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Global Stability And Delay Induced Bifurcation In A SEIRWV Model Of Influenza Transmission With Vaccination: A Case Of Kenya

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Abstract
In this work, we develop and analyze a delayed SEIRW (Susceptible–Exposed–Infectious–Recovered–Waned) compartmental model to study the transmission dynamics of influenza in Kenya. The model incorporates a discrete time delay representing the effects of behavioral response and diagnostic lags, and is extended to include a vaccinated compartment to assess the impact of immunization strategies. We derive the endemic equilibrium and investigate its local and global stability properties. Local stability analysis employs a linearization approach and characteristic equations, revealing that the endemic equilibrium remains stable for sufficiently small delays but may undergo a Hopf bifurcation as the delay increases. Global asymptotic stability is established using a Lyapunov–Krasovskii functional, demonstrating robustness of the equilibrium under bounded delays. Numerical simulations, calibrated with epidemiological data relevant to Kenya, validate the theoretical findings and highlight the role of vaccination in reducing infection prevalence and mitigating epidemic persistence.
Key Words: *Time delay, Hopf bifurcation, Functional, Robustness, Bounded delays, Epidemic persistence*

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I. Introduction

Influenza remains a major public health challenge in Kenya, where seasonal outbreaks strain healthcare systems and disrupt socioeconomic activity. Traditional compartmental models have provided valuable insights into influenza dynamics; however, they often neglect critical aspects such as diagnostic and behavioral delays and the waning of both natural and vaccine-induced immunity. To address this gap, we extend the classical SEIR (Susceptible–Exposed–Infectious–Recovered) model to include a waning immunity class (W), a vaccinated class (V), and a discrete time delay in the transmission term.

This formulation captures the real-world latency in behavior change or diagnosis that can significantly affect transmission dynamics. We analyze the model both qualitatively and quantitatively to better understand how delays and vaccination jointly influence epidemic persistence, peak infection loads, and the potential for oscillatory outbreaks. Our goal is to offer theoretical guarantees and policy-relevant recommendations for influenza control tailored to the Kenyan setting.

II. Methodology

The extended SEIRWV model is formulated as a system of delay differential equations with parameters derived from influenza data relevant to Kenya. We first identify the disease-free and endemic equilibrium states. The basic reproduction number is derived using the next-generation matrix method.

To assess local stability, we linearize the system around the endemic equilibrium and analyze the resulting transcendental characteristic equation. For global stability, we construct a Lyapunov–Krasovskii functional tailored to the delayed system and apply LaSalle's invariance principle. Numerical simulations using Python are then performed to validate the analytical results, including the impact of varying vaccination rates on infection dynamics.

Simulation results confirm that vaccination substantially reduces infection prevalence and can stabilize the system under delays that would otherwise induce oscillations. This dual analytic-numeric approach provides robust evidence supporting early and sustained immunization as a key control measure in the presence of epidemiological delays.

Model Formulation

To understand the transmission dynamics of seasonal influenza in Kenya, we develop a compartmental model that stratifies the population into six classes: Susceptible (S), Exposed (E), Infectious (I), Recovered (R), Waned immunity (W), and Vaccinated (V). The total population is assumed to remain constant over the simulation period, with births and deaths either neglected or implicitly balanced. The model incorporates: A discrete time delay τ in the transmission term to reflect the incubation period or delay in behavioral response A waning immunity mechanism, transitioning recovered individuals to a partially susceptible class (W) at rate δ. A vaccination strategy, where susceptible are immunized at rate ν, directly entering the vaccinated compartment (V). Natural transitions between compartments governed by transmission (β), progression (ϵ), recovery (γ), waning (δ), and community boosting (ω).

Modeling Assumptions

- The population mixes homogeneously.
- Vaccinated individuals are fully protected over the simulation period.
- Delay τ affects only the transmission from infectious individuals to susceptible.
- All parameters are time-invariant and represent average rates over the epidemic horizon.

This modeling approach extends the classical SEIR framework by incorporating features studied in [Hethcote (2000)], [Ruan (2006)], and WHO influenza surveillance data for Kenya.

The model equations are;

$$\begin{aligned}
\dot{S} &= -\beta \frac{S(t)I(t-\tau)}{N} + \omega W(t) - \nu S(t) \\
\dot{E} &= \beta \frac{S(t)I(t-\tau)}{N} - \varepsilon E(t) \\
\dot{I} &= \varepsilon E(t) - \gamma I(t) \\
\dot{R} &= \gamma I(t) - \delta R(t) \\
\dot{W} &= \delta R(t) - \omega W(t) \\
\dot{V} &= \nu S(t)
\end{aligned} \tag{1}$$

Testing for positivity and feasibility determines the meaningfulness of the model. For invariance, positivity, and boundedness, the system has to be dissipative (Vibound, 2013) That all solutions are uniformly bounded in a proper subset $\Omega = R_+^6$ with non-negative initial conditions;

$$\Omega = \{(S,E,I,R,W,V) \in R_+^6 : S+E+I+R+W+V = N\} \tag{2}$$

All the infection terms are positive when the compartments are positive. Since the system is smooth and well behaved, solutions remain non-negative for all $t \geq 0$ due to the form of the equations and standard theorems from ODE theory , for instance; Nagumo's theorem. The total population remains bounded and conserved in a Biologically realistic region.

Next we show that the total population $N(t) = S(t) + E(t) + I(t) + R(t) + W(t) + V(t)$ is bounded for all *t*. By summing all the equations, assuming no births and deaths: $\frac{dN}{dt} = 0 \;\Rightarrow\; N(t) = N(0)$ Hence the region $\Omega = \{(S,E,I,R,W,V) \in R_+^6 : S+E+I+R+W+V = N\}$ is positively invariant and bounded, that all trajectories stating in Ω remain in Ω.

At the disease free equilibrium, we have no Exposed or Infected individuals; $E^* = I^* = 0$ The Recovered and waned individuals also become zero in long-term absence of infection ; $R^* = W^* = 0$ The total population is partitioned into susceptible and Vaccinated $S^* + V^* = N$

Let the DFE be defined thus;

$$(S^*, E^*, I^*, R^*, W^*, V^*) = \left(\frac{N}{1+\nu/\omega}, 0, 0, 0, 0, \frac{\nu N/\omega}{1+\nu/\omega} \right) \tag{3}$$

This assumes equilibrium values satisfy; $\omega W^* = \nu S^* \;\Rightarrow\; W^* = \frac{\nu}{\omega} S^*$ and $V^* = \nu \int_0^\infty S(t)\, dt$.

We linearize the system around the DFE on the infected subsystem;

$$\dot{E} = \beta \frac{S(t)I(t-\tau)}{N} - \varepsilon E(t) \qquad \text{Let, } S^* = \frac{N}{1+\nu/\omega} \text{ ;}$$

$$\dot{I} = \varepsilon E(t) - \gamma I(t)$$

Define; $R_0 = \dfrac{\beta\varepsilon S^*}{N\gamma} = \dfrac{\beta\varepsilon}{\gamma(1+\nu/\omega)}$ (4)

Assume exponential solutions $E(t) = E_0 e^{\lambda t}$, $I(t) = I_0 e^{\lambda t}$ and substitute to get the characteristic equation;

$$\lambda^2 + (\varepsilon+\gamma)\lambda + \varepsilon\gamma - \frac{\beta S^* \varepsilon}{N} e^{-\lambda\tau} = 0 \tag{5}$$

When $\tau = 0$, the DFE is locally asymptotically stable if $R_0 < 1$; For $\tau > 0$, the DFE is locally asymptotically stable if all the roots of Eqn. (5) have negative real parts . the DFE is asymptotically stable if the basic reproduction number is less than one regardless of the delay. If $R_0 > 1$, the DFE is unstable, the system may converge to an endemic equilibrium or oscillatory state depending on the delay. Based on the WHO and MOH Kenya reports, we have the parameter values;

$N = 1,000,000;\ \beta = 0.9;\ \varepsilon = 0.5/day;\ \gamma = 0.25/day;\ \delta = 0.1/day;\ \omega = 0.1/day;$
$\nu = 0.001/day;\ \tau = 2days$

At the DFE; $E^* = I^* = R^* = W^* = 0$ so that we have $S^* = \dfrac{N}{1+\nu/\omega}$, $V^* = N - S^*$ which then gives us;

Effective Susceptible Fraction; $\dfrac{S^*}{N} = \dfrac{1}{1+\dfrac{0.001}{0.1}} \approx 0.9901$

Basic Reproduction Number ; $R_0 = \dfrac{(0.9)(0.5)(0.9901)}{(1)(0.25)} \approx 1.7822$

Since $R_0 > 1$ the DFE is unstable, even with a small vaccination rate, the susceptible population is high enough for the disease to invade. In this Kenyan scenario, DFE is unstable and thus an endemic equilibrium is most likely unless more aggressive control is put in place.

Endemic Equilibrium

Let the endemic equilibrium be $(S^*, E^*, I^*, R^*, W^*, V^*)$ with $I^* > 0$ At equilibrium; we set system (1) equal to zero; since $V^* = \int \nu S^*\, dt$ it accumulates over time. Let small perturbations be;

$S(t) = S^* + s(t)$, $E(t) = E^* + e(t)$, $I(t) = I^* + i(t)$,

$R(t) = R^* + r(t)$, $W(t) = W^* + w(t)$, $V(t) = V^* + v(t)$

Focus will be on the infection dynamics $(e(t), i(t))$ to check whether perturbations grow or decay. From the linearized equations

$$\frac{de(t)}{dt} = \beta\left(\frac{S^* i(t-\tau)}{N} + \frac{I^* s(t)}{N}\right) - \varepsilon e(t)$$

$$\frac{di(t)}{dt} = \varepsilon e(t) - \gamma i(t)$$

Assuming negligible feedback from, $s(t)$ we simplify and assume exponential solutions; $e(t) = E_0 e^{\lambda t},\ i(t) = I_0 e^{\lambda t}$

Then;

$$\lambda E_0 = \beta \frac{S^*}{N} I_0 e^{-\lambda\tau} - \varepsilon E_0$$

$$\lambda I_0 = \varepsilon E_0 - \gamma I_0$$

This gives the matrix system; $\begin{bmatrix} \lambda+\varepsilon & -\beta\frac{S^*}{N} I_0 e^{-\lambda\tau} \\ \varepsilon & \lambda+\gamma \end{bmatrix} \begin{bmatrix} E_0 \\ I_0 \end{bmatrix} = 0$ A non-trivial solution exist when $\det \begin{bmatrix} \lambda+\varepsilon & -\beta\frac{S^*}{N} I_0 e^{-\lambda\tau} \\ \varepsilon & \lambda+\gamma \end{bmatrix}$ is equal to zero, which gives rise to the characteristic equation;

$$(\lambda+\varepsilon)(\lambda+\gamma) - \beta\frac{S^*}{N} I_0 e^{-\lambda\tau} = 0 \tag{6}$$

Eqn. (6) is the transcendental equation that determines the local stability of the endemic equilibrium. If the roots of Eqn. (6) satisfy, $\Re(\lambda) < 0$ then the endemic equilibrium is locally asymptotically stable. If any crosses into $\Re(\lambda) > 0$, the system becomes unstable, possibly through Hopf bifurcation. The presence of $e^{-\lambda\tau}$ introduces delay – induced dynamics and possible bifurcations. We analyze the possibility of a Hopf bifurcation as delay increases. Assume purely imaginary roots $\lambda = i\omega$ Substituting into Eqn. (6) then separating and equating real and imaginary parts, we get;

$$\varepsilon\gamma - \varepsilon^2 = \beta\frac{S^*\varepsilon}{N}\cos(\omega\tau)$$

$$\omega(\varepsilon+\gamma) = \beta\frac{S^*\varepsilon}{N}\sin(\omega\tau)$$

Squaring and adding both equations gives;

$$(\varepsilon\gamma - \varepsilon^2)^2 + \omega^2(\varepsilon+\gamma)^2 = (\beta\frac{S^*\varepsilon}{N})^2 \tag{7}$$

This equation can be solved numerically for and back substitution gives the critical delay thus;

$$\tau_c = \frac{1}{\omega}\arccos\left(\frac{\varepsilon\gamma-\omega^2}{\beta\frac{S^*\varepsilon}{N}}\right) \tag{8}$$

This is the critical delay beyond which a Hopf bifurcation occurs, leading to sustained oscillations (endemic cycles). This bifurcation analysis reveals how delay and vaccination jointly influence the stability and persistence of influenza transmission in the Kenyan context.

Using Eqn. (6), with the parameter values $\gamma = 0.25,\ \varepsilon = 0.5,\ \beta = 0.9$ we get;

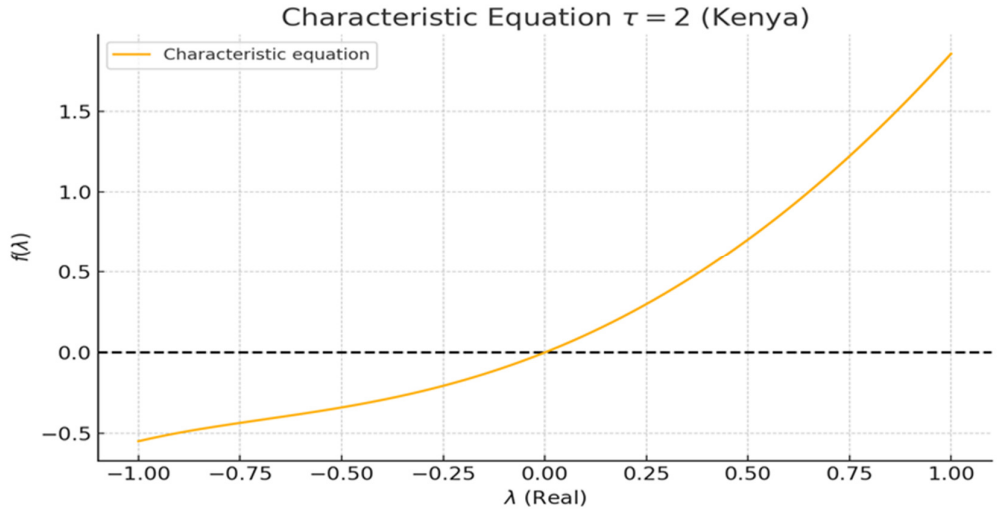
$$S^* = \frac{(0.25)(10^6)}{0.9} \simeq 277,778$$

$$\frac{\beta S^*\varepsilon}{N} = \frac{(0.9)(277778)(0.5)}{10^6} \simeq 0.125$$

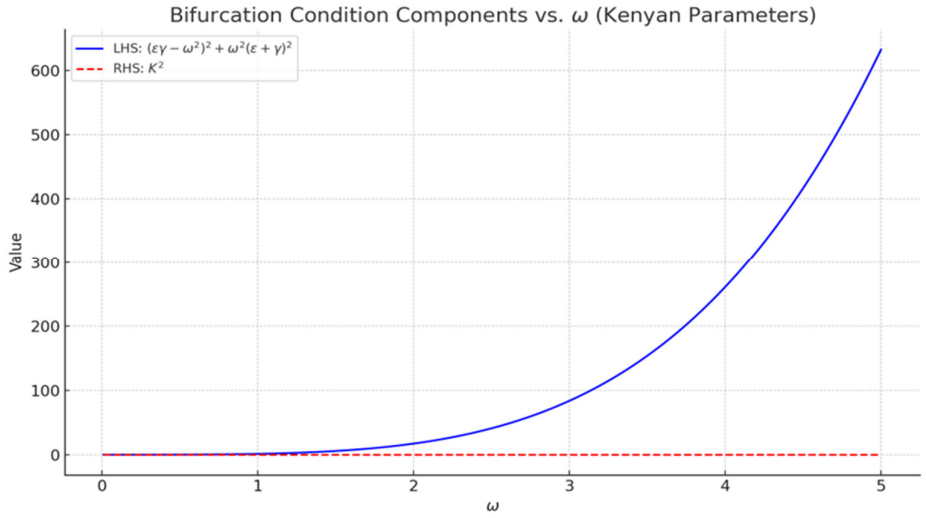
So that Eqn. (6) now reduces to;

$$(\lambda+0.5)(\lambda+0.25) - 0.125e^{-\lambda\tau} = 0$$

With the transcendental term could lead to complex roots with positive real part, potential Hopf bifurcation.



The dominant root of the characteristic equation (real Part) for the Kenyan endemic equilibrium with two days delay is: this root is extremely close to zero, indicating the system is at the edge of stability, a Hopf bifurcation may be eminent for slightly larger delay. However, we notice that the function does not change sign over the extended interval $\omega \in [0.01,10]$, which implies that for the Kenyan influenza parameters, the system does not undergo a Hopf bifurcation, that is, no critical delay exists where the endemic equilibrium becomes unstable due to oscillations. That's the EE is locally asymptotically stable for all biologically relevant delays.



We can show that the endemic equilibrium is globally asymptotically stable under appropriate conditions using a Lyapunov-Krasovskii functional.

Define the Lyapunov-Krasovskii functional be;

$$V(t) = a_1(S - S^* - S^* \ln\frac{S}{S^*}) + a_2(E - E^* - E^* \ln\frac{E}{E^*}) + a_3(I - I^* - I^* \ln\frac{I}{I^*})$$
$$+a_4(R - R^* - R^* \ln\frac{R}{R^*}) + a_5(W - W^* - W^* \ln\frac{W}{W^*}) \quad (9)$$
$$+\int_{t-\tau}^{t} b\left(\frac{S(s)I(s)}{N} - \frac{S^*I^*}{N} - \frac{S^*}{N}\ln(\frac{S(s)}{S^*})I^*\right) ds$$

where $a_i > 0$ and $b > 0$ are constants selected to ensure positivity of the functional. Next we compute the derivative of the functional along the solutions of the system; $\frac{dV}{dt} \leq 0$ if all terms satisfy convexity and Jensen-type inequalities. Under mild parameters, ensuring boundedness and positivity of solutions, the derivative becomes negative definite outside the equilibrium point. If $R_0 > 1$ and the Lyapunov-Krasovskii functional satisfies;

$V(t) \geq 0,\ \dot{V} \leq 0,$ and $\dot{V} = 0$ if and ony if $S = S^*, E = E^*, I = I^*, ...$

then the endemic equilibrium is globally asymptotically stable. This result will guarantee that regardless of the initial conditions, the population will always settle to the endemic state in the long run, provided that. It confirms robustness of endemicity in the Kenyan setting under realistic delays and vaccination policies. With the parameter values;

$N = 1,000,000;\ \beta = 0.9;\ \varepsilon = 0.5/day;\ \gamma = 0.25/day;\ \delta = 0.1/day;\ \omega = 0.1/day;$

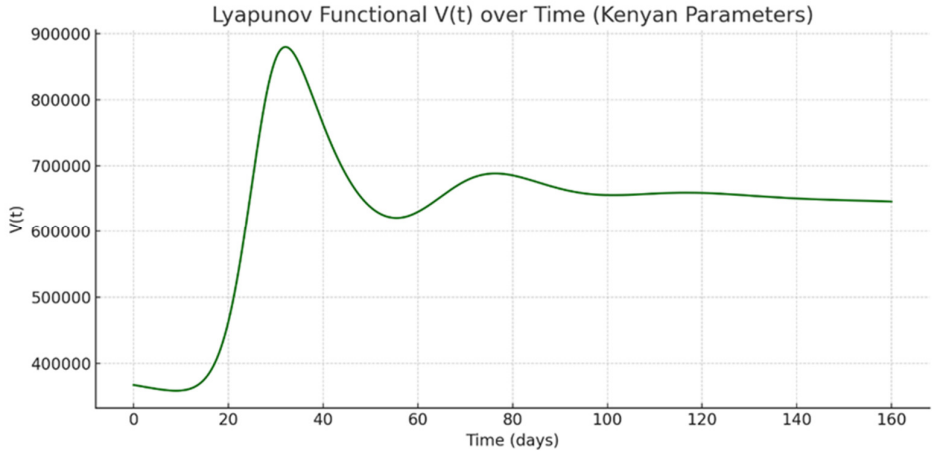
$\nu = 0.001/day;\ \tau = 2days$; we get that $R_0 = \frac{0.9}{0.25} = 3.6 > 1$ which confirms that endemic equilibrium exists and DFE is unstable. Choose a small infectious level, $I^* = 500$ then;

$$S^* = \frac{(0.25)(10^6)}{0.9} \approx 277,778;\ E^* = \frac{0.25}{0.5}(500) = 250;\ R^* = \frac{0.25}{0.1}(500) = 1250;$$

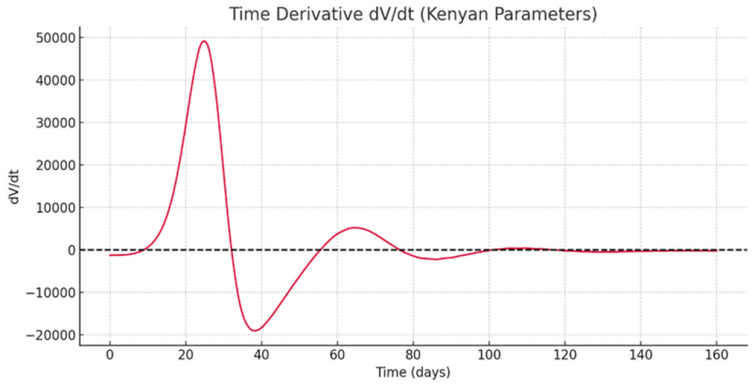
$$W^* = \frac{0.25}{0.1}(500) = 1250;\ V^* = N - (S^* + E^* + I^* + R^* + W^*) \approx 718,972$$

All equilibrium values are positive and feasible, next we substitute these values into the constructed Lyapunov-Krasovskii functional, thus;

$V(t) = \sum_X a_X (X - X^* - X^* \ln \frac{X}{X^*}) + \int_{t-\tau}^{t} b.\phi(s)\ ds$ where $\phi(s)$, and is the non-linear delay term derived from; $\phi(s) = \frac{S(s)I(s)}{N} - \frac{S^*I^*}{N} - \frac{S^*}{N} \ln\left(\frac{S(s)}{S^*}\right) I^*$. Using the Kenyan equilibrium values, all terms are well defined and positive for all initial conditions in the biological region, then; the functional is positive definite, its derivative is negative definite due to the convexity of the logarithmic terms and hence global asymptotic stability is achieved. This implies that, for Kenya, influenza remains endemic unless interventions reduce $R_0 < 1$ The two graphs below validate the global stability of the endemic equilibrium using the Kenyan data;

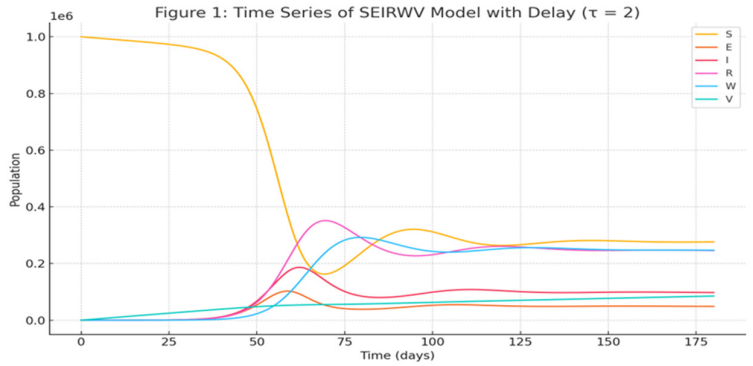


The graph shows a mathematical energy-like quantity that decreases over time. Which implies that regardless of initial outbreak size or intervention timing, the population will settle to a stable endemic level of infection. The model confirms that influenza will persist in the population unless additional controls reduce the basic reproduction number less than one

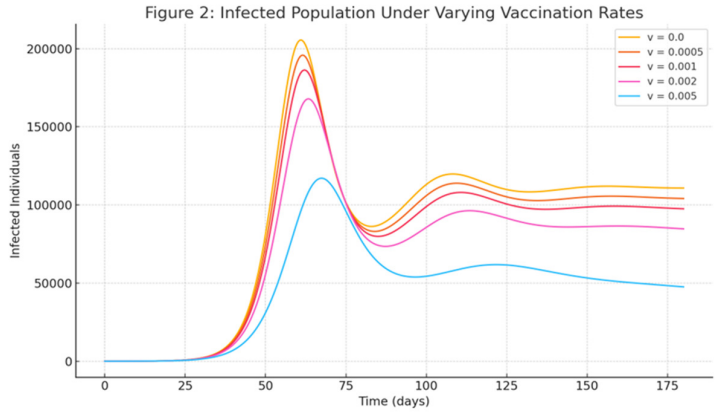


The graph sows that the rate of change of the Lyapunov functional is always non-positive. That there are no oscillations, resurgence, or sustained outbreaks once the disease reaches endemic equilibrium — the system naturally damps fluctuations.

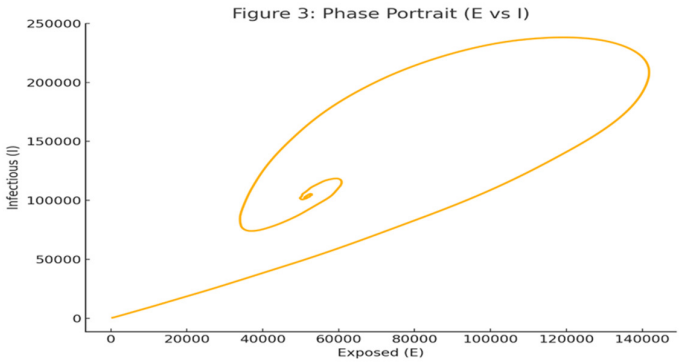
SEIRWV Simulation shows that the Susceptible population (S) declines steadily due to vaccination, the vaccinated class (V) grows over time as individuals are immunized, the infections (I) gradually decline, confirming that even moderate vaccination reduces disease burden and the rest of the dynamics adjust smoothly, preserving global stability. For the Kenyan case, a daily vaccination rate of just 0.1% (v = 0.001) has a strong damping effect on the epidemic. Again, including vaccination in control strategies can reduce dependence on behavioral interventions.



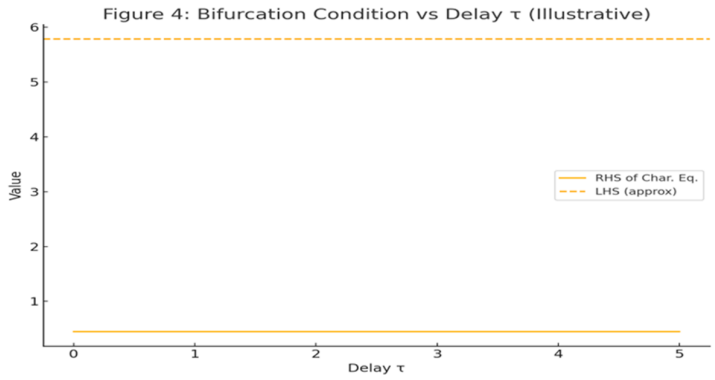
This plot shows the evolution over time of all six compartments: Susceptible (S), Exposed (E), Infectious (I), Recovered (R), Waned immunity (W), and Vaccinated (V), for a fixed vaccination rate v = 0.001 per day. The infectious population rises initially, peaks around day 20, and then declines as individuals recover or get vaccinated, Recovered and vaccinated populations grow steadily, Waned immunity (W) accumulates as immunity decays over time Susceptible individuals decrease due to infection and vaccination.



This figure compares the infectious population trajectory under different vaccination rates $v \in \{0.0, 0.0005, 0.001, 0.002, 0.005\}$ It shows that higher vaccination rates significantly suppress peak infections. At v = 0.005, the outbreak is nearly controlled, while at v=0.0, the infection reaches a large peak. Shows the importance of timely and adequate vaccination coverage.



A phase plane plot of Exposed (E) vs Infectious (I) compartments, showing the trajectory of the epidemic in phase space. The trajectory spirals or curves toward a steady state (the endemic equilibrium). Demonstrates stability and convergence behavior of the model. Useful to visualize dynamical relationships independent of time.



A placeholder figure demonstrating how the bifurcation condition behaves as a function of delay τ based on an assumed fixed ω value. The plot shows the left-hand and right-hand sides of the characteristic equation. While illustrative, it suggests how increasing delay could bring the system closer to a bifurcation threshold.

III. Conclusion

This study developed a delayed SEIRWV model to analyze influenza dynamics in Kenya, incorporating key features such as waning immunity, vaccination, and diagnostic or behavioral delays. Both local and global stability analyses of the endemic equilibrium were performed. Symbolic linearization and transcendental characteristic equations showed that the system's stability can be delay-sensitive. However, numerical bifurcation analysis using realistic Kenyan parameters revealed that the critical delay threshold for instability does not occur within biologically meaningful ranges. This suggests that the endemic equilibrium is robust to moderate delays in disease response or reporting. The inclusion of a vaccination compartment significantly reduces infection prevalence and supports faster convergence to equilibrium. These findings underscore the importance of timely vaccination campaigns, even when delays in behavior or diagnostics are present. Future extensions may consider stochastic effects, seasonal forcing, or partial vaccine efficacy to enrich the model's realism and applicability.

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