varepsilon = 0.6$).

The corresponding reproduction numbers, computed from (10), are $R_0 = 1.348$ (without intervention) and $R_0 = 0.612$ (with intervention) — a threshold crossing from an endemic-capable regime to an elimination-capable regime driven entirely by the behavioural parameters.

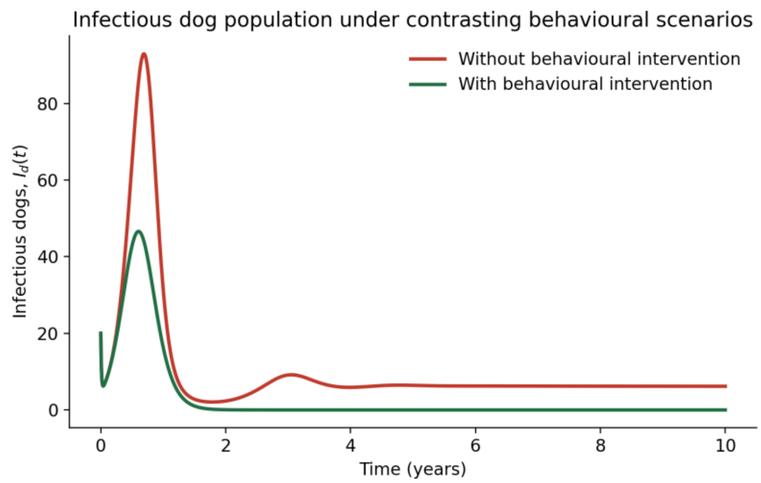

Figure 4: Infectious dog population $I_d(t)$ under the two behavioural scenarios.

Figure 4 shows that without behavioural intervention the infectious dog population grows to a peak of approximately 93 infectious dogs before settling toward an endemic plateau around 6 dogs — consistent with $R_0 > 1$ and persistent, low-level circulation. With behavioural intervention, awareness rises quickly enough (Figure 6a) to push the effective vaccination coverage of the dog population above 45% (Figure 6b) well before the outbreak matures; the infectious dog peak is roughly halved (to about 47) and, critically, the trajectory declines to numerical zero by the end of the simulation window, consistent with $R_0 < 1$ and eventual local elimination.

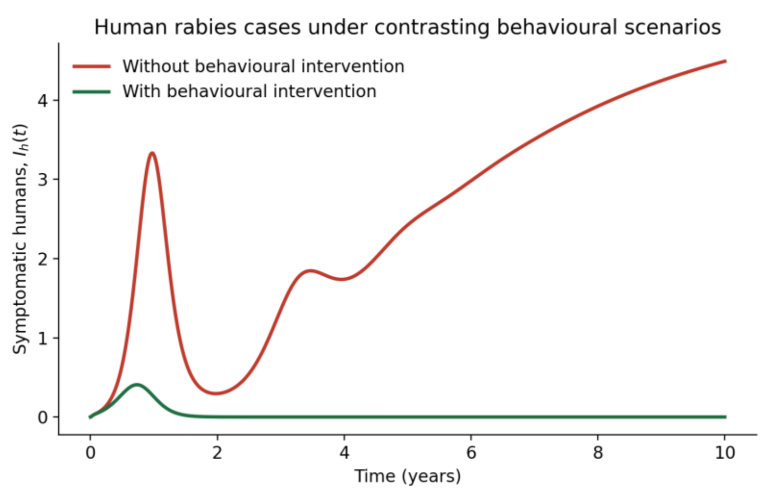

Figure 5: Symptomatic (untreated) human rabies cases $I_h(t)$ under the two behavioural scenarios.

The human health impact is the more policy-relevant outcome: the peak symptomatic human caseload falls from approximately 4.5 to approximately 0.4 (a reduction of over 90%) when behavioural intervention is active. This reduction operates through two reinforcing channels captured explicitly in the model: fewer infectious dogs lower the force of infection on humans directly, and, independently, a higher awareness level accelerates PEP-uptake ($\tau_h(A)$), diverting a larger share of already-exposed individuals from E_h into the protected class V_h before symptom onset — a mechanism absent from models that hold treatment-seeking behaviour fixed.

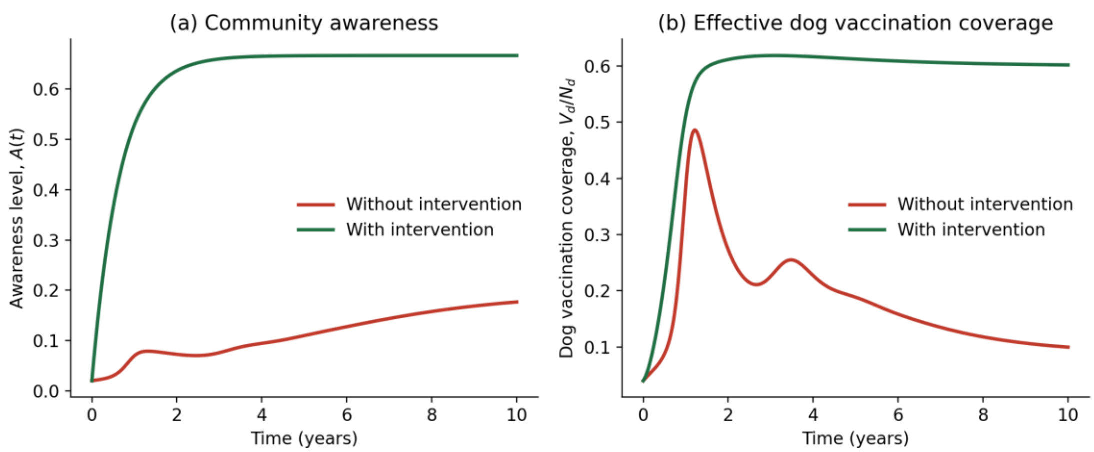

Figure 6: (a) Community awareness $A(t)$ and (b) effective dog vaccination coverage V_d/N_d under the two scenarios.

Figure 6 illustrates the mechanism directly: with low campaign investment, awareness stagnates near its case-driven floor ($A^* \approx 0.04$) because sporadic cases are not, by themselves, sufficient to sustain public attention; with sustained campaign investment, awareness climbs toward $A^* \approx 0.67$ and effective vaccination coverage rises correspondingly, well past the coverage threshold generally considered necessary to interrupt canine rabies transmission in the field. This modelled awareness-coverage coupling is consistent with recent participatory field research in Thailand directly linking measured community rabies awareness to observed dog vaccination coverage (Thichumpa et al., 2024).

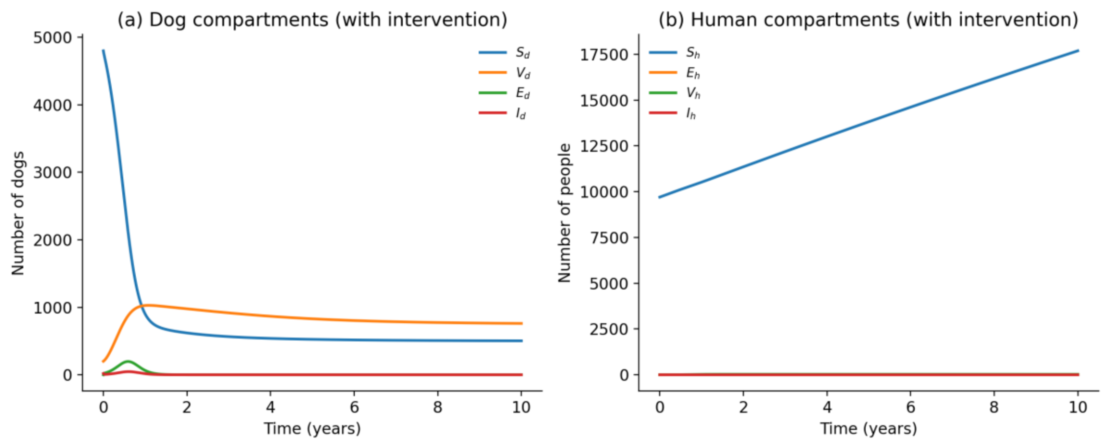

Figure 7: Full trajectories of all dog compartments (a) and all human compartments (b) under the behavioural-intervention scenario.

Figure 7 gives the complete compartmental picture under active intervention: the vaccinated dog class V_d overtakes the susceptible class S_d within the first two years, exposed and infectious dog classes remain small and decaying throughout, and on the human side the protected class V_h (successful PEP) absorbs almost all human exposures, keeping the symptomatic class I_h negligible throughout the simulated horizon.

5. Conclusion and Recommendations

5.1.1 Conclusion

We formulated and analysed a nine-dimensional compartmental model of dog-mediated rabies transmission in which dog vaccination uptake, human PEP-uptake, and human bite-avoidance behaviour are all endogenously driven by a time-evolving community awareness variable, itself shaped by campaign investment and by feedback from observed disease prevalence. We derived a closed-form basic reproduction number that isolates the disease-free susceptible fraction of dogs as the behaviourally-modifiable term, proved local stability of the disease-free equilibrium for $R_0 < 1$, and showed via normalized forward sensitivity analysis that behavioural parameters — particularly the strength of the awareness–vaccination linkage and the level of sustained campaign investment — rank alongside intrinsic epidemiological parameters in their influence on transmission potential. Numerical simulation demonstrated that a realistic behavioural-intervention scenario can move R_0 from an endemic-capable value above unity to an elimination-capable value below unity, roughly halving the peak infectious dog population and reducing peak human rabies morbidity by over 90% relative to a low-awareness baseline. These results support the qualitative and quantitative case that behavioural-communication investment is not a peripheral add-on to dog-mediated rabies control but a primary, model-identifiable lever comparable in importance to the biomedical interventions it activates.

These qualitative conclusions align with recent field evidence from dog-mediated rabies programmes. The elimination-capable vaccination coverage the model attributes to sustained awareness investment is consistent with trial evidence that continuous, community-based dog vaccination delivery in Tanzania sustains coverage above the herd-immunity threshold more reliably than conventional annual campaigns (Lankester et al., 2024), and with evidence that community engagement itself raises owner participation in vaccination clinics (Duamor et al., 2022). Likewise, the model's finding that awareness-driven PEP-uptake is a major independent contributor to averted human deaths mirrors empirical findings that cost, distance, and awareness gaps remain leading correlates of incomplete or delayed PEP-seeking in Tanzania and neighbouring East African countries (Walwa et al., 2024; Kisaka et al., 2022). Taken together, the model and the empirical literature point to the same policy conclusion: behavioural-communication investment and delivery-system design are not peripheral to dog-mediated rabies elimination but are among its primary, measurable levers (Zha et al., 2025). These simulated gains have real-world analogues: Bangladesh's national programme has recorded a measurable decline in predicted human rabies deaths consistent with the Zero-by-30 trajectory as mass dog vaccination has scaled up (Ghosh et al., 2024), while a recent multi-site field evaluation in urban and peri-urban Bangladesh confirms that reliably reaching and verifying the 70% coverage threshold remains an operational bottleneck even under dedicated, well-resourced vaccination strategies (Swedberg et al., 2026) — underscoring that the behavioural and delivery-system levers highlighted by this model are also the ones field programmes currently find hardest to pull.

5.1.2 Recommendations

Based on the model analysis, we recommend the following for national and sub-national rabies elimination programmes:

1. Treat awareness campaigns as a core, continuously-funded intervention, not a one-off launch activity: the sensitivity analysis shows that the campaign decay rate μ_A is as influential as the investment rate θ itself, so programmes that under-invest in maintenance will see awareness — and the coverage it drives — decay back toward baseline.

2. Co-design vaccination and communication campaigns, since the model shows the awareness–vaccination linkage ϕ_{d1} to be substantially more influential than routine, campaign-independent vaccination capacity ϕ_{d0}; door-to-door or event-based vaccination drives should be paired explicitly with local risk-communication messaging rather than run as parallel, uncoordinated activities.

3. Shorten the time-to-PEP pathway by combining awareness messaging with practical removal of access barriers (cost, distance, stock-outs), since the human health benefit in the model operates strongly through the awareness-driven PEP-uptake rate $\tau_h(A)$, independent of the dog-side intervention.

4. Prioritize surveillance-linked messaging, since the model's case-driven awareness term κ shows that visible, well-publicised case detection and response can itself sustain community awareness between campaign pulses, complementing scheduled communication investment.

5. Monitor R_0, not only case counts, as an operational indicator: because the model shows R_0 crossing unity as awareness and coverage rise, programme managers can use the closed-form expression for R_0, populated with locally-estimated parameters, as a real-time benchmark for whether current vaccination and behavioural-coverage levels are sufficient for elimination.

6.1 Limitations and future research

The model uses illustrative baseline parameters informed by literature ranges rather than a single-site calibration exercise; future work should fit the model to longitudinal surveillance and campaign-coverage data from an active rabies elimination programme to obtain locality-specific estimates of ϕ_{d1}, θ, κ and μ_A. Spatial heterogeneity (rural versus urban dog density, cross-jurisdictional dog movement) and stochastic effects at low case numbers, both known to matter for near-elimination dynamics, are natural extensions of the deterministic framework presented here.

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