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Mathematical Modelling and Analysis of Influenza (H5N1)

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Abstract In this study, we introduce a continuous MSEIHR model and explore its dynamic behavior and fundamental properties. Using Lyapunov functions and the Routh-Hurwitz conditions, we perform a stability analysis of the model. Our results confirm that when the basic reproduction ratio $R_0 < 1$, the system is both globally and locally stable at the disease-free equilibrium E_{ef}. Conversely, when $R_0 > 1$, an endemic equilibrium E_{eq} emerges, and the system stabilizes at this equilibrium. Additionally, we analyze the sensitivity of the MSEIHR model to identify the parameters with the most substantial influence on R_0. Finally, we validate our theoretical findings with numerical simulations using MATLAB.

Keywords Epidemiological process, Avian influenza, Positivity, Stability.

AMS 2010 subject classifications 92D30, 93C55, 93B03, 93Dxx

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1. Influenza (H5N1) Overview

Avian influenza (AI), caused by an Orthomyxoviridae RNA virus, is a respiratory disease that affects both birds and mammals (see [1] for more details). The virus is classified into three primary types: A, B, and C, derived from differences in two key proteins [4]. Among these, the (AI) virus type A is epidemiologically more significant and dangerous, raising concerns due to its ecological and evolutionary implications across diverse bird and mammal species. This virus frequently undergoes substantial changes in its immunological characteristics. According to [5], type A avian influenza includes three subtypes AH5, AH9, and AH7, that can be transmitted to both birds and humans. Human transmission occurs either through inhalation of airborne viral particles or contact with infected surfaces. Symptoms in humans typically include fever, cough, chills, and headaches. Although the virus circulates naturally in birds, and human infections usually results from exposure to infected excrement, often leading to severe health outcomes.

In 1998, there were 16 reported human cases and three suspected ones [6]. More recently, [7] reports that Indonesia reported 151 cases, leading to 52 deaths, while Vietnam confirmed 119 cases with 59 deaths. In Hong Kong in 2004, the avian influenza virus was detected in migratory birds, though no infections were found in local poultry, pet birds, or wild birds [8]. Further, in 2023, a man from Chile contracted the virus, despite having no underlying conditions or recent travel history. WHO confirmed that a poultry farm worker in England tested positive for the severe avian influenza AH5N1 virus in mid-May, followed by a second case in workers involved in slaughter operations at the same farm. Concerns over the rapid global spread of avian influenza remain high. Most studies on avian influenza complications have employed discrete-time models expressed through differential equations. The severity of these complications varies, with some being manageable while others progressing to

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a critical, incurable stage. Our research is driven by the ongoing debate surrounding the origins of the virus and availability of vaccines. Some theories suggest the virus is introduced via contaminated poultry imports, while others implicate migratory birds as seasonal carriers. Typically, mathematical models in epidemiology divide the population into distinct groups, each representing different health states related to the disease being studied. These models evolve over time as the number of individuals in each group changes with their health status. This approach is particularly important for diseases such as avian influenza, measles, chickenpox, rubella, and mumps, where the duration of illness is relatively short. As a result, the impact of birth and death rates during the outbreak is often considered negligible. One of the fundamental models used in epidemiology is the SIR model, which categorizes the community into Susceptible, Infected, and Recovered/Immune subgroups. This model was first introduced by Kermack and McKendrick in 1927 (see [15]) and has significantly influenced the study of disease spread. Subsequent models, such as the Greenwood and Reed-Frost models, the SI model (where recovery is not possible) [17], and more sophisticated SIR models, have expanded on this framework. Some models divide the susceptible population into subgroups with various infection rates or consider varying severity levels of infection [11]. Advanced models may also incorporate antiviral treatments and vaccination strategies [12, 13, 16]. Various epidemiological models can be developed based on the categories considered, including the SIS model [19], which expands the classical (SIS) epidemic model by evolving from a deterministic to a stochastic framework, formulating it as a differential stochastic system (SDE) for the size of the infected population. The SIRS model [20] introduces Lyapunov mapping for the well-known SIR, SIS and SIRS models, to demonstrate the global stability of these models. The SEIR model [21] presents explicit Lyapunov mappings for SIR and SEIR models involving nonlinear incidence. Finally, the MSEIR model [22] introduces a novel approach to address the asymptotic dynamics of age-structured epidemic equations and explores their applications to the MSEIR models, among others.

In our study, we examine the following compartments within the MSEIHR model. The (M) class consists of individuals with passive immunity, having acquired protective antibodies from their mothers. The (S) class includes those who are susceptible to the disease but have not yet been exposed. The (E) class refers to individuals who have been exposed but are not yet infectious. The (I) class represents infected individuals who are capable of transmitting the disease. The (H) class is composed of hospitalized individuals, while the (R) class includes those who have recovered and developed lasting immunity. To describe this model mathematically, we propose a continuous mathematical model using differential equations. Initially, we will examine the local stability at both disease-free and endemic equilibrium points. Since data collection often involves measurement and parameter estimation, we will also conduct a sensitivity analysis to identify the parameters that significantly influence the basic reproduction number, R_0.

The paper is structured as follows: Section 2 presents the formulation of the model and its basic properties. In Section 3, we discuss the equilibrium points of the model. Section 4 analyzes the local stability of the equilibrium points. In Section 5, we address the sensitivity analysis of the model parameters. Section 6 presents numerical simulations that validate the theoretical findings. Finally, we conclude by summarizing and discussing the results.

2. A Classical MSEIHR Model and Basic Results

We introduce the MSEIHR model to represent the spread of H5N1 within a population, segmented into six compartments: M, S, E, I, H, and R, where the total population size $N = M + S + E + I + H + R$ remains constant over time.

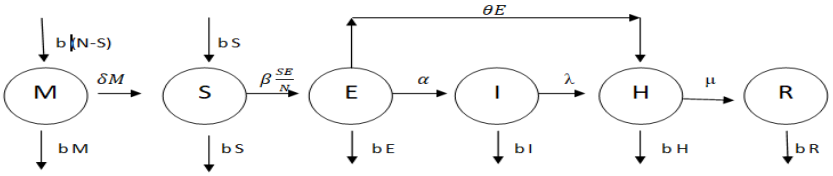

Figure 1. MSEIHR model

We examine the following system of differential equations for the classical MSEIHR model:

$$\begin{cases} \frac{dM(t)}{dt} = b(N - S(t)) - (b+\delta)M(t) \\ \frac{dS(t)}{dt} = bS(t) + \delta M(t) - \beta\frac{S(t)E(t)}{N} - bS(t) \\ \frac{dE(t)}{dt} = \beta\frac{S(t)E(t)}{N} - (\alpha + b + \theta)E(t) \\ \frac{dI(t)}{dt} = \alpha E(t) - (b+\lambda)\, I(t) \\ \frac{dH(t)}{dt} = \lambda I(t) - (\mu + b)\, H(t) \\ \frac{dR(t)}{dt} = \mu H(t) + \theta E(t) - bR(t). \end{cases} \tag{1}$$

The initial states are represented by the nonnegative constants M_0, S_0, E_0, I_0, H_0, and R_0. The coefficients are defined as follows: δ is the rate at which individuals leave the M group, β the transmission rate from susceptible to asymptomatic infected cases, b the natural death rate across all compartments, α the progression rate from asymptomatic to symptomatic cases, λ is the transmission rate from symptomatic individuals to hospitalized cases, μ is the recovery rate of hospitalized cases, and θ is the recovery rate of asymptomatic cases due to strong immunity.

The (M) compartment represents individuals with passive immunity from maternal antibodies. Its population increases by $bN(t)$ and decreases by $bS(t)$, reflecting natural mortality, and by $\delta M(t)$ due to immunity loss. The (S) compartment includes individuals susceptible to the disease but not yet exposed. It increases by $bS(t)$ and $\delta M(t)$ and decreases by $\beta\frac{S(t)E(t)}{N}$, representing exposure through contact, and by b for natural mortality. The (E) compartment includes individuals exposed but non-infectious individuals. It increases by $\beta\frac{S(t)E(t)}{N}$ and decreases by $\alpha E(t)$, $\theta E(t)$, and b. The (I) compartment represents infectious individuals, increasing by $\alpha E(t)$ and decreasing by b and $\lambda I(t)$, which reflects recovery or hospitalization. The (H) compartment represents hospitalized individuals, increasing by $\lambda I(t)$ and decreasing by $\mu H(t)$ and b. The (R) compartment represents recovered individuals with lasting immunity, increasing by $\mu H(t)$ and $\theta E(t)$ and decreasing by b.

Since the total population size N remains constant, a second set of variables is introduced to represent the proportion of the population in each compartment:

$$m = M/N,\ s = S/N,\ e = E/N,\ i = I/N,\ h = H/N \ \text{ and } \ r = R/N.$$

Thus, model 1 can be reformulated as:

$$\begin{cases} \frac{dm(t)}{dt} = b(1 - s(t)) - (b+\delta)m(t) \\ \frac{ds(t)}{dt} = bs(t) + \delta m(t) - \beta s(t)e(t) - bs(t) \\ \frac{de(t)}{dt} = \beta s(t)e(t) - (\alpha + b + \theta)e(t) \\ \frac{di(t)}{dt} = \alpha e(t) - (b+\lambda)\, i(t) \\ \frac{dh(t)}{dt} = \lambda i(t) - (\mu + b)\, h(t) \\ \frac{dr(t)}{dt} = \mu h(t) + \theta e(t) - br(t) \end{cases} \tag{2}$$

where the following initial values are assumed to belong in $(0, 1)$

$$m(0) = m_0,\ e(0) = e_0,\ s(0) = s_0,\ i(0) = i_0,\ h(0) = h_0 \ \text{ and } \ r(0) = r_0. \tag{3}$$

Now, we demonstrate that all solutions of System 2 with nonnegative initial conditions remain nonnegative for all time. This will be confirmed through the following lemma.

Lemma 2.1 (Invariant Region)
The feasible region of System 2 is defined as $\Omega = \big\{(m, s, e, i, h, r) \in \mathbb{R}^6_+ \ : \ m + s + e + i + h + r = 1\big\}$.

Proof
First, we remark that we have

$$\begin{aligned}
&\frac{dm(t)}{dt} + \frac{ds(t)}{dt} + \frac{de(t)}{dt} + \frac{di(t)}{dt} + \frac{dh(t)}{dt} + \frac{dr(t)}{dt} \\
&= b(1 - s(t)) - (b + \delta)m(t) + bs(t) + \delta m(t) - \beta s(t)e(t) - bs(t) + \beta s(t)e(t) \\
&\quad - (\alpha + b + \theta)e(t) + \alpha e(t) - (b + \lambda)\, i(t) + \lambda i(t) - (\mu + b)\, h(t) + \mu h(t) + \theta e(t) - br(t) \\
&= b - b(m(t) + s(t) + e(t) + i(t) + h(t) + r(t)).
\end{aligned}$$

Thus, to get $b - b(m(t) + s(t) + e(t) + i(t) + h(t) + r(t)) = 0$, we must impose

$$m(t) + s(t) + e(t) + i(t) + h(t) + r(t) = 1.$$

□

Theorem 2.2 (Positivity)
If m_0, s_0, e_0, i_0, h_0 and r_0 are non-negative, the solutions $\big(m(t), s(t), e(t), i(t), h(t), r(t)\big)$ of System 2 will stay nonnegative for each $t > 0$.

Proof
We have

$$\frac{ds(t)}{dt} = \delta m(t) - \beta s(t)e(t) = \delta m(t) - \mathfrak{Z}(t)s(t), \tag{4}$$

where $\mathfrak{Z}(t) = \beta e(t)$. We next multiply the equation (4) by $\exp\big(\int_0^t \mathfrak{Z}(s)\, ds\big)$ to infer

$$\frac{ds(t)}{dt} * \exp\big(\int_0^t \mathfrak{Z}(s)\, ds\big) = [\delta m(t) - \mathfrak{Z}(t)s(t)] * \exp\big(\int_0^t \mathfrak{Z}(s)\, ds\big), \tag{5}$$

which yields that

$$\frac{ds(t)}{dt} * \exp\big(\int_0^t \mathfrak{Z}(s)\, ds\big) + \mathfrak{Z}(t)s(t) * \exp\big(\int_0^t \mathfrak{Z}(s)\, ds\big) = \delta m(t) * \exp\big(\int_0^t \mathfrak{Z}(s)\, ds\big). \tag{6}$$

Hence, we deduce

$$\frac{d}{dt}\Big[s(t) * \exp\big(\int_0^t \mathfrak{Z}(s)\, ds\big)\Big] = \delta m(t) * \exp\big(\int_0^t \mathfrak{Z}(s)\, ds\big). \tag{7}$$

Next, we take the integral with respect to s from 0 to t, we get

$$s(t) * \exp\Big[\int_0^t \mathfrak{Z}(s)\, ds\Big] = s(0) + \delta * \int_0^t m(w)\Big[\exp\Big(\int_0^N \mathfrak{Z}(s)\, ds\Big)\Big] dw. \tag{8}$$

Multiply this relation by $\exp\big[-\int_0^t \mathfrak{Z}(s)\, ds\big]$, we get

$$s(t) - s(0) * \exp\Big[-\int_0^t \mathfrak{Z}(s)\, ds\Big] = \delta * \exp\Big[-\int_0^t \mathfrak{Z}(s)\, ds\Big] * \int_0^t m(w)\Big[\exp\Big(\int_0^w \mathfrak{Z}(s)\, ds\Big)\Big] dw.$$

Thus, the solution $s(t)$ is non-negative, since

$$s(t) = s(0) * \exp\Big[-\int_0^t \mathfrak{Z}(s)\,ds\Big] + \delta * \exp\Big(-\int_0^t \mathfrak{Z}(s)ds\Big) * \int_0^t m(w)\Big(\exp\Big(\int_0^w \mathfrak{Z}(s)ds\Big)\Big)dw \geq 0.$$

Similarly, from the other equations in System 2, we have $m(t) \geq 0$, $e(t) \geq 0$, $i(t) \geq 0$, and $r(t) \geq 0$ for all $t \geq 0$. Consequently, the solutions $m(t)$, $s(t)$, $e(t)$, $i(t)$, $h(t)$, and $r(t)$ of System 2 stay non-negative for $t \geq 0$.

□

The first three relations in System 2 do not involve i, h, and r. Thus, the dynamics of System 2 is equivalent to

$$\begin{cases} \dfrac{dm(t)}{dt} = b(1-s) - (b+\delta)m \\ \dfrac{ds(t)}{dt} = bs + \delta m - \beta se - bs = \delta m - \beta se \\ \dfrac{de(t)}{dt} = \beta se - (\alpha + b + \theta)e. \end{cases} \tag{9}$$

3. Analysis of Stability and Model Sensitivity

We distinguish two equilibrium points for this model: the disease-free equilibrium point and the disease-present equilibrium point. To find these equilibria points, we set the right-hand side of equation (9) equal to 0. The disease-free equilibrium, $E^0_{ef}(0,1,0)$, occurs when the virus is absent ($m = e = 0$). The disease-present equilibrium, $E^*_{eq} = (m^*, s^*, e^*)$, is reached when the disease is present ($s \neq 0$ and $e \neq 0$), where:

$$m^* = \frac{\beta b - b(\alpha+b+\theta)}{\beta(b+\delta)}\,,\quad s^* = \frac{\alpha+b+\theta}{\beta}\,,\quad e^* = \frac{\delta b}{\beta(b+\delta)}\Big(\frac{\beta}{\alpha+b+\theta} - 1\Big)\,,\quad R_0 = \frac{\beta}{\alpha+b+\theta}.$$

Note that the number R_0 indicates the expected number of new infections that a solitary infected person could cause in a population of individuals who have not yet been infected. A high value of R_0 suggests a higher potential for an epidemic. We will now explore the local stability of E^0_{ef} and E^*_{eq}. We will start by analyzing the local stability of the disease-free equilibrium E^0_{ef}.

Theorem 3.1 (Illness-free equilibria)
The disease-free equilibrium $E^0_{ef}\big(0,1,0\big)$ of System 9 is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof
First, the Jacobian at the point E_{ef} is given by the matrix below

$$J(E_{\text{ef}}) = \begin{bmatrix} -(b+\delta) & -b & 0 \\ \delta & -\frac{\beta E}{N} & -\frac{\beta S}{N} \\ 0 & \frac{\beta E}{N} & \frac{\beta S}{N} - (\alpha+b+\theta) \end{bmatrix}.$$

For the disease-free equilibrium, the Jacobian matrix is

$$J(E^0_{\text{ef}}) = \begin{bmatrix} -(b+\delta) & -b & 0 \\ \delta & 0 & -\beta \\ 0 & 0 & \beta - \alpha - b - \theta \end{bmatrix}.$$

The $J(E_{ef}^0)$ eigenvalues are determined by $\det(J(E_{\text{ef}}^0) - \lambda I) = 0$, and they are

$$\lambda_1 = \beta - \alpha - b - \theta\,,\ \ \lambda_2 = \frac{-A - \sqrt{A^2 - 4B}}{2}\,,\ \ \lambda_3 = \frac{-A + \sqrt{A^2 - 4B}}{2},$$

where $A = b + \delta$ and $B = b\delta$. Therefore, if $R_0 < 1$, the disease-free equilibrium is locally asymptotically stable and, unstable when $R_0 > 1$. □

Next, we examine the local stability of the disease-present equilibrium. To do this, we impose the conditions:

$$\frac{dm(t)}{dt} = 0\,,\ \frac{ds(t)}{dt} = 0 \ \text{ and } \ \frac{de(t)}{dt} = 0.$$

From these, we find $m^* = \frac{b}{b+\delta}\big(1 - \frac{1}{R_0}\big)$. Substituting this into the second equation of System (9) yields

$$s^* = \frac{1}{R_0},$$

Additionally, the third equation of System 9 provides

$$e^* = \frac{\delta b}{\beta(b+\delta}(R_0 - 1).$$

Consider the theorem below, regarding the local stability of the disease-present equilibrium.

Theorem 3.2 (Disease-present equilibrium)
If $R_0 > 1$, the disease-present equilibrium E_{eq}^* is locally asymptotically stable and, unstable when $R_0 \leq 1$.

Proof
Let $E_{eq}^*(S^*, E^*, I^*)$ denote the disease-present equilibrium of Model 9, where $S^* \neq 0$, $E^* \neq 0$ and $I^* \neq 0$. The Jacobian at the point E_{eq}^* is the matrix below:

$$J(E_{eq}^*) = \begin{bmatrix} -(b+\delta) & -b & 0 \\ \delta & -\frac{\delta b}{b+\delta}(R_0 - 1) & -\frac{\beta}{R_0} \\ 0 & \frac{\delta b}{b+\delta}(R_0 - 1) & \frac{\beta}{R_0} - (\alpha + b + \theta) \end{bmatrix}.$$

We find that the characteristic equation of $J(E_{eq}^*)$ is as as follows:

$$\varphi(\zeta) = \zeta^3 + a_1\zeta^2 + a_2\zeta + a_3,$$

where

$$a_1 = b + \delta + \frac{\delta b}{b+\delta}\,(R_0 - 1) > 0\,,\ \ a_2 = \delta b\,(R_0 - 1) + \frac{\delta b\,(\alpha + b + \theta)}{b+\delta}\,(R_0 - 1) + \delta b > 0,$$

and

$$a_3 = \delta b\,(\alpha + b + \theta)\,(R_0 - 1).$$

By utilizing the Routh-Hurwitz criterion, Model 9 is locally asymptotically stable for

$$a_1 > 0\,,\ a_2 > 0\,,\ a_3 > 0 \ \text{ and } \ a_1 a_2 > a_3.$$

Thus, the disease-present equilibrium E_{eq}^* of System 9 is locally asymptotically stable for $R_0 > 1$. □

4. Global Stability of the MSEIHR model

To attest to the global asymptotic stability of System 9, we employ Lyapunov function properties at both the disease-present and the disease-free equilibriums. we first consider with the disease-free equilibrium E^0_{ef}.

Theorem 4.1 (Disease-free equilibrium)
The disease-free equilibria E^0_{ef} is asymptotically globally stable in Ω if $R_0 \leq 1$, and unstable for $R_0 > 1$.

Proof
Introduce Lyapunov map $V : \Gamma \to \mathbb{R}$ defined by $V(m, s, e) = e$ so that

$$\Gamma = \big\{(m, s, e) \in \Gamma \ : \ m > 0,\ s > 0,\ e > 0\big\}.$$

Hence, the derivative of a Lyapunov map is given as follows:

$$\begin{aligned}\frac{dV(m,s,e)}{dt} = \frac{de}{dt} &= \big(\beta s - (\alpha + b + \theta)\big)\, e \\ &= \big(R_0(\alpha + b + \theta) - (\alpha + \theta + b)\big)\, e = \big(\alpha + \theta + b\big)\,(R_0 - 1)\, e.\end{aligned}$$

Thus, we infer that $\dfrac{dV}{dt} \leq 0 \iff R_0 \leq 1$ and $\dfrac{dV}{dt} = 0 \iff e = 0$. By employing LaSalle's invariance principale, it yields that E^0_{ef} is globally asymptotically stable in Γ, see for more details [23]. □

The final result here concerns the global stability of the disease-present equilibrium E^*_{eq}.

Theorem 4.2 (Disease-present equilibrium)
The disease-present equilibrium E^*_{eq} is globally asymptotically stable if $R_0 > 1$.

Proof
Take the Lyapunov map $V : \Gamma \to \mathbb{R}$ given by

$$V(m, s) = \frac{m^*}{b}\left(m - m^* \ln\left(\frac{m}{m^*}\right)\right) + s^*\left(s - s^* \ln\left(\frac{s}{s^*}\right)\right),$$

where

$$\Gamma = \big\{(m, s) \in \Gamma : m > 0,\ s > 0\big\},$$

and

$$V(m, s) = \frac{m^*}{b}\left(m - m^* \ln\left(\frac{m}{m^*}\right)\right) + s^*\left(s - s^* \ln\left(\frac{s}{s^*}\right)\right)$$

Then, the derivative of the mapping V is as follows:

$$\begin{aligned}\frac{dV(m,s)}{dt} &= \frac{m^*}{b}(m - m^*)\Big[\frac{b(1-s)}{m} - (b + \delta)\Big] + s^*(s - s^*)\Big[\frac{\delta m}{s} - \beta e\Big] \\ &= -m^*\,\frac{(m - m^*)^2}{mm^*} + (ms^* - sm^*)\Big[s^*\delta\,\frac{s - s^*}{ss^*} + m^*\,\frac{m - m^*}{mm^*}\Big] \\ &= -m^*\,\frac{(m - m^*)^2}{mm^*} + (ms^* - sm^*)\Big[s^*\delta\,\big(\frac{1}{s^*} - \frac{1}{s}\big) + m^*\,\big(\frac{1}{m^*} - \frac{1}{m}\big)\Big] \\ &= -m^*\,\frac{(m - m^*)^2}{mm^*} + (ms^* - sm^*)\Big[\frac{s^*\delta}{s}\,\big(\frac{s}{s^*} - 1\big) + \frac{m^*}{m}\big(\frac{m}{m^*} - 1\big)\Big] \\ &= -m^*\,\frac{(m - m^*)^2}{mm^*} + ms\left[\frac{s^*}{s} - \frac{m^*}{m}\right]\Big[\frac{s^*\delta}{s}\,\big(\frac{s}{s^*} - 1\big) + \frac{m^*}{m}\big(\frac{m}{m^*} - 1\big)\Big].\end{aligned}$$

So, we have

$$\frac{dV(m,s)}{dt} = -\frac{m^*(1-s)}{mm^*}\left(m-m^*\right)^2 - \frac{ms^*\delta}{ss^*}\left(s-s^*\right)^2$$
$$= -\frac{(1-s)(m-m^*)^2}{m} - \frac{m\delta\,(s-s^*)^2}{s} \leq 0.$$

Additionally, we obtain

$$\frac{dV(m,s)}{dt} = 0 \iff m = m^* \text{ and } s = s^*.$$

Employing LaSalle's principle, the disease-present equilibrium E^*_{eq} is globally asymptotically stable in Γ.

□

5. A MSEIHR Model Sensitivity

Sensitivity analysis is employed to assess the effectiveness of a model with respect to parameter values, helping us identify which parameters significantly affect the reproduction number R_0. This is particularly important given the potential errors in data collection and the assumptions made about parameter values. Using the method outlined by Chitnis [3], we determine the forward sensitivity indices of R_0. Specifically, we define the sensitivity index as

$$\Upsilon_n^{R_0} = \frac{\partial R_0}{\partial n} * \frac{n}{R_0}.$$

This represents the sensitivity of R_0, relating to the parameter n. Hence, we can write

$$R_0 = \frac{\beta}{\alpha+b+\theta}\,,\ \Upsilon_\beta^{R_0} = 1\,,\ \Upsilon_\alpha^{R_0} = -\frac{\alpha}{\alpha+b+\theta}\,,\ \Upsilon_\theta^{R_0} = -\frac{\theta}{\alpha+b+\theta} \text{ and } \Upsilon_b^{R_0} = -\frac{b}{\alpha+b+\theta}.$$

From this analysis, we see that R_0 is most sensitive to variation in β. An increase in β will result in a proportional increase in R_0, while a decrease in β will cause R_0 to decrease proportionally. Conversely, parameters such as α, b, and θ are inversely related to R_0. Thus, an increase in any of these coefficients will lead to a decrease in R_0.

Parameter	Rates description	index of sensitivity
β	Effective transmission	1
α	Transmission E to I	-0.0833
θ	Propagation E to H	-0.25
b	Natural death	-0.6666

Table 1. Academic parameters for Model 2.1

6. Numerical Simulation and Interpretations

We present the numerical solutions of our model for various parameters, with a total population size of

$$M + S + E + I + H + R = 30000.$$

We start by examining the disease-free equilibrium, using numerical simulation of System 1 to confirm our findings. Specifically, by estimating the parameters as $N = 30000$, $\beta = 0.05$, $\alpha = 0.01$, $\lambda = 0.09$, $\theta = 0.03$, $\delta = 0.09$ and $b = 0.08$, we find that the basic reproduction term is $R_0 = 0.3846 < 1$. Under these conditions, the disease-free equilibrium E^0_{ef} of System 1 is locally asymptotically stable. The following observations are derived from the obtained figures (a)-(e), which use different initial values for the variables M_0, S_0 and E_0, we obtained the following remarks:

1. The population with passive immunity decreases and tends to 0 (see Figure 2).
2. The susceptible population increases and approaches the total population size, $S_0 = 2.10^4$ (see Figure 3).
3. The exposed cases number decreases to 0 (see Figure 4).
4. The number of symptomatic infected persons and carriers decreases to 0 (see Figure 5).
5. The hospitalized number decreases to 0 (see Figure 6).
6. The number of recovered cases initially increases, then decreases to 0 (see Figure 7).

Consequently, the solution curves converge to the equilibrium $(0, S_0, 0)$ when $R_0 < 1$, indicating that the proposed model is locally asymptotically stable.

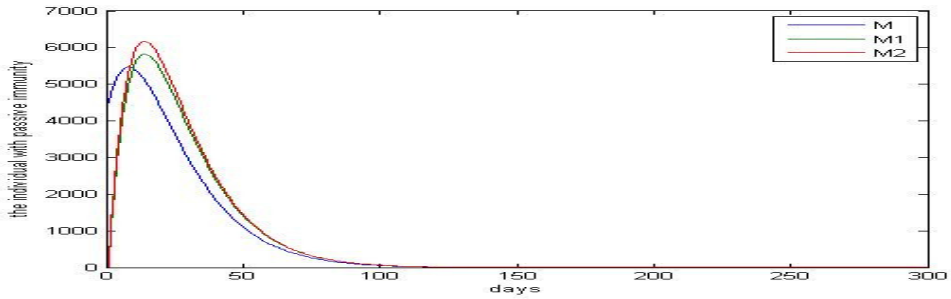

Figure 2

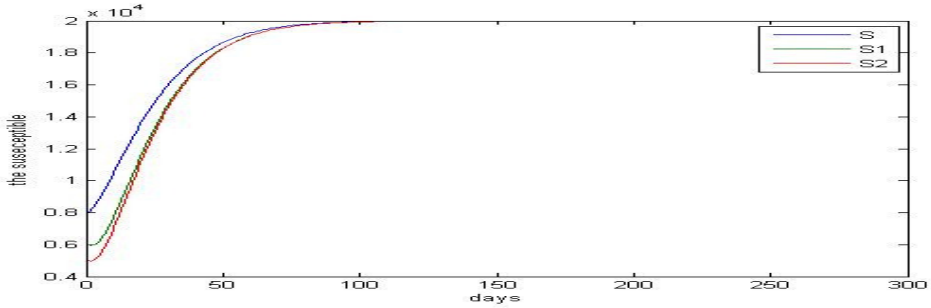

Figure 3

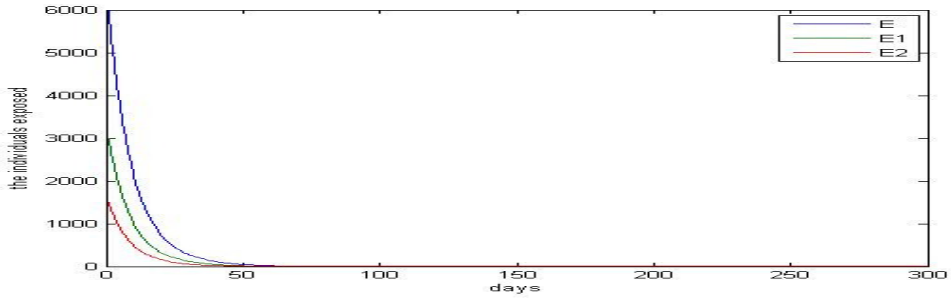

Figure 4

We now turn our attention to the endemic equilibrium point. Considering the parameters $N = 30000$, $\delta = 0.09$, $\beta = 0.7$, $\alpha = 0.01$, $\mu = 0.05$, $\lambda = 0.09$, $\theta = 0.03$, $b = 0.08$ and with $R_0 = 5.3846 > 1$, we find that the equilibrium

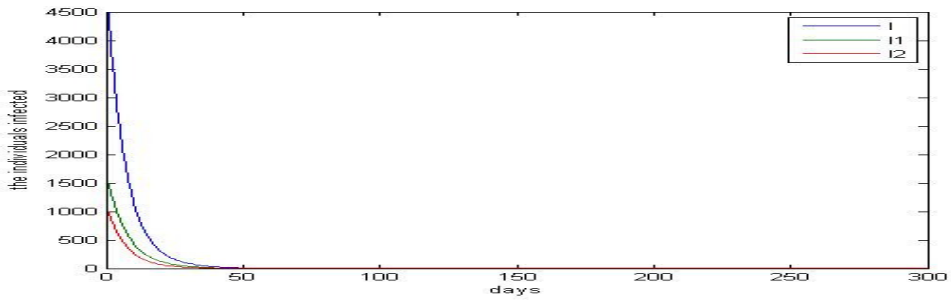

Figure 5

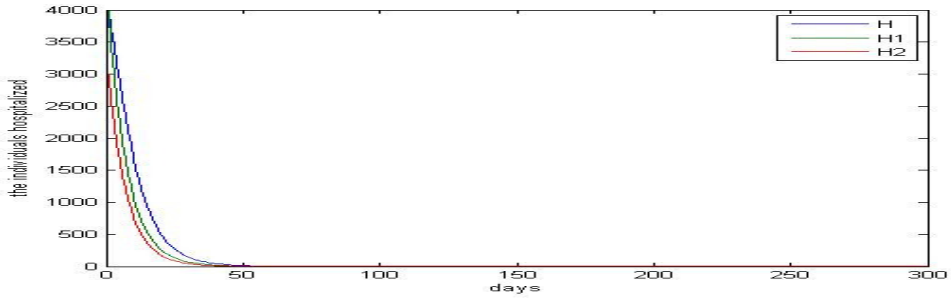

Figure 6

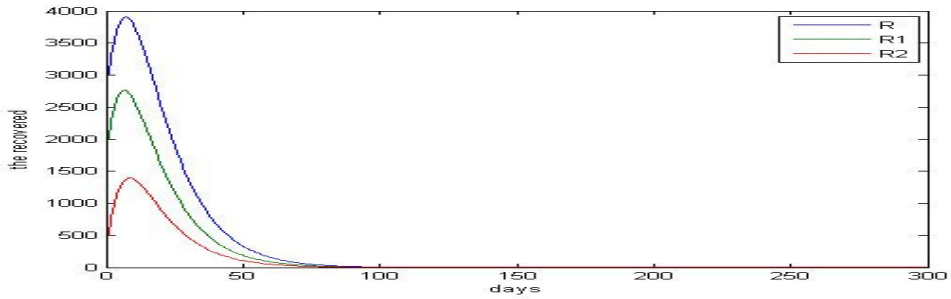

Figure 7

point associated with the influenza (H5N1) disease, E^*_{eq}, in System (1) is globally asymptotically stable. For this scenario, we notice that

1. The population with passive immunity increases and tends to $M^* = 7.5e + 03$ (Figure 8).
2. The susceptible individuals number increases and converges toward the value $S^* = 3.7e + 03$ (Figure 9).
3. The number of exposed cases converges toward the value $E^* = 5.5e + 03$ (Figure 10).
4. The infected individuals number converges toward $I^* = 5.5e + 02$ (Figure 11).
5. The hospitalized individuals number decreases and approaches the value $H^* = 2.5e + 02$ (Figure 12).
6. The recovered individuals number decreases and tends toward the value $R^* = 2.4e + 03$ (Figure 13).

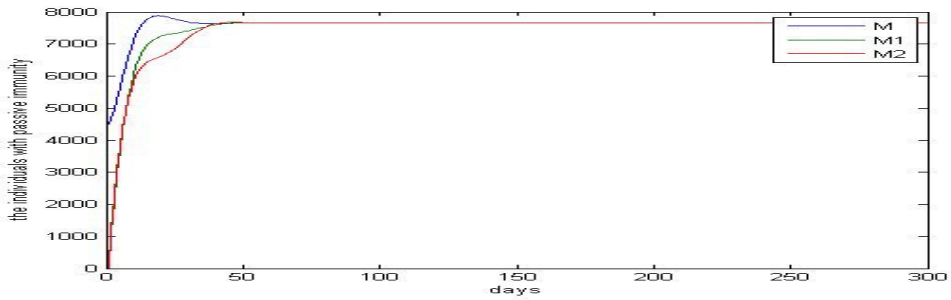

Figure 8

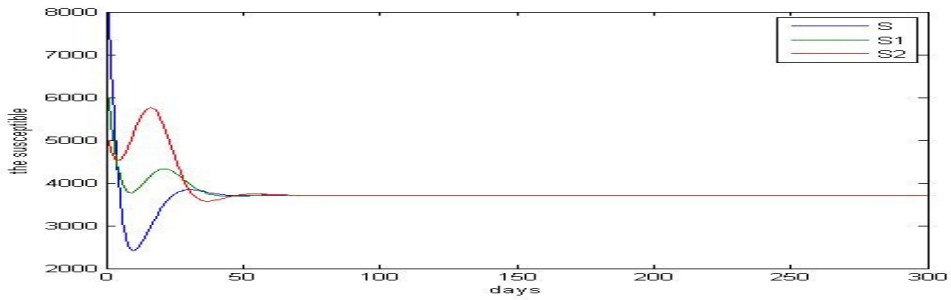

Figure 9

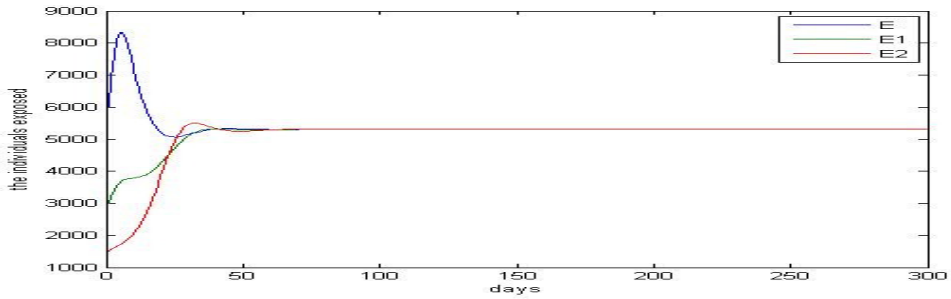

Figure 10

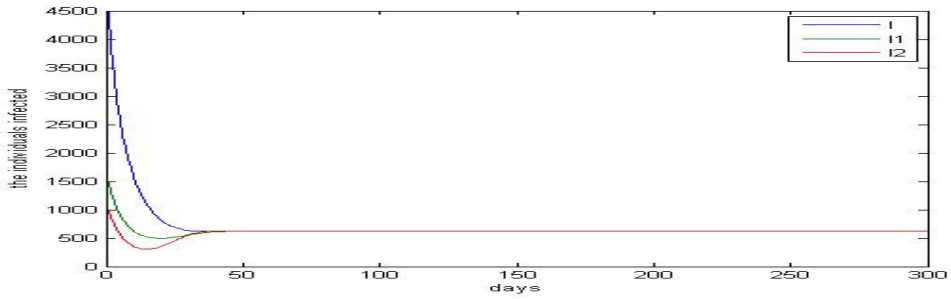

Figure 11

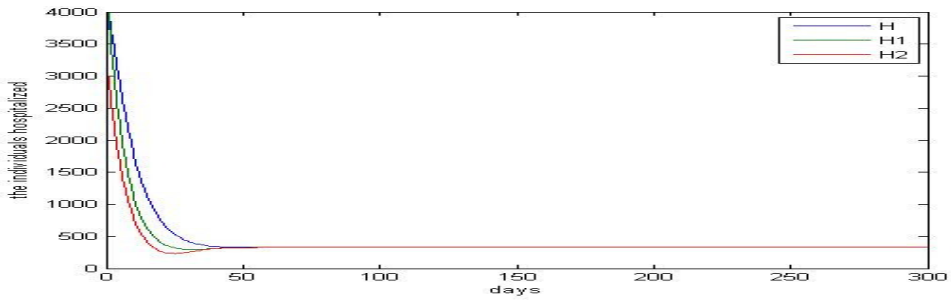

Figure 12

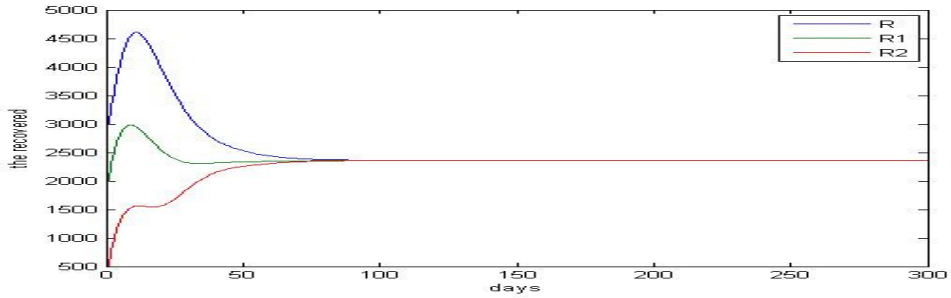

Figure 13

Conclusion

Epidemic models are crucial globally, as they enable health officials to understand disease transmission and formulate strategies for controlling outbreaks. In our study, we designed an appropriate MSEIHR model for influenza (H5N1) and identified the number, R_0, as a principal factor in understanding the disease's spread. Through stability analysis, we investigated the model to assess its local stability.

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