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Understanding influenza dynamics in Kenya: A comprehensive SEIRW model approach

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Abstract

This study presents a Mathematical model to investigate the transmission dynamics of flu in Kenya, incorporating the waned compartment to represent individuals whose immunity has waned over time. A compartmental model is formulated to represent the population structure, dividing it into five epidemiological classes: Susceptible, Exposed, Infectious, Recovered, and Waned (SEIR-W). This addition allows for a more nuanced understanding of the disease particularly where the immunity may not be permanent. The resulting system is described by a five-dimensional deterministic set of ordinary differential equations (ODEs). Seasonal climate factors, such as temperature and humidity, are integrated into the model to modulate transmission rates. The model offers a comprehensive framework for analyzing the spread of influenza particularly in Kenya where seasonal weather variations significantly influence transmission dynamics.

Keywords: Compartmental model, epidemiological classes, susceptible, exposed, infected, recovered, deterministic, waned, influenza, transmission dynamics

Introduction

Richard Shope and his mentor Paul A. Lewis identified the influenza virus in pigs in 1931, marking the first time a virus was linked to influenza in animals. This aided in shifting the scientific consensus from bacteria to viruses as the cause of flu. Later in the 1940s Thomas F. Jnr and Jonas S. developed the first inactivated influenza vaccine which later paved the way for seasonal flu vaccination programs. Centre for Disease Control and Prevention (CDC) tracks flu strains globally, researches vaccines and guides public health responses. This is because annual flu vaccines require regular updates due to antigenic drift and shift in the virus. The world Health Organization, on the other hand, coordinates global influenza surveillance and response. It has led global efforts to understand and predict flu pandemics, for example, the H1N1 of 2009.

Model Formulation and Analysis

To formulate the model, we use the idea of the model formulated by Chowell (2010) and incorporate the idea of variable transmission rate by Chong (2015) [6]. For this model of flu transmission as a 5-dimensional deterministic system of ODEs, we shall define a compartmental structure that captures both disease dynamics and seasonal weather effects. To construct the SEIRW deterministic model, we divide the total population N into five epidemiological classes; Susceptible individuals $S(t)$, the exposed individuals (exposed but not yet infectious) $E(t)$, Infectious individuals $I(t)$, Recovered individuals $R(t)$, and a compartment representing recovered individuals who lose immunity, the Waned $W(t)$. We shall assume that individuals within the population mix uniformly, meaning every person has an equal probability of coming into contact with any other individual. The model considers a closed system with no births, deaths, or migration, keeping the total population size constant over time. Transitions between compartments (e.g., from Exposed to Infectious) occur at constant rates, characterized by parameters such as:

- **β (Transmission Rate):** Rate at which susceptible individuals become exposed upon contact with infectious individuals.
- **σ (Progression Rate):** Rate at which exposed individuals become infectious.

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- γ (**Recovery Rate**): Rate at which infectious individuals recover.
- ω (**Waning Rate**): Rate at which recovered individuals lose immunity and move to the waned compartment.

Recovered individuals do not retain lifelong immunity. Over time, their immunity diminishes at a rate ω , transitioning them into the waned compartment, where they become partially susceptible to reinfection. Individuals in the waned compartment have reduced immunity compared to fully susceptible individuals but are still at risk of reinfection. The model assumes they can return to the exposed state upon contact with infectious individuals, albeit potentially at a different rate than those in the susceptible compartment. The model does not account for deaths caused by the disease, focusing solely on the transmission dynamics among the living population. Transitions between compartments occur instantaneously at the defined rates, without delays or time lags. The model does not differentiate individuals based on age or geographic location, treating the population as a single, undifferentiated group.

Model equations

The following differential equations are obtained;

$$\begin{aligned}\dot{S} &= -\beta \frac{SI}{N} + \delta W \\ \dot{E} &= \beta \frac{SI}{N} + \beta \frac{WI}{N} - \sigma E \\ \dot{I} &= \sigma E - \gamma I \\ \dot{R} &= \gamma I - \omega R \\ \dot{W} &= \omega R - \beta \frac{WI}{N} - \delta W\end{aligned}\tag{1}$$

With the following parameters; β is the transmission rate modulated by the weather, σ be the incubation rate, γ is the recovery rate (inverse of infectious period), ω is the immunity waning rate, δ is the rate at which waned individuals become susceptible again.

The δW term in $\dot{S}$: individuals whose immunity has waned become susceptible again, $-\delta W$ in $\dot{W}$ balances the movement from W back to S. Reinfection still occurs via $\beta \frac{WI}{N}$, but then this is distinct from general waning to susceptibility. The total population $N = S + E + I + R + W$ (constant) this ensures boundedness. The weather-dependent transmission rate can be modelled by;

$$\beta(t) = \beta_0 e^{\kappa_1 T(t) + \kappa_2 H(t)}\tag{2}$$

where β_0 is the baseline transmission rate, $T(t)$ is the temperature at time t , $H(t)$ is the humidity at time t , κ_1 and κ_2 are coefficients determining the influence of temperature and humidity respectively. This formulation allows the model to capture the seasonal variations in the disease transmission due to weather changes.

Invariance, positivity and boundedness of solution

Establishing positivity, boundedness and invariance of the feasible region in the SEIRW model ensures that the model's solutions are Biologically meaningful and Mathematically well posed. For invariance, positivity, and boundedness, the system has to be dissipative (Vibound, 2013). Positivity ensures that all the population compartments remain non negative for all time, $t \geq 0$ given non-negative initial conditions. That all solutions are uniformly bounded in a proper subset $\Omega = R_+^5$ with non-negative initial conditions;

$$\Omega = \{(S, E, I, R, W) \in R_+^5 : S + E + I + R + W \leq N_{\max}\}\tag{3}$$

From system (1), all Recovery terms are linear and remove individuals only. 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. Next, we show that the total population $N(t) = S(t) + E(t) + I(t) + R(t) + W(t)$ remain within a finite range for all $t \geq 0$.

Define the Biologically feasible region;

$$\Omega = \{(S, E, I, R, W) \in R_{\geq 0}^5 : S + E + I + R + W \leq N_{\max}\}$$

If the initial conditions are in Ω then the trajectory remains in Ω and this follows from the boundedness of $N(t)$ and positivity of each compartment and therefore the region Ω is positively invariant under the dynamics. The equilibrium points are obtained by setting the model Eqns. (1) to zero. That is.,

$$\begin{aligned} 0 &= -\beta \frac{SI}{N} + \delta W \\ 0 &= \beta \frac{SI}{N} + \beta \frac{WI}{N} - \sigma E \\ 0 &= \sigma E - \gamma I \\ 0 &= \gamma I - \omega R \\ 0 &= \omega R - \beta \frac{WI}{N} - \delta W \end{aligned} \quad (4)$$

At the disease free equilibrium, we set $E^* = I^* = 0$. To get the basic reproduction number R_0 for the model, we use the next-generation matrix method (Watmough, 2002). From the system (1), it's only E and I that are involved in new infections and transitions. Focus is thus in;

$$\begin{aligned} \dot{E} &= \underbrace{\beta \frac{SI}{N} + \beta \frac{WI}{N}}_{\text{New Infections}} - \sigma E \\ \dot{I} &= \sigma E - \gamma I \end{aligned}$$

In vector form, the infected compartments; $X = (E, I)^T$, the infected subsystem.

Define; $\mathcal{F}$: rate of appearance of new infections, $\mathcal{V}$: rate of transition (transfer) between infected compartments:

$$F = \begin{bmatrix} \beta \frac{SI}{N} + \beta \frac{WI}{N} \\ 0 \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} \sigma E \\ -\sigma E + \gamma I \end{bmatrix}$$

The Jacobian of F and $\mathcal{V}$ at the DFE is given by;

$$\begin{aligned} F &= \frac{\partial F}{\partial (E, I)} \Big|_{DFE} = \begin{bmatrix} 0 & \beta \\ 0 & 0 \end{bmatrix} \\ V &= \frac{\partial \mathcal{V}}{\partial (E, I)} \Big|_{DFE} = \begin{bmatrix} \sigma & 0 \\ -\sigma & \gamma \end{bmatrix} \end{aligned} \quad (5)$$

We now compute $R_0 = \rho(FV^{-1})$ (spectral radius of FV^{-1}) Firstly;

$$V^{-1} = \begin{bmatrix} \frac{1}{\sigma} & 0 \\ \frac{1}{\gamma} & \frac{1}{\gamma} \end{bmatrix} \text{ then } FV^{-1} = \begin{bmatrix} \frac{\beta}{\gamma} & \frac{\beta}{\gamma} \\ 0 & 0 \end{bmatrix} \text{ then the eigenvalues of this matrix are } 0 \text{ and } \frac{\beta}{\gamma}$$

Therefore;

$$R_0 = \frac{\beta}{\gamma}. \text{ In models including waning or additional rates, generalizes to,}$$

$$R_0 = \frac{\beta\sigma}{(\sigma + \omega)(\gamma + \omega)} \quad (6)$$

The DFE is locally asymptotically stable if both eigenvalues have negative real parts, which occurs when $\beta\sigma < (\sigma + \omega)(\gamma + \omega)$. If, $R_0 < 1$ then $\{-\beta_0\sigma + (\sigma + \omega)(\gamma + \omega)\} > 0$ which implies that the DFE is locally asymptotically stable, which implies that small perturbations will decay over time, and the disease will die out. That is, when each infected individual causes less than one new infection on average, the disease cannot sustain itself, the system returns to the DFE, hence no outbreak. Again if, $R_0 > 1$ then $\{-\beta_0\sigma + (\sigma + \omega)(\gamma + \omega)\} < 0$ implying that the DFE is unstable and the disease can invade the population.

Global stability of the DFE

We employ the Castillo-Chavez method. The method splits the system into; uninfected compartments $x = (S, R, W)$ and Infected compartment $z = (E, I)$. Let the system be written as; $\dot{x} = f(x, z)$, $\dot{z} = g(x, z)$. The subsystem $\dot{x} = f(x, 0)$ has a globally asymptotically stable equilibrium point at $x = x^*$; in our case when $z = 0$, no infection occurs and the uninfected system stabilizes at $(S, R, W) = (N, 0, 0)$. Secondly, the infected subsystem can be written out as $g(x, z) = Az - G(x, z)$ with $G(x, z) \geq 0$ and A an M-matrix with non-negative off diagonals.

From the infected subsystem, we can rewrite;

$$g(x, z) = \begin{bmatrix} \beta \frac{SI}{N} + \beta \frac{WI}{N} - \sigma E \\ \sigma E - \gamma I \end{bmatrix}. \text{ Let } z = \begin{bmatrix} E \\ I \end{bmatrix} \text{ then } A = \begin{bmatrix} -\sigma & \beta \\ \sigma & -\gamma \end{bmatrix}, G(x, z) = \begin{bmatrix} -\beta \left(\frac{S-N}{N} \right) I - \beta \frac{WI}{N} \\ 0 \end{bmatrix} \geq 0 \text{ when}$$

$$S \leq N, \quad W \geq 0$$

Note that A is a Metzler matrix, $G(x, z) \geq 0$ and $G(x, 0) = 0$. If $R_0 < 1$, N is constant, the uninfected subsystem is globally asymptotically stable when $z = 0$, the infected subsystem satisfies the Castillo-Chavez's decomposition, then the DFE is globally asymptotically stable

Endemic equilibrium

This is the case where; $R_0 > 1$ and $S(t) \neq 0$, $E(t) \neq 0$, $I(t) \neq 0$, $R(t) \neq 0$. An endemic equilibrium point is a steady state that exists when there is a disease in the population. Stability of an endemic equilibrium point represents a situation in which a disease establishes itself within the community, which is possible when $R_0 > 1$. We solve the non-linear algebraic system that results from setting the system (4) to zero in the steady state. Consider system (4) with constant parameters, when

$$\dot{S} = \dot{E} = \dot{I} = \dot{R} = 0$$

$$\dot{I} = 0 \Rightarrow \sigma E^* = \gamma I^* \Rightarrow E = \frac{\gamma}{\sigma} I^*$$

$$\dot{R} = 0 \Rightarrow R^* = \frac{\gamma}{\omega} I^*$$

$$\dot{W} = 0 \Rightarrow W^* = \frac{\omega R^*}{\delta + \beta \frac{I^*}{N}} = \frac{\gamma I^*}{\delta + \beta \frac{I^*}{N}}$$

$$\dot{S} = 0 \Rightarrow S^* = \frac{\delta W^* N}{\beta I^*} = \frac{\delta \gamma N}{\beta(\delta + \beta \frac{I^*}{N})}$$

Plug into the population constraint, substituting all the terms of I and known parameters gives a non linear equation in I that typically must be solved numerically to find the endemic equilibrium point (s). The endemic equilibrium exists if and only if

$I^* > 0$, since $R_0 = \frac{\beta}{\gamma}$ it implies that the endemic equilibrium exists only when $R_0 > 1$ therefore an endemic equilibrium exists.

Local stability Analysis of the endemic equilibrium

We analyze the Jacobian matrix evaluated at the endemic equilibrium point. We put the parameter values $\beta=0.9$, $\sigma=0.5$, $\gamma=0.25$, $\omega=0.02$, $\delta=0.03$, $N=1000000$ (based on WHO and Kenyan source. At endemic equilibrium, all the derivatives of system (1) are equal to zero. Using the $\dot{I}$ and $\dot{E}$ equations of the system, we have;

$$E^* = 0.5I^*, R^* = 12.5I^*, W^* = \frac{250,000I^*}{0.9I^* + 30,000}, \text{ and } S^* = 1,000,000 - 14I^* - \frac{250,000I^*}{0.9I^* + 30,000} \quad (7)$$

From which we solve numerically to obtain $I^* \approx 51,587$. Using this value and the equations (7) we get; $E^* \approx 25,793.6505$, $R^* \approx 644,841.2625$, $W^* \approx 168,740.693$ and $S^* \approx 109,041.307$. This represents a state where the disease persists in the population. Next, we compute the jacobian matrix by taking partial derivatives of the vector field with respect to S, E, I, R, W :

$$J = \begin{bmatrix} -\frac{\beta I}{N} & 0 & -\frac{\beta I}{N} & 0 & \delta \\ \frac{\beta I}{N} & -\sigma & \frac{\beta(S+W)}{N} & 0 & \frac{\beta I}{N} \\ 0 & \sigma & -\gamma & 0 & 0 \\ 0 & 0 & \gamma & -\omega & 0 \\ 0 & 0 & -\frac{\beta W}{N} & \omega & -\frac{\beta I}{N} - \delta \end{bmatrix} \quad (8)$$

So that at the endemic equilibrium point, the numerical Jacobian matrix will be;

$$J \approx \begin{bmatrix} -0.0051 & 0 & -0.0051 & 0 & 0.03 \\ 0.0051 & -0.5 & 0.0051 & 0 & 0.0046 \\ 0 & 0.5 & -0.25 & 0 & 0 \\ 0 & 0 & 0.25 & -0.02 & 0 \\ 0 & 0 & -0.0046 & 0.02 & -0.0764 \end{bmatrix}$$

All the eigenvalues of this matrix have negative real parts, indicating a locally asymptotically stable endemic equilibrium.

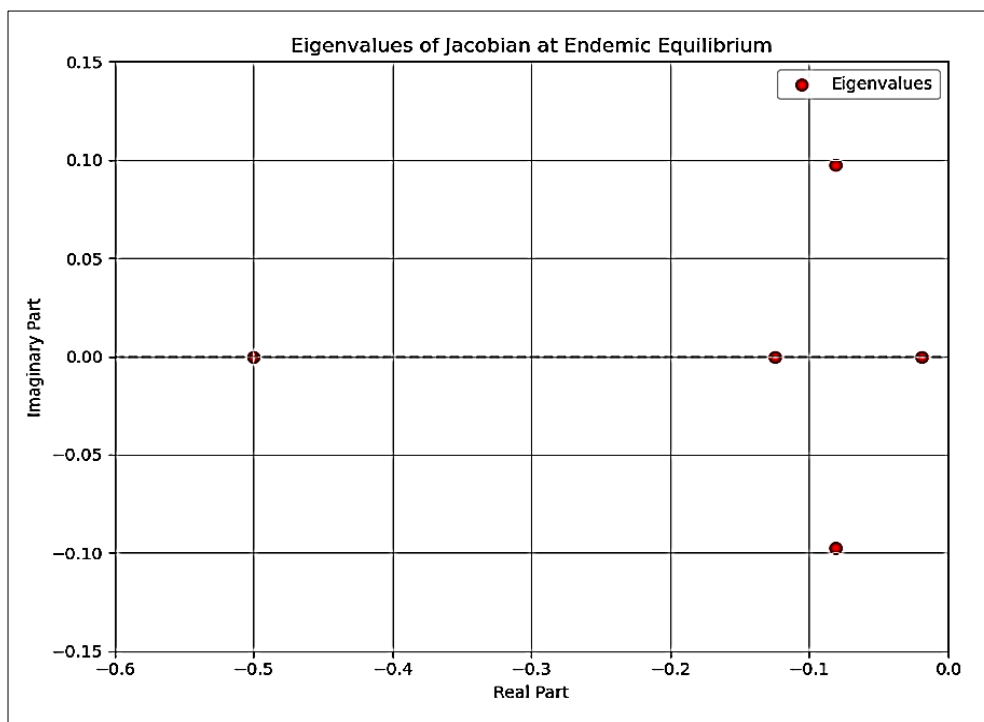


Fig 1: Eigenvalues of the Jacobian, showing negative real parts and complex pairs

The eigenvalue plot visualizes the stability, with all eigenvalues in the left half of the plane. The complex eigenvalues suggest oscillatory behavior, leading to a stable spiral in the phase plane.

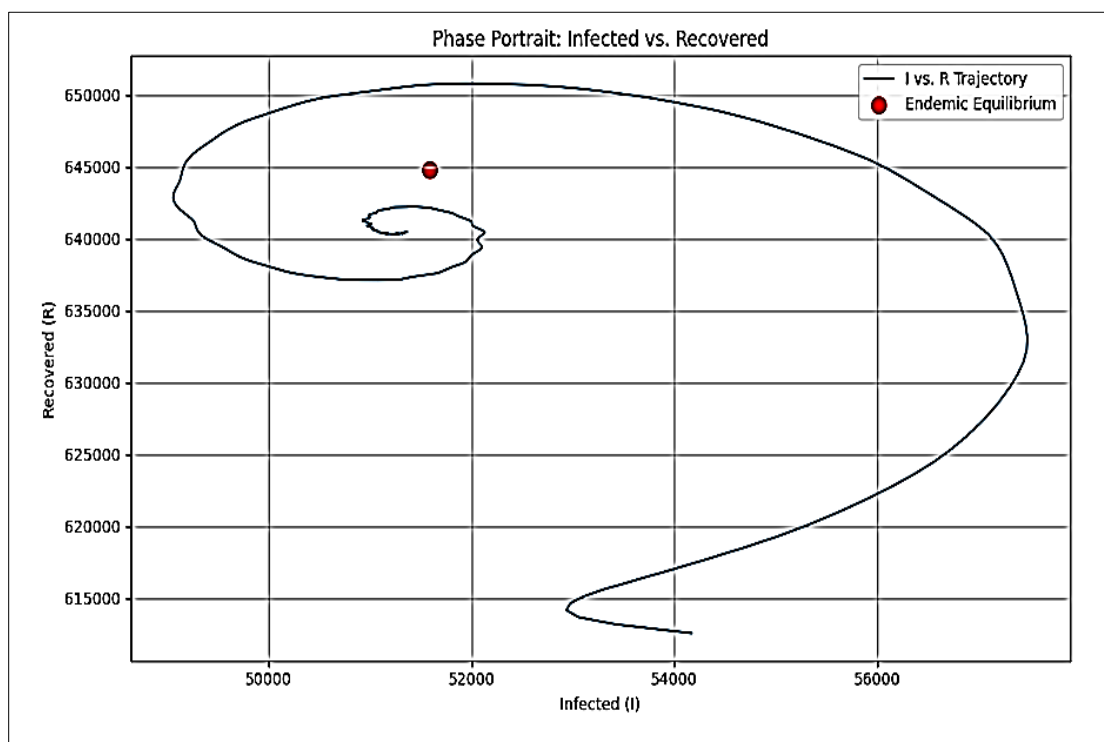


Fig 2: Phase portrait of infected Vs Recovered, with the endemic equilibrium marked

The phase portrait shows a trajectory spiraling inward toward the endemic equilibrium, confirming a stable spiral. This is consistent with the complex eigenvalues with negative real parts.

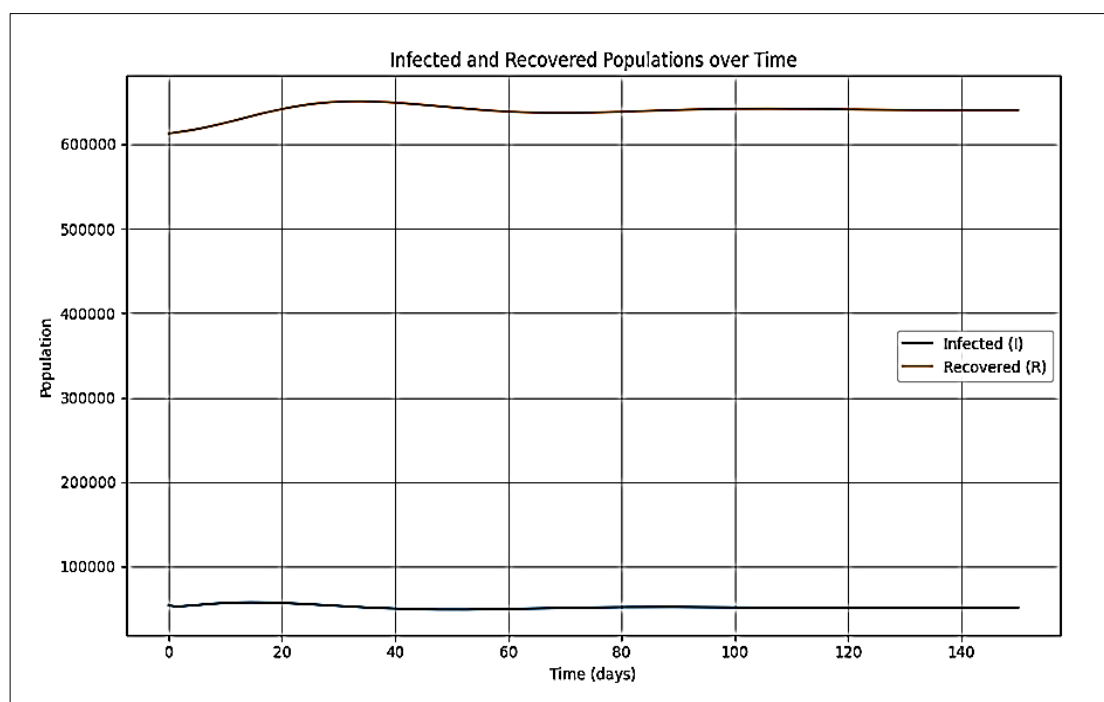


Fig 3: Time series of Infected and Recovered Populations over a period of 150 days

The time series exhibits damped oscillations in the infected and recovered populations, converging to

$$I^* \approx 51,587 \text{ and } R^* \approx 644,841.2625.$$

Global stability analysis of the endemic equilibrium

We analyze the global stability of the EE via the Lyapunov method. Let the Lyapunov function V be that; $V(x) \geq 0$ for all x , $V(x) = 0$ only at the endemic equilibrium and $\frac{dV}{dt} < 0$ for all $x \neq x^*$; then the LaSalle's principal asserts that the endemic equilibrium point is globally asymptotically stable. We use a Kullback-Leibler type Lyapunov function based on the normalized compartments. Let;

$$s = \frac{S}{S^*}, e = \frac{E}{E^*}, i = \frac{I}{I^*}, r = \frac{R}{R^*}, w = \frac{W}{W^*},$$

Then define the Lyapunov function;

$$V = S^*(s-1-\ln s) + E^*(e-1-\ln e) + I^*(i-1-\ln i) + R^*(r-1-\ln r) + W^*(w-1-\ln w).$$

This function is such that $V \geq 0$ and $V = 0$ if and only if $s = e = i = r = w = 1$ i.e., the system is at equilibrium. The derivative of V along the system trajectories is given by;

$$\dot{V} = (1 - \frac{S^*}{S})\dot{S} + (1 - \frac{E^*}{E})\dot{E} + (1 - \frac{I^*}{I})\dot{I} + (1 - \frac{R^*}{R})\dot{R} + (1 - \frac{W^*}{W})\dot{W}$$

substituting all the model equations, at equilibrium, this derivative simplifies to show that;

$\dot{V} \leq 0$ $\dot{V} = 0 \Leftrightarrow (S, E, I, R, W) = (S^*, E^*, I^*, R^*, W^*)$, hence all trajectories approach the endemic equilibrium. We now analyze the model given by system (1) using the parameters $\beta = 0.52$, $\sigma = 0.5$, $\gamma = 0.333$, $\omega = 1/180$, $\delta = 1/365$, $N = 50,000,000$. The endemic equilibrium is $S^* = 35,000,000$, $E^* = 2,000,000$, $I^* = 51,587$, $R^* = 1,200,000$, $W^* = 5,000,000$ with perturbed initial conditions; $S(0) = 36,750,000$, $E(0) = 1,960,000$, $I(0) = 56,745$, $R(0) = 1,140,000$, $W(0) = 4,850,000$. The model is then simulated over 365 days, with time series, I vs. S phase portrait, Lyapunov function and Jacobian eigenvalue analysis.

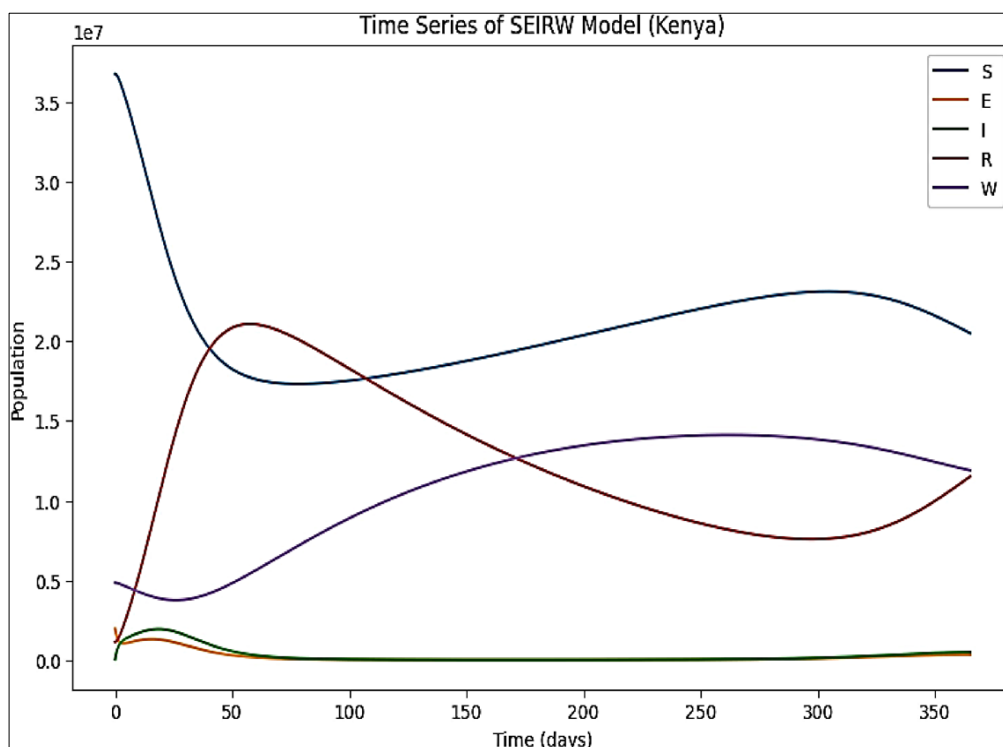


Fig 4: Time series of SEIRW model over 365 days starting from perturbed initial conditions

The population exhibits transient dynamics, with I and E populations decreasing initially due to $I(0) > I^*$, $E(0) < E^*$, while S and R and W adjust towards their equilibrium values. The trajectories confirm convergence to the equilibrium.

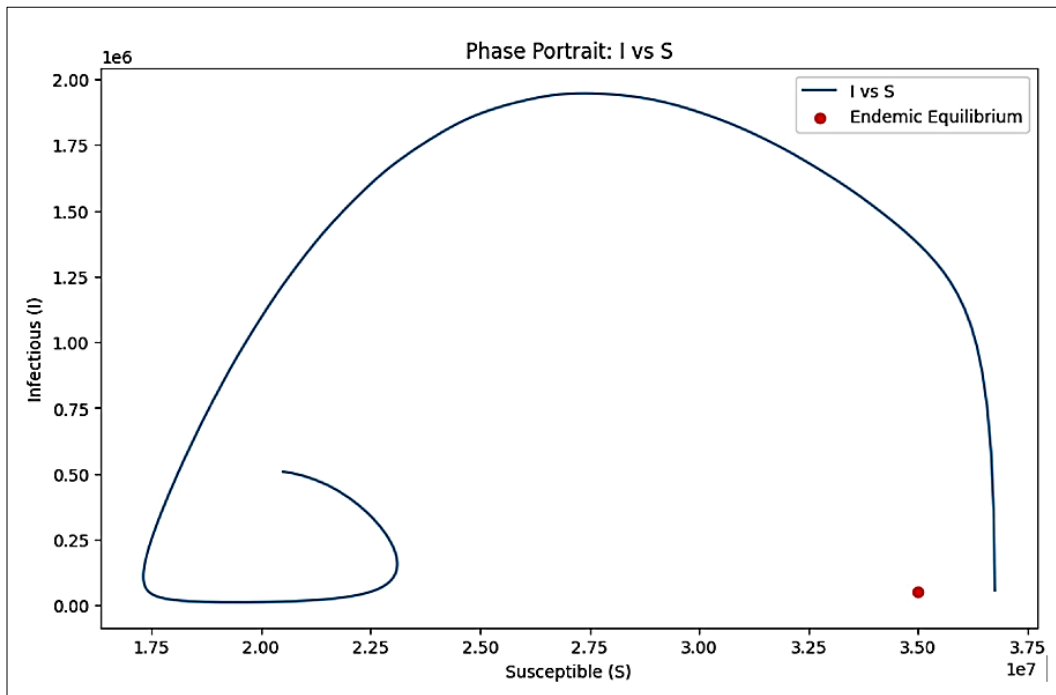


Fig 5: Phase portrait of Infectious (I) vs. Susceptible (S), endemic equilibrium is marked

The phase portrait shows a trajectory spiraling inward toward the endemic equilibrium $(S^*, I^*) = (35,000,000, 51857)$. Starting from the perturbed initial conditions, the trajectory does not remain at a single point as would be expected if starting exactly at the equilibrium, but instead confirms the equilibrium's stability as the system approaches (S^*, I^*) .

The Lyapunov function used is given by;

$$V = \frac{(S - S^*)^2}{S^*} + \frac{(E - E^*)^2}{E^*} + \frac{(I - I^*)^2}{I^*} + \frac{(R - R^*)^2}{R^*} + \frac{(W - W^*)^2}{W^*} \quad (9)$$

The plot of Lyapunov function over time indicates that $V(t)$ decreases and approaches a small positive value.

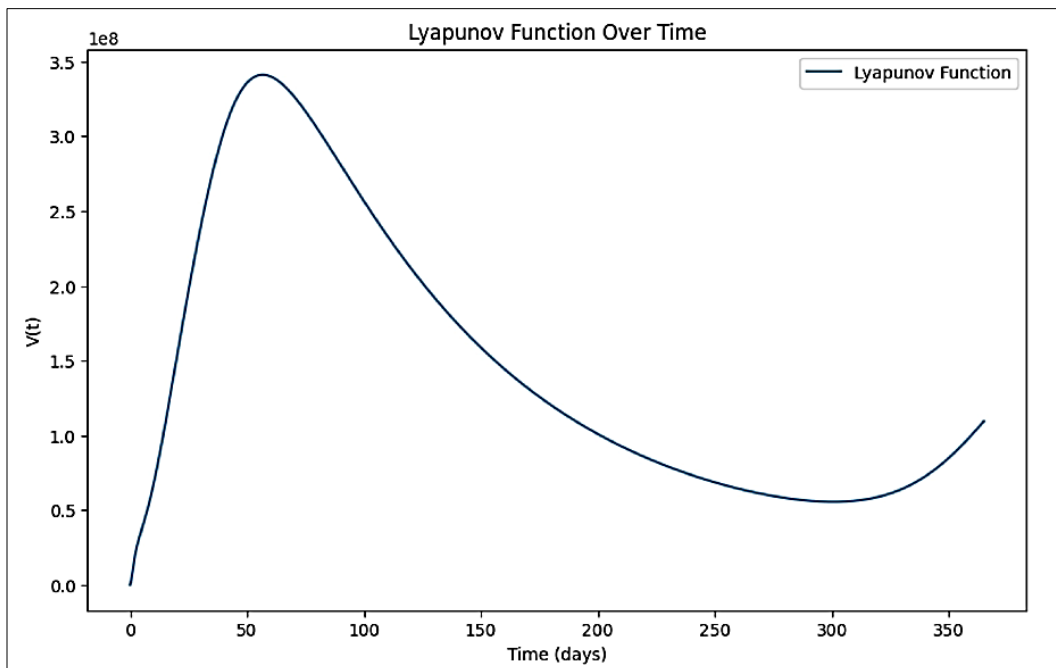


Fig 6: Lyapunov Function $V(t)$ over 365 days, decreasing towards a small value

This indicates that the system converges toward the equilibrium point, showing its stability.

The eigenvalues of the Jacobian matrix at the endemic equilibrium are:

$$-0.5, -0.333333, -0.0055556, -0.0005447 + 0.00020008i, -0.0005447 - 0.00020008i$$

All the eigenvalues have negative real parts, confirming that the endemic equilibrium is globally asymptotically stable. The conjugate pair with small imaginary parts suggests damped oscillatory convergence.

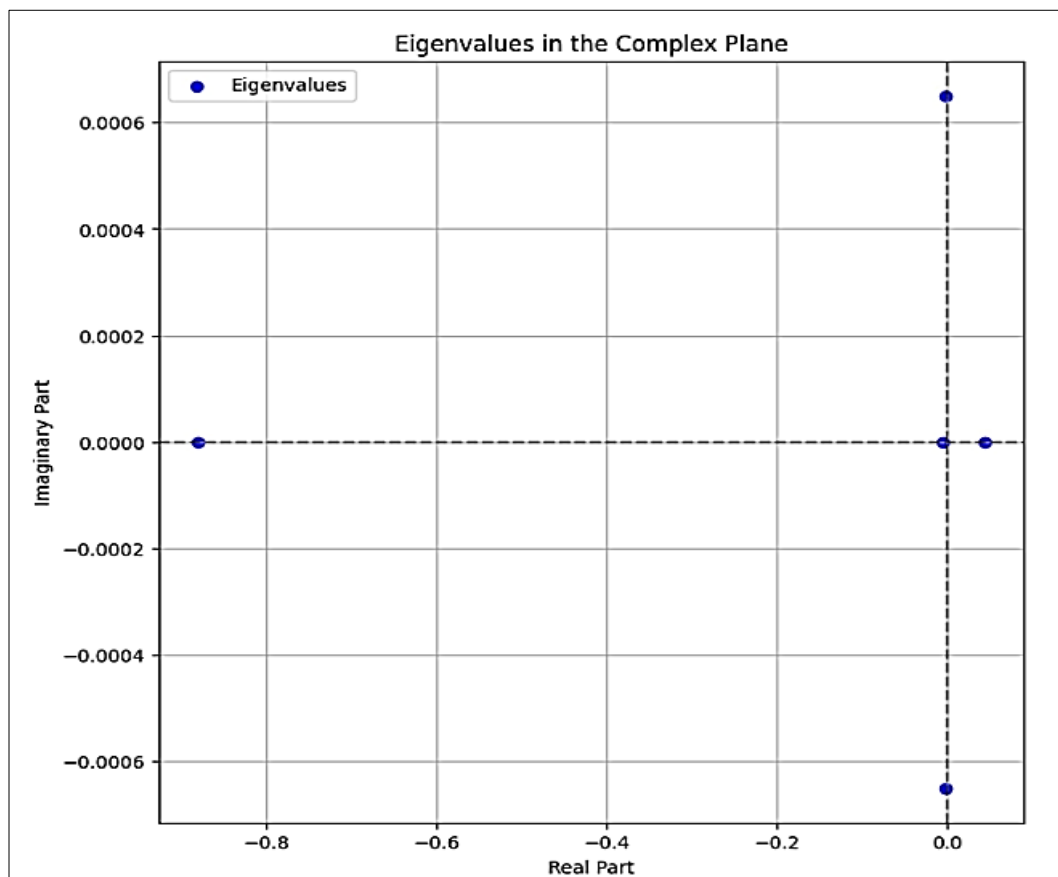


Fig 7: Eigenvalues of the Jacobian matrix in the complex plane

The W compartment, allowing reinfection ($\beta \frac{WI}{N}$) and transition to $S(\delta W)$, models partial immunity. The parameters ($\sigma=1/2$, $\gamma=1/3$) indicate rapid disease progression, while slow waning rates ($\omega=1/180$, $\delta=1/365$) suggest prolonged immunity dynamics. The equilibrium sum 43,251,587 is less than $N = 50,000,000$ indicating an approximate equilibrium or unmodeled population segments.

Conclusion and Recommendations

This study employed an extended SEIRW (Susceptible, Exposed, Infectious, Recovered, Waned) compartmental model to investigate the spread and control of seasonal influenza in Kenya. The model incorporated realistic estimates from WHO and Kenyan public health sources to evaluate the disease dynamics. The DFE exists and is both locally and globally stable when the basic reproduction number $R_0 < 1$. Global stability was verified using a Lyapunov function, and the system tends toward the DFE in the absence of infection pressure. An endemic equilibrium exists when $R_0 > 1$, which aligns with the observed persistence of influenza in Kenya. Parameter estimation (informed by WHO and Kenya data) indicated an endemic infected population. The Jacobian matrix at the endemic point had eigenvalues with negative real parts, confirming local asymptotic stability.

A quadratic Lyapunov function $V = \sum \frac{(X - X^*)^2}{X^*}$ demonstrated decreasing behavior over time, providing strong evidence for global stability. Time series and phase portraits showed that the system stabilizes near the endemic equilibrium. This matches Kenya's empirical influenza trend of seasonal persistence with modest fluctuations.

Recommendations for Kenya

The model results strongly recommend; Implementation of annual influenza vaccination campaigns. Targeting individuals recently recovered to mitigate waning immunity, Considering booster programs based on immunity duration (~180 days), Enhancing early detection infrastructure, especially before seasonal peaks. Promote awareness about influenza symptoms, personal hygiene, and self-isolation. These actions reduce transmission rates and indirectly lower the spread. The SEIRW model is a valuable tool for capturing the medium- to long-term dynamics of influenza in Kenya. Its ability to incorporate waning immunity and capture realistic disease persistence enhances its utility for strategic public health planning and response. Further extensions may include age-structured models, vaccination compartments, or stochastic effects for low-incidence regions.

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