MODELING THE TRANSMISSION DYNAMICS OF AVIAN INFLUENZA WITH SATURATION AND PSYCHOLOGICAL EFFECT

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Abstract. The present paper describes the mathematical analysis of an avian influenza model with saturation and psychological effect. The virus of avian influenza is not only a risk for birds but the population of human is also not safe from this. We proposed two models, one for birds and the other one for human. We consider saturated incidence rate and psychological effect in the model. The stability results for each model that is birds and human is investigated. The local and global dynamics for the disease free case of each model is proven when the basic reproduction number \( R_0 < 1 \) and \( R_0 < 1 \). Further, the local and global stability of each model is investigated in the case when \( R_{0b} > 1 \) and \( R_0 > 1 \). The mathematical results show that the considered saturation effect in population of birds and psychological effect in population of human does not effect the stability of equilibria, if the disease is prevalent then it can affect the number of infected humans. Numerical results are carried out in order to validate the theoretical results. Some numerical results for the proposed parameters are presented which can reduce the number of infective in the population of humans.

1. Introduction. Influenza virus has basically three types: A, B, and C. Virus A is further categorized into two groups the hemagglutinin (H) and the neuraminidase (N) [1]. All the Avian influenza A viruses (H1,H16) and (N1,N9) and all its subtypes mostly isolated from avian species [6]. Naturally, worldwide, these viruses infect
wild aquatic birds, domestic, other bird and animal species. Normally do not infect human population. However, sporadic human infections with bird flu virus has occurred [2].

The viruses of avian influenza are divided into two groups: low and high pathogenic avian influenza A. The other three subtypes of A, which are AH9, AH5, and AH7, which infect humans and birds both. Viruses of (H1-H9) identified in poultry and wild birds worldwide and are of low a pathogenic avian influenza. Rare, Sporadic H9N2 infections to humans have been reported which cause mild upper respiratory tract illness. Worldwide viruses of the type H5 and H7 are identified in poultry and wild birds which are of a low pathogenic avian influenza. From more than 15 countries, its documented the evidence of H5N1 virus in humans, which is most of severe pneumonia with nearly 60% percent of mortality. In human the virus H7 is not common, but the reports shows the evidence of virus, those who have direct in contact with infected birds, in particular during the H7 virus outbreak among poultry.

The low pathogenic avian influenza virus, which includes H7N2, H7N3 and H7N7 in human caused mild to moderate illness and high pathogenic avian influenza, that is H7N3, H7N7 virus infections have caused mild to severe and fatal illness. The early infection of infection H7N9 virus cases in human are first identifier in 2013, which include death and severe respiratory illness [3].

In order to better understand and explore the dynamics of infectious diseases, various mathematical models have been modeled based on their epidemiology properties, and implemented useful control and preventive measure for the disease elimination [10, 27]. Iwami et al. [42] in 2007, formulated a mathematical in ODE that describes the human behavior and avian population. After that, numerous mathematical models are designed for avian influenza H5N1 virus [25, 43, 26, 41, 23, 4, 47, 16, 22, 34] with different aspects. In recent times, the dynamics of influenza A H7N9 virus has been analyzed [50, 48, 49]. Besides this, various approaches have been used to obtain solution to models of infectious diseases [11, 36, 9, 12, 7, 8, 13].

In infectious disease the role of human behavior and social responses gaining an important role [14, 21, 38, 39] as it is the key to improve control efforts. The general public reacts regarding to avian influenza, psychologically, behaviorally and socially. To obtain reasonable knowledge in a general population about the attitudes and practices associated to avian influenza after the outbreak of A H5N1, some survey have been conducted in China and Thailand [45, 35], in both urban and rural populations, that show a significant awareness of human avian influenza and a higher level of proper hygienic practice among urban residents.

The human exposure to live-poultry and changes in risk perception and behavior after the influenza A H7N9 outbreak in 2013 in China, the author’s in [31] visited urban and rural residents and concluded that 77% urban respondents visit less live market after the outbreak. The decrease in visiting the live markets help to reduce the spread of disease further and support the government agencies to make control measure to prevent avian influenza in general population. The human behavior and social responses to the spread of infectious diseases are frequently reported but there is a little systematic study on their effect have been conducted [39, 37, 40]. It is considered difficult in mathematical models to incorporate social and behavior response and how to analyze human behavior and social response [40].

In the transmission of infectious disease the role of incidence rate is the important one. In most of the avian influenza models the incidence rate is considered the
mass action from the bilinear interactions, which is increasing and unbounded. The survey [45, 35] shows that the incidence rate will decreases, if the cases are reported on the media, so that the people then less visiting the markets. The non-monotone incidence function proposed in [33] is a useful to such phenomena, also see [44]. The poultry farmers will be careful by adopting preventive measure and cautions when the number of infected birds grows larger, so that the incidence rate from infective birds to susceptible may saturate, such saturated incoincidence rate is investigated by Capasso and Serio [17]. This incidence function for avian influenza model is used recently in [44].

In this paper, we incorporating the exposed class in both birds and human populations. The nonlinear incidence rate for the infective to susceptible birds is used that describing the saturation effect and a nonmonotone incidence rate from infective birds to susceptible humans that describe the psychological effect in human population.

The organization of the paper follows: Section 2 is devoted to mathematical formulation of the model and discussed their parameters in detail. In section 3, the formulation of the model and discussed their parameters in detail. In section 3, the feasibility of the solution for the proposed model is investigated. In section 3, the birds only model with detail mathematical analysis is presented. The full model with detail mathematical analysis is presented in section 4. In section 5, numerical simulation of the model is presented. Finally, a brief discussion with conclusion made on the presented work is shown in section 6.

2. Model framework. In this section, we formulate the mathematical modeling of avian influenza epidemic. We denote the total population of birds by \( N_a \) and subdivided into \( S_a \)-susceptible birds, exposed birds- \( E_a \) not yet infectious and \( I_a \) the infected birds at any time \( t \), so that \( N_a = S_a + E_a + I_a \). The population of human is denoted by \( N_h \) which is further subdivided into \( S_h \)-susceptible humans, \( E_h \)-exposed humans, \( I_h \)-infected humans and \( R_h \) the recovered humans, so that \( N_h = S_h + E_h + I_h + R_h \). We assume that the virus is not spread between human and mutate, the main source of infection is the domestic birds. All the new bores and recruitment are considered susceptible in each population. Further, the virus is contagious \( I_a \) to \( S_a \) and \( a \) to susceptible human \( S_h \). Moreover, when infective avian becomes larger, the incidence rate between \( I_a \) and \( S_a \) reaches saturation level due to crowding of the infected birds and the protection measures taken by the poultry farmers [17]. Based on the above discussions the avian influenza epidemic model is presented in the following:

\[
\begin{align*}
\frac{dS_a(t)}{dt} &= \Lambda_a - \gamma_a S_a - \frac{\beta_a I_a S_a}{1 + \alpha I_a}, \\
\frac{dE_a(t)}{dt} &= \frac{\beta_a I_a S_a}{1 + \alpha I_a} - \left( \gamma_a + \delta_a \right) E_a, \\
\frac{dI_a(t)}{dt} &= \delta_a E_a - \left( \gamma_a + \mu_a \right) I_a, \\
\frac{dS_h(t)}{dt} &= \Lambda_h - \gamma_h S_h - \frac{\beta_h I_h S_h}{1 + \alpha I_h}, \\
\frac{dE_h(t)}{dt} &= \frac{\beta_h I_h S_h}{1 + \alpha I_h} - \left( \gamma_h + \delta_h \right) E_h, \\
\frac{dI_h(t)}{dt} &= \delta_h E_h - \left( \gamma_h + \theta_h + \psi_h \right) I_h, \\
\frac{dR_h(t)}{dt} &= \psi_h I_h - \gamma_h R_h.
\end{align*}
\]

subject to initial conditions

\( S_a(0) \geq 0, \ E_a(0) \geq 0, \ I_a(0) \geq 0, \ S_h(0) \geq 0, \ E_h(0) \geq 0, \ I_h(0) \geq 0, \ R_h(0) \geq 0. \)
In system (1) the recruitment rate of birds and human population is denoted by \( \Lambda_a \) and \( \Lambda_h \) respectively. The natural mortality rate of birds population is shown by \( \gamma_a \) and the disease related death of infected birds is \( \mu_a \), \( \delta_a \) is the transfer of exposed birds to infected birds. The natural mortality rate of human is \( \gamma_h \) and the disease related death is \( \theta_h \). At a rate \( \delta_h \) the exposed human becomes infected and join \( I_h \). At a rate of \( \psi_h \) the infected human recovered and join \( R_h \). The parameter \( \alpha \) and \( m \) are constants that measure the inhibitions effect and psychological effect constant respectively.

The total dynamics of the birds population is

\[
\frac{dN_a}{dt} = \Lambda_a - \gamma_a N_a - \mu_a I_a
\]  

i. e.,

\[
N'_a(t) + \gamma_a N_a \leq \Lambda_a. 
\]  

The integration of (3) and the use of [15], we get

\[
0 \leq N_a(S_a, E_a, I_a) \leq \frac{\Lambda_a}{\gamma_a} (1 - e^{-\gamma_a t}) + N_a(S_a(0) + E_a(0) + I_a(0)) e^{-\gamma_a t}. 
\]  

Now, taking, \( t \to \infty \), we get \( 0 < N_a \leq \frac{\Lambda_a}{\gamma_a} \). Similarly

The total dynamics of human population is

\[
\frac{dN_h}{dt} = \Lambda_h - \gamma_h N_h - \theta_h I_h 
\]  

Using the same procedure that is for birds population, we obtain

\[
0 \leq N_h(S_h, E_h, I_h, R_h) \leq \frac{\Lambda_h}{\gamma_h} (1 - e^{-\gamma_h t}) + N_h(S_h(0) + E_h(0) + I_h(0) + R_h(0)) e^{-\gamma_h t}. 
\]  

Now, taking, \( t \to \infty \), we get \( 0 < N_h \leq \frac{\Lambda_h}{\gamma_h} \). The closed set

\[
\Omega = \left\{ (S_a, E_a, I_a, S_h, E_h, I_h, R_h) \in R^7_+ : N_a \leq \frac{\Lambda_a}{\gamma_a}, N_h \leq \frac{\Lambda_h}{\gamma_h} \right\} 
\]  

represent a feasible region of the system. The boundedness and positive invariant property is presented in the following proposition.

The closed set \( \Omega \) is bounded and positively invariant.

We know that

\[
\frac{dN_a}{dt} \leq \Lambda_a - \gamma_a N_a, \quad \frac{dN_h}{dt} \leq \Lambda_h - \gamma_h N_h. 
\]  

\( N_a \) is bounded above by \( \frac{\Lambda_a}{\gamma_a} \). Hence \( \frac{dN_a}{dt} < 0 \) whenever \( N_a(t) > \frac{\Lambda_a}{\gamma_a} \). Using an integrating factor, we have

\[
N_a(t) \leq N_a(0) e^{-\gamma_a t} + \frac{\Lambda_a}{\gamma_a} \left( 1 - e^{-\gamma_a t} \right). 
\]  

when \( t \to \infty, e^{-\gamma_a t} \to 0 \) and hence \( \lim_{t \to \infty} N_a(t) \leq \frac{\Lambda_a}{\gamma_a} \).

In similar fashion, we can show the other case. Thus, the closed set \( \Omega \) is bounded and positively invariant.
3. **Analysis of the Avian-only sub model.** In this section, we present the mathematical results for the Avian-only sub-model. The avian only sub-model is:

\[
\begin{align*}
    \frac{dS_a(t)}{dt} &= \Lambda_a - \gamma_a S_a - \frac{\beta_a I_a}{1 + \alpha I_a}, \\
    \frac{dE_a(t)}{dt} &= \frac{\beta_a I_a}{1 + \alpha I_a} - (\gamma_a + \delta_a)E_a, \\
    \frac{dI_a(t)}{dt} &= \delta_a E_a - (\gamma_a + \mu_a)I_a,
\end{align*}
\] (10)

The basic reproduction number of the birds only model (10) is obtain by using the method [20]. It follows

\[
F = \begin{bmatrix} \frac{\beta_a I_a S_a}{1 + \alpha I_a} \\ 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} (\gamma_a + \delta_a)E_a \\ (\gamma_a + \mu_a)I_a - E_a \delta_a \end{bmatrix}.
\] (11)

Thus, \( R_{0b} \) is the basic reproduction number for the avian sub-model (10)

\[
R_{0b} = \frac{\beta_a \delta_a \Lambda_a}{\gamma_a (\gamma_a + \mu_a)(\gamma_a + \delta_a)}.
\] (12)

The disease free equilibrium of the avian sub-model only (10), denoted by \( E_{0b} \) and is given by

\[
E_{0b} = \left( S^0_a, 0, 0 \right) = \left( \frac{\Lambda_a}{\gamma_a}, 0, 0 \right).
\] (13)

The endemic equilibrium of the avian sub-model only (10) is denoted by \( E^*_a \) and is given by

\[
\begin{align*}
    S^*_a &= \frac{\Lambda_a (1 + \alpha I^*_a)}{(\gamma_a (1 + \alpha I^*_a) + \beta_a I^*_a)}, \\
    E^*_a &= \frac{\Lambda_a \beta_a I^*_a}{(\gamma_a + \delta_a)(\gamma_a (1 + \alpha I^*_a) + \beta_a I^*_a)}, \\
    I^*_a &= \frac{\gamma_a}{\alpha \gamma_a + \beta_a} (R_{0b} - 1).
\end{align*}
\] (14)

If \( R_{0b} > 1 \) then a positive endemic equilibrium exists.

3.1. **Local stability of avian sub-model only.**

**Theorem 3.1.** The avian only sub-model (10) at \( E_{0b} \) is locally asymptotically stable if \( R_{0b} < 1 \).

**Proof.** The jacobian matrix \( J_0 \) of avian only sub-model (10), evaluated at DFE, \( E_{0b} \) is given by

\[
J_0 = \begin{bmatrix} -\gamma_a & 0 & -\beta_a S^0_a \\ 0 & -\gamma_a - \delta_a & \beta_a S^0_a \\ 0 & \delta_a & -\gamma_a - \mu_a \end{bmatrix}.
\] (15)

In \( J_0 \), clearly, one of the eigenvalue \(-\gamma_a < 0\) and the other two roots can be obtained through the following equation:

\[
x^2 + x((\gamma_a + \mu_a) + (\gamma_a + \delta_a)) + (\gamma_a + \mu_a)(\gamma_a + \delta_a)(1 - R_{0b}) = 0.
\] (16)

Thus, the disease free equilibrium of the avian sub-model only (10) is stable locally asymptotically, whenever, \( R_{0b} < 1 \).

Next, we present the local stability of the avian sub-model (10) at \( E^*_a \).
The time derivative of $V$ globally asymptotically, whenever, The sub only model (10) at disease free equilibrium Theorem 3.3. of the sub only model (10). We have the following results: Proof. The Jacobian matrix $J_1$ evaluated at $E_0^*$ of the avian sub-model (10) is

$$J_1 = \begin{bmatrix} -\gamma_a - \frac{\beta_a I_a^*}{1 + \alpha I_a^*} & 0 & -\frac{\beta_a S_a^*}{(1 + \alpha I_a^*)^2} \\ \frac{\beta_a I_a^*}{1 + \alpha I_a^*} & -(\gamma_a + \delta_a) & \frac{\beta_a S_a^*}{(1 + \alpha I_a^*)^2} \\ 0 & \delta_a & -(\gamma_a + \mu_a) \end{bmatrix} \quad (17)$$

The associated characteristic equation of $J_1$ is

$$\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3 = 0, \quad (18)$$

where,

$$c_1 = 3\gamma_a + \delta_a + \frac{\beta_a I_a^*}{1 + \alpha I_a^*} + \mu_a,$n$$

$$c_2 = \left(\frac{\gamma_a + \delta_a}{1 + \alpha I_a^*}\right) (\gamma_a + I_a^* (\alpha\gamma_a + \beta_a) + (\gamma_a + \mu_a) (\alpha I_a^* + 1)) - \frac{\beta_a \delta_a S_a^*}{(1 + \alpha I_a^*)^2},$$

$$c_3 = \frac{\alpha I_a^2 (\gamma_a + \delta_a) (\gamma_a + \mu_a) (\alpha\gamma_a + \beta_a) + I_a^* (\gamma_a + \delta_a) (\gamma_a + \mu_a) (2\alpha\gamma_a + \beta_a)}{(1 + \alpha I_a^*)^2}$$

$$+ \frac{\gamma_a ((\gamma_a + \delta_a) (\gamma_a + \mu_a) - \beta_a \delta_a S_a^*)}{(\alpha I_a^* + 1)^2}. \quad (19)$$

The Routh Hurtwiz criteria $c_i > 0$, for $i = 1, 2, 3$ and $c_1c_2 - c_3 > 0$, could be easily verified. Thus, the birds only model (10) at endemic equilibrium $E_0^*$ is locally asymptotically stable if $R_{0b} > 1$ and the Routh-Hurtwiz criteria satisfy. □

3.2. Global stability. The aims of this section is to present the global dynamics of the sub only model (10). We have the following results:

Theorem 3.3. The sub only model (10) at disease free equilibrium $E_{0b}$ is stable globally asymptotically, whenever, $R_{0b} < 1.$

Proof. We define the following Lyapunov function:

$$V(S_a, E_a, I_a) = \delta_a \left( S_a - S_a^0 - S_a^0 \log \frac{S_a}{S_a^0} \right) + \delta_a E_a + (\delta_a + \delta_a) I_a. \quad (20)$$

The time derivative of $V$, along the solution of the model (10) is

$$V' = \delta_a \left( S_a - S_a^0 \right) S_a' + \delta_a E_a' + (\gamma_a + \delta_a) I_a',$$

$$= \delta_a \left( S_a - S_a^0 \right) \left[ \Lambda_a - \gamma_a S_a - \frac{\beta_a S_a I_a}{1 + \alpha I_a} \right] + \delta_a \left[ \frac{\delta_a S_a I_a}{1 + \alpha I_a} - (\gamma_a + \delta_a) E_a \right] + (\gamma_a + \delta_a) \left[ \delta_a E_a - (\gamma_a + \mu_a) I_a \right] \quad (21)$$

$$V' = I_a (\gamma_a + \mu_a) (\gamma_a + \delta_a) \left( R_{0b} - 1 \right).$$
V(t) is negative for $R_{0b} < 1$ and zero if $I_a = 0$. Consequently, the largest compact invariant set in $\{(S_a, E_a, I_a) \in \Omega|V'(t) = 0\}$, when $R_{0b} < 1$, is the singleton set $E_{0b}$. Hence by Lasalle’s Invariance Principle [28], $E_{0b}$ is globally asymptotically stable in $\Omega$.

3.3. Global stability of endemic equilibrium. In order to show the global stability of (10), we follow the results in [24, 30, 5].

**Theorem 3.4.** If $R_{0b} > 1$, then the endemic equilibrium $E^*_b$ of the system (10) is globally asymptotically stable.

**Proof.** At a steady state the avian only model (10) at $E^*_b$, gives

$$
\begin{align*}
\Lambda_a &= \gamma_a S_a^* + \frac{\beta_a S_a^* E_a^*}{1 + \alpha I_a^*}, \\
E_a^* &= \frac{(\gamma_a + \mu_a) I_a^*}{\delta_a}, \\
(\gamma_a + \delta_a) E_a^* &= \frac{\beta_a S_a^* I_a^*}{1 + \alpha I_a^*}, \\
(\gamma_a + \delta_a)(\gamma_a + \mu_a) I_a^* &= \frac{\delta_a S_a^* I_a^*}{1 + \alpha I_a^*}.
\end{align*}
$$

(22)

Now, we define the following Lyapunov function

$$
V(t) = (S_a - S_a^* - S_a^* \log \frac{S_a}{S_a^*}) + \left(E_a - E_a^* - E_a^* \log \frac{E_a}{E_a^*}\right) + \frac{\gamma_a + \delta_a}{\delta_a} \left(I_a - I_a^* - I_a^* \log \frac{I_a}{I_a^*}\right).
$$

(23)

Calculating the time derivative of $V$ along the solutions of the system (10), we obtain

$$
V'(t) = (1 - \frac{S_a^*}{S_a}) S'_a + (1 - \frac{E_a^*}{E_a}) E'_a + (1 - \frac{I_a^*}{I_a}) I'_a.
$$

(24)

By direct calculations, we have that:

$$
\begin{align*}
\left(1 - \frac{S_a^*}{S_a}\right) S_a' &= \left(1 - \frac{S_a^*}{S_a}\right) \left[\Lambda_a - \gamma_a S_a - \frac{\beta_a S_a I_a}{1 + \alpha I_a}\right], \\
&= \left(1 - \frac{S_a^*}{S_a}\right) \left[\gamma_a S_a^* + \frac{\beta_a S_a I_a^*}{1 + \alpha I_a^*} - \gamma_a S_a - \frac{\beta_a S_a I_a}{1 + \alpha I_a}\right], \\
&= \gamma_a S_a^* \left(2 - \frac{S_a^*}{S_a}\right) - \frac{S_a^*}{S_a} + \left(1 - \frac{S_a^*}{S_a}\right) \left[\frac{\beta_a S_a I_a^*}{1 + \alpha I_a^*} - \frac{\beta_a S_a I_a}{1 + \alpha I_a}\right]
\end{align*}
$$

(25)

$$
\begin{align*}
\left(1 - \frac{E_a^*}{E_a}\right) E_a' &= \left(1 - \frac{E_a^*}{E_a}\right) \left[\frac{\beta_a I_a S_a}{1 + \alpha I_a} - \left(\gamma_a + \delta_a\right) E_a\right], \\
&= \left(1 - \frac{E_a^*}{E_a}\right) \left[\frac{\beta_a I_a S_a}{1 + \alpha I_a} - \frac{\beta_a I_a S_a^*}{1 + \alpha I_a^*} E_a\right], \\
&= \frac{\beta_a I_a S_a}{1 + \alpha I_a} - \frac{\beta_a I_a S_a^*}{1 + \alpha I_a^*} E_a + \frac{\beta_a I_a S_a^*}{1 + \alpha I_a^*},
\end{align*}
$$

(26)
\[
\frac{(\gamma_a + \delta_a)}{\delta_a} (1 - \frac{I_a^*}{I_a}) I_a^* = \frac{(\gamma_a + \delta_a)}{\delta_a} (1 - \frac{I_a^*}{I_a}) [\delta_a E_a - (\gamma_a + \mu_a) I_a],
\]
\[
= (\gamma_a + \delta_a) (1 - \frac{I_a^*}{I_a}) E_a - \frac{(\gamma_a + \delta_a)}{\delta_a} (\gamma_a + \mu_a) \times \frac{I_a^*}{I_a}
\]
\[
= \frac{\beta_a S_a^* I_a^*}{1 + \alpha I_a} \frac{E_a}{E_a} - \frac{1}{1 + \alpha I_a} \beta_a S_a^* I_a^* E_a - \frac{1}{1 + \alpha I_a} \beta_a S_a^* I_a^* + \beta_a S_a I_a^*
\]
\[
\frac{1}{1 + \alpha I_a}.
\]

(27)

It follows from (25-27),
\[
V'(t) = \gamma_a S_a^* \left( 2 - \frac{S_a^*}{S_a} - \frac{S_a}{S_a^*} \right) + \frac{\beta_a S_a^* I_a^*}{1 + \alpha I_a} \left( 3 - \frac{S_a^*}{I_a} - \frac{I_a^*}{I_a} \right) E_a
\]
\[
+ \frac{\beta_a S_a I_a}{1 + \alpha I_a} \left( 1 - \frac{S_a}{S_a^*} \right).
\]

(28)

In equation (28),
\[
\left( 2 - \frac{S_a^*}{S_a} - \frac{S_a}{S_a^*} \right) \leq 0,
\]
\[
\left( 3 - \frac{S_a^*}{I_a} - \frac{I_a^*}{I_a} \right) E_a \leq 0,
\]
\[
\left( \frac{S_a}{S_a^*} \frac{E_a}{E_a} - 1 \right) \leq 0.
\]

(29)

Thus, the condition (22) ensures that \( V'(t) \leq 0 \) for all \((S_a, E_a, I_a) \in \Omega\), and the strict equality \( V'(t) = 0 \) holds only for \( S_a = S_a^* \), \( E_a = E_a^* \) and \( I_a = I_a^* \). Then, the equilibrium state \( E_b^* \) is the only positively invariant set of the system (10) contained entirely in \( \Omega = (S_a, E_a, I_a) \), \( S_a = S_a^* \), \( E_a = E_a^* \) and \( I_a = I_a^* \) and hence by the asymptotic stability theorem [29], the positive endemic equilibrium state \( E_b^* \) is globally asymptotically stable on \( \Omega \). \( \square \)

4. Analysis of the influenza full epidemic model. In this section, we describe the mathematical analysis of the full model (1). First, we find the basic reproduction number of the full model (1), that is necessary for onward analysis.

4.1. Basic reproduction number. The basic reproduction number of the full model (1) is obtained by using the method in [20]. It follows that,
\[
F = \begin{pmatrix}
\frac{\beta_a S_a}{1 + \alpha I_a} & 0 \\
0 & \frac{\beta_b S_a}{1 + \alpha I_a} \\
\frac{\beta_a I_a S_a}{1 + \alpha I_a} & 0
\end{pmatrix}
\] and \( V = \begin{pmatrix}
(\gamma_a + \delta_a) & E_a \\
(\gamma_a + \mu_a) & I_a - E_a \delta_a \\
(\gamma_h + \delta_h) E_h & I_a - E_a \delta_a
\end{pmatrix}. \)

(30)
So,

\[
F = \begin{pmatrix}
0 & \frac{\beta a S_a}{(\alpha I_a + 1)^2} & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & \frac{\beta a S_a}{m I_a + 1} & 0 & -\frac{\beta h S_a I_a (2mI_h)}{(m I_h + 1)^2} \\
0 & 0 & 0 & 0 \\
\end{pmatrix}, \text{ and (31)}
\]

\[
V = \begin{pmatrix}
\gamma_a + \delta_a & 0 & 0 & 0 \\
-\delta_a & \gamma_a + \mu_a & 0 & 0 \\
0 & 0 & \gamma_h + \delta_h & 0 \\
0 & 0 & -\delta_h & \gamma_h + \theta_h + \psi_h \\
\end{pmatrix}. \quad (32)
\]

Thus, the spectral radius \(\rho(FV^{-1})\), is the basic reproduction number \(R_0\) of the system (1) and is given by

\[
R_0 = \frac{\beta a \delta_a \Lambda_a}{\gamma_a (\gamma_a + \delta_a) (\gamma_a + \mu_a)}. \quad (33)
\]

### Equilibrium points.

The disease free equilibrium of the system (1) denoted by \(E_0\) and is given by

\[
E_0 = (S^0_a, 0, 0, S^0_h, 0, 0, 0) = \left(\frac{\Lambda_a}{\gamma_a}, 0, 0, \frac{\Lambda_h}{\gamma_h}, 0, 0, 0\right). \quad (34)
\]

The endemic equilibrium of the full model (1) denoted by \(E_1\) and is given by

\[
E_1 = (S^*_a, E^*_a, I^*_a, S^*_h, E^*_h, I^*_h, R^*_h), \quad (35)
\]

where

\[
\begin{align*}
S^*_a &= \frac{\Lambda_a (1 + \alpha I^*_a)}{(\gamma_a (1 + \alpha I^*_a) + \beta_a I^*_a)} \\
E^*_a &= \frac{\Lambda_a \beta_a I^*_a}{(\gamma_a + \delta_a) (\gamma_a (1 + \alpha I^*_a) + \beta_a I^*_a)} \\
I^*_a &= \frac{\gamma_a (R_0 - 1)}{\alpha \gamma_a + \beta_a} \\
S^*_h &= \frac{\Lambda_h (1 + m I^*_h)}{\gamma_h (1 + m I^*_h) + \beta_h I^*_a} \\
E^*_h &= \frac{\beta_h I^*_h \Lambda_h}{(\gamma_h + \delta_h) (\gamma_h (1 + m I^*_h) + \beta_h I^*_h)} \\
R^*_h &= \frac{\psi_h I^*_h}{\gamma_h}.
\end{align*}
\]

### 4.2. Local stability of the full model.

**Theorem 4.1.** The full model (1) is locally asymptotically stable if \(R_0 < 1\).

**Proof.** The jacobian matrix \(J(E_0)\) evaluated at \(E_0\) of the model (1) is given by

\[
J(E_0) = \begin{pmatrix}
-\gamma_a & 0 & -\frac{\beta a \Lambda_a}{\gamma_a} & 0 & 0 & 0 & 0 \\
0 & -p_1 & \frac{\beta a \Lambda_a}{\gamma_a} & 0 & 0 & 0 & 0 \\
0 & \delta_a & -p_2 & 0 & 0 & 0 & 0 \\
0 & 0 & -\frac{\beta a \Lambda_a}{\gamma_a} & -\gamma_h & 0 & 0 & 0 \\
0 & 0 & \frac{\beta a \Lambda_a}{\gamma_a} & 0 & -p_3 & 0 & 0 \\
0 & 0 & 0 & 0 & \delta_h & -p_4 & 0 \\
0 & 0 & 0 & 0 & 0 & \psi_h & -\gamma_h
\end{pmatrix}. \quad (37)
\]
where \( p_1 = \gamma_a + \delta_a, p_2 = \gamma_a + \mu_a, p_3 = \gamma_h + \delta_h, p_4 = \gamma_h + \theta_h + \psi_h \). The five eigenvalues of the \( J(E_0) \) are clearly negative, that is \(-\gamma_a, -\gamma_h, -p_3, -p_4 \) and \(-\gamma_h\).

The remaining eigenvalues can obtained through the following equation,

\[
y^2 + (p_1 + p_2)y + p_1p_2(1 - R_0) = 0.
\]

if \( R_0 < 1 \), then, the quadratic equation above will give two eigenvalue with negative real parts. Thus, the model (1) at disease free equilibrium \( E_0 \), has eigenvalues with negative real parts if \( R_0 < 1 \). Therefore the system (1) at \( E_0 \) is locally asymptotically stable if \( R_0 < 1 \).

**4.3. Endemic equilibrium of the full model.** In this section, we present the endemic equilibrium of the full model (1) at \( E_1 \). We have the following result:

**Theorem 4.2.** The endemic equilibrium \( E_1 \) of the model (1) is locally asymptotically stable if \( R_0 > 1 \).

**Proof.** The evaluation of the Jacobian matrix at \( E_1 \), gives

\[
J_2 = \begin{pmatrix}
-L_1 & 0 & -L_3 & 0 & 0 & 0 \\
L_2 & -p_1 & L_3 & 0 & 0 & 0 \\
0 & \delta_a & -p_2 & 0 & 0 & 0 \\
0 & 0 & -L_4 & -L_5 & 0 & L_7 \\
0 & 0 & 0 & 0 & \delta_h & -p_4 \\
0 & 0 & 0 & 0 & 0 & \delta_h \\
\end{pmatrix}
\]

where

\[
L_1 = \gamma_a + \frac{\beta_aI^*_a}{1 + \alpha I^*_a}, \quad L_2 = \frac{\beta_aI^*_a}{1 + \alpha I^*_a}, \quad L_3 = \frac{\beta_aS^*_a}{(1 + \alpha I^*_a)^2}, \quad L_4 = \frac{\beta_hS^*_h}{1 + mI^*_h},
\]

\[
L_5 = \frac{\beta_hI^*_a}{1 + mI^*_h} + \gamma_h, \quad L_6 = \frac{I^*_a\delta_h}{1 + mI^*_h}, \quad L_7 = \frac{2mI^*_a\beta_hI^*_hS^*_h}{(1 + mI^*_h)^2},
\]

\[
p_1 = \gamma_a + \delta_a, \quad p_2 = \gamma_a + \mu_a, \quad p_3 = \gamma_h + \delta_h, \quad p_4 = \gamma_h + \theta_h + \psi_h.
\]

The associated characteristic equation of \( J_2 \) is

\[
(\lambda + \gamma_a)(\lambda^6 + k_1\lambda^5 + k_2\lambda^4 + k_3\lambda^3 + k_4\lambda^2 + k_5\lambda + k_6) = 0,
\]

where,

\[
k_1 = L_1 + L_5 + p_1 + p_2 + p_3 + p_4,
\]

\[
k_2 = -L_3\delta_a + L_7\delta_h + L_5(p_1 + p_2 + p_3 + p_4) + L_1(L_5 + p_1 + p_2 + p_3 + p_4) + p_2p_3 + p_1(p_2 + p_3) + (p_1 + p_2 + p_3) p_4,
\]

\[
k_3 = L_5(-L_3\delta_a + L_7\delta_h + p_3p_4 + p_2(p_3 + p_4) + p_1(p_2 + p_3 + p_4)) + L_3(L_2\delta_a - p_4\delta_a - p_3\delta_a) + L_1(-L_3\delta_a + L_7\delta_h + L_5(p_1 + p_2 + p_3 + p_4)) + L_1(p_2p_3 + (p_2 + p_3)p_4 + p_1(p_2 + p_3 + p_4)) + L_7\delta_h(-L_6 + p_1 + p_2 + p_3p_4 + p_1(p_3p_4 + p_2(p_3 + p_4)),
\]

\[
k_4 = -L_7\delta_h(L_3\delta_a + L_6(p_1 + p_2) - p_3p_4) + L_5(L_3\delta_a(L_2 - p_3 - p_4)) + L_5(p_2(L_7\delta_h + p_3p_4) + L_5p_1((L_7\delta_h + p_3p_4 + p_2(p_3 + p_4)))
\]
It can easily prove that all \( k_i > 0 \) for \( i = 1, 2, \ldots, 7 \) where

\[
H_1 = k_1, \quad H_2 = \begin{pmatrix} k_1 & 1 & 0 \\ k_3 & k_2 & k_1 \\ 0 & 0 & k_3 \end{pmatrix}, \quad H_3 = \begin{pmatrix} k_1 & 1 & 0 \\ k_3 & k_2 & k_1 \\ 0 & 0 & k_3 \end{pmatrix},
\]

\[
H_4 = \begin{pmatrix} k_1 & 1 & 0 & 0 \\ k_3 & k_2 & k_1 & 0 \\ 0 & k_4 & k_3 & k_2 \\ 0 & 0 & 0 & k_4 \end{pmatrix}, \quad H_5 = \begin{pmatrix} k_1 & 1 & 0 & 0 \\ k_3 & k_2 & k_1 & 0 \\ k_5 & k_4 & k_3 & k_2 \\ 0 & 0 & k_5 & k_4 \\ 0 & 0 & 0 & 0 & k_5 \end{pmatrix},
\]

\[
H_6 = \begin{pmatrix} k_1 & 1 & 0 & 0 & 0 \\ k_3 & k_2 & k_1 & 1 & 0 \\ k_5 & k_4 & k_3 & k_2 & 1 \\ 0 & k_6 & k_5 & k_4 & k_3 \\ 0 & 0 & 0 & k_6 & k_5 \\ 0 & 0 & 0 & 0 & k_6 \end{pmatrix}.
\]

Here, if all \( k_i > 0 \) for \( i = 1, 2, \ldots, 6 \) and the conditions in \( (H_1 - H_6) \) satisfies, then the theorem above ensure the local endemic stability of the endemic equilibrium of the model (1).

\[\square\]

4.4. GAS disease free case.

**Theorem 4.3.** If \( R_0 < 1 \), then the disease-free equilibrium point \( E_0 \) of the full model (1) is globally asymptotically stable.

**Proof.** To show this result, we use the method [18]. To do this, let \( X = (S_a, S_h) \) and \( Z = (E_a, I_a, E_h, I_h) \) and the full model (10) without \( R_h \) can then be re-written as

\[
\frac{dX}{dt} = F(X, Z),
\]

\[
\frac{dZ}{dt} = G(X, Z)
\]

(41)
where
\[
F(X,Z) = \begin{pmatrix}
\Lambda_a - \gamma_a S_a - \frac{\beta_a I_a S_a}{1 + \alpha I_a} \\
\Lambda_h - \gamma_h S_h - \frac{\beta_h I_h S_h}{1 + m I_h}
\end{pmatrix}
\] (42)
and
\[
G(X,Z) = \begin{pmatrix}
\frac{\beta_a I_a S_a}{1 + \alpha I_a} - \left(\gamma_a + \delta_a\right) E_a \\
\delta_a E_a - \left(\gamma_a + \mu_a\right) I_a \\
\frac{\beta_h I_h S_h}{1 + m I_h} - \left(\gamma_h + \delta_h\right) E_h \\
\delta_h E_h - \left(\gamma_h + \theta_h + \psi_h\right) I_h
\end{pmatrix}
\]. (43)

Consider the reduced system
\[
\frac{dX}{dt} \bigg|_{Z=0} = \begin{pmatrix}
\Lambda_a - \gamma_a S_a \\
\Lambda_h - \gamma_h S_h
\end{pmatrix}
\]. (44)

Clearly, \(X^* = (\frac{\Lambda_a}{\gamma_a}, \frac{\Lambda_h}{\gamma_h})\) is a globally asymptotically stable equilibrium point of (44). It can be obtained very easily that the solution of (44), namely \(S_a(t)\) and \(S_h(t)\) converge to \(X^*\), as \(t \to \infty\) implying the global convergence of solution of (44) in \(\Omega\) and \(S_h(t)\) converges to \(X^*\), as \(t \to \infty\) implying the global convergence of solution of (44) in \(\Omega\).

Let
\[
L = \begin{pmatrix}
-(\gamma_a + \delta_a) & -\frac{\beta_a S_a^0}{1 + \alpha I_a} & 0 & 0 \\
\delta_a & -(\gamma_a + \mu_a) & 0 & 0 \\
0 & -\delta_h & -(\gamma_h + \delta_h) & \beta_h S_h^0 I_a \\
0 & 0 & -\delta_h & -(\gamma_h + \theta_h + \psi_h)
\end{pmatrix},
\] (45)
then, \(G(X,Z)\) can be rewritten as \(G(X,Z) = LZ - \hat{G}(X,Z)\), where
\[
\hat{G}(X,Z) = \begin{pmatrix}
\beta_a I_a \left(S_a^0 - \frac{S_a}{1 + \alpha I_a}\right) \\
0 \\
\beta_h I_h \left(S_h^0 - \frac{S_h}{1 + I_h (1 + m I_h)}\right) \\
0
\end{pmatrix}
\]. (46)

Obviously, the conditions given in [18] are satisfied, that is \(G(X,0) = 0\) and \(G(X,Z) = LZ - \hat{G}(X,Z), \hat{G}(X,Z) \geq 0\). Thus, the model (10) at the DFE is stable globally asymptotically.
4.5. Global stability of endemic equilibrium. In this subsection, we give the global stability of the full model (1) at $E_1$. We present the following theorem by following [30]:

**Theorem 4.4.** If $R_0 > 1$, then, the endemic equilibrium $E_1$ of the system (1) is globally asymptotically stable on $\Omega$.

**Proof.** In a steady state the model (1) at $E_1$ gives

\[
\begin{align*}
\Lambda_a &= \gamma_a S_a^* + \frac{\beta_a S_a^* I_a^*}{1 + \alpha I_a^*}, \\
(\gamma_a + \delta_a)E_a^* &= \frac{\beta_a S_a^* I_a^*}{1 + \alpha I_a^*}, \\
(\gamma_a + \delta_a)(\gamma_a + \mu_a)I_a^* &= \frac{\beta_a S_a^* I_a^*}{1 + \alpha I_a^*}. \\
(\gamma_a + \delta_a)E_a^* &= \frac{\beta_a S_a^* I_a^*}{1 + \alpha I_a^*}.
\end{align*}
\]

Now, we define the Lyapunov function

\[
V(t) = \left( S_a^* - S_a^* S_a^* \log \frac{S_a^*}{S_a^*} \right) + \left( E_a^* - E_a^* - E_a^* \log \frac{E_a^*}{E_a^*} \right) + \frac{\gamma_a + \delta_a}{\delta_a} \left( I_a^* - I_a^* - I_a^* \log \frac{I_a^*}{I_a^*} \right) + \frac{\gamma_a + \delta_a}{\delta_a} \left( I_h^* - I_h^* - I_h^* \log \frac{I_h^*}{I_h^*} \right).
\]

Calculating the time derivative of $V$ along the solutions of the system (10), we obtain,

\[
V'(t) = (1 - \frac{E_a^*}{E_a^*}) E_a^* + \frac{\gamma_a + \delta_a}{\delta_a} (1 - \frac{I_a^*}{I_a^*}) I_a^* + (1 - \frac{S_a^*}{S_a^*}) S_h^* + (1 - \frac{E_h^*}{E_h^*}) E_h^* + \frac{\gamma_a + \delta_a}{\delta_a} (1 - \frac{I_h^*}{I_h^*}) I_h^*.
\]

By direct calculations, we have that:

\[
(1 - \frac{S_a^*}{S_a^*}) S_a^* = (1 - \frac{S_a^*}{S_a^*}) \left[ \Lambda_a - \gamma_a S_a - \frac{\beta_a S_a I_a}{1 + \alpha I_a} \right],
\]

\[
= \left( 1 - \frac{S_a^*}{S_a^*} \right) \left[ \gamma_a S_a^* + \frac{\beta_a S_a^* I_a^*}{1 + \alpha I_a^*} - \gamma_a S_a - \frac{\beta_a S_a I_a}{1 + \alpha I_a} \right],
\]

\[
= \gamma_a S_a^* \left( 2 - \frac{S_a^*}{S_a^*} \right) + \left( 1 - \frac{S_a^*}{S_a^*} \right) \left[ \frac{\beta_a S_a^* I_a^*}{1 + \alpha I_a^*} - \frac{\beta_a S_a I_a}{1 + \alpha I_a} \right],
\]

\[
\left( 1 - \frac{E_a^*}{E_a^*} \right) E_a^* = \left( 1 - \frac{E_a^*}{E_a^*} \right) \left[ \frac{\beta_a S_a I_a}{1 + \alpha I_a} - (\gamma_a + \delta_a) E_a \right],
\]

\[
= \left( 1 - \frac{E_a^*}{E_a^*} \right) \left[ \frac{\beta_a S_a I_a}{1 + \alpha I_a} - \frac{\beta_a S_a I_a}{1 + \alpha I_a} \right].
\]
\[
\begin{align*}
\frac{\gamma_a + \delta_a}{\delta_a} (1 - \frac{I_a^*}{I_a}) I_a' &= \frac{(\gamma_a + \delta_a)}{\delta_a} (1 - \frac{I_a^*}{I_a})[\delta_a E_a - (\gamma_a + \mu_a)I_a], \\
&= \frac{(\gamma_a + \delta_a)}{\delta_a} (1 - \frac{I_a^*}{I_a})E_a \\
&= \frac{\beta_a S_a I_a}{1 + \alpha I_a} - \frac{\beta_a S_a I_a I_a^*}{1 + \alpha I_a} E_a - \frac{\beta_a S_a^2 I_a^* E_a}{1 + \alpha I_a} + \frac{\beta_a S_a^2 I_a^*}{1 + \alpha I_a}, \quad (51)
\end{align*}
\]

\[
\begin{align*}
(1 - \frac{S_a^*}{S_a}) S_a' &= (1 - \frac{S_a^*}{S_a})[\Lambda_a - \gamma_a S_h - \frac{\beta_a S_a I_a}{1 + m(I_h)^2}], \\
&= (1 - \frac{S_a^*}{S_a})[\gamma_a S_a^* + \frac{\beta_a S_a I_a^*}{1 + m(I_h)^2}] - \gamma_a S_h - \frac{\beta_a S_a I_a}{1 + m(I_h)^2}], \\
&= \gamma_a S_a^* \left(2 - \frac{S_a^*}{S_a} - \frac{S_h}{S_a^*} \right) + \left(1 - \frac{S_a^*}{S_a^*} \right) \left(1 - \frac{S_a^*}{S_a^*} \right) \left(1 - \frac{S_h}{S_a^*} \right) \\
&= \frac{\beta_a S_a I_a}{1 + \alpha I_a} E_a - \frac{\beta_a S_a^2 I_a^*}{1 + \alpha I_a} E_a - \frac{\beta_a S_a^2 I_a^*}{1 + \alpha I_a} I_a \\
&= \frac{\beta_a S_a^2 I_a^*}{1 + \alpha I_a} E_a - \frac{\beta_a S_a^2 I_a^*}{1 + \alpha I_a} I_a - \frac{\beta_a S_a^2 I_a^*}{1 + \alpha I_a} I_a, \quad (52)
\end{align*}
\]

\[
\begin{align*}
(1 - \frac{E_h^*}{E_h}) &= (1 - \frac{E_h^*}{E_h})[\beta_h S_h I_a - \frac{\beta_a S_a I_a^*}{1 + m(I_h)^2} - \gamma_h S_h - \frac{\beta_a S_h I_a}{1 + m(I_h)^2}], \\
&= (1 - \frac{E_h^*}{E_h}) \left(1 - \frac{E_h^*}{E_h} \right) \left(1 - \frac{E_h^*}{E_h} \right) \left(1 - \frac{E_h^*}{E_h} \right) \left(1 - \frac{E_h^*}{E_h} \right) \left(1 - \frac{E_h^*}{E_h} \right) \left(1 - \frac{E_h^*}{E_h} \right) \left(1 - \frac{E_h^*}{E_h} \right) \left(1 - \frac{E_h^*}{E_h} \right), \\
&= \frac{\beta_h S_h I_a}{1 + m(I_h)^2} - \frac{\beta_h S_h I_a^* E_h}{1 + m(I_h)^2} E_h, \\
&= \frac{\beta_h S_h I_a}{1 + m(I_h)^2} E_h - \frac{\beta_h S_h I_a^* E_h}{1 + m(I_h)^2} E_h, \quad (53)
\end{align*}
\]

\[
\begin{align*}
\frac{\gamma_h + \delta_h}{\delta_h} (1 - \frac{I_h^*}{I_h}) I_h' &= \frac{(\gamma_h + \delta_h)}{\delta_h} (1 - \frac{I_h^*}{I_h})[\delta_h E_h - (\gamma_h + \theta_h + \psi_h)I_h], \\
&= \frac{(\gamma_h + \delta_h)}{\delta_h} (1 - \frac{I_h^*}{I_h})E_h \\
&= \frac{\beta_h S_h I_a}{1 + m(I_h)^2} E_h - \frac{\beta_h S_h I_a^*}{1 + m(I_h)^2} E_h \left(1 - \frac{I_h^*}{I_h} \right) I_h, \\
&= \frac{\beta_h S_h I_a^*}{1 + m(I_h)^2} E_h - \frac{\beta_h S_h I_a^*}{1 + m(I_h)^2} E_h - \frac{\beta_h S_h I_a^*}{1 + m(I_h)^2} E_h, \quad (54)
\end{align*}
\]
In equation (56),

\[
V'(t) = \gamma_aS^*_a \left( 2 - \frac{S^*_a - S_a}{S^*_a} \right) + \frac{\beta_h S^*_a I^*_a}{1 + m(I^*_h)^2} \left( 3 - \frac{S^*_h - E_h I^*_h}{E^*_h I^*_h - \frac{I_h}{I^*_h}} \right) + \frac{\beta_a S^*_a I^*_a}{1 + \alpha I^*_a} \left( 3 - \frac{S^*_a - E_a I^*_a}{E^*_a I^*_a - \frac{I_a}{I^*_a}} \right) + \frac{\beta_h S^*_a I^*_a}{1 + mI^*_h} \left( 1 - \frac{S^*_h E^*_h}{S^*_h E_h} \right) + \gamma_h S^*_h \left( 2 - \frac{S^*_h - S_h}{S^*_h} \right).
\]

(56)

In equation (56),

\[
\begin{align*}
2 - \frac{S^*_a - S_a}{S^*_a} & \leq 0, \\
3 - \frac{S^*_a - E_a I^*_a}{E^*_a I^*_a - \frac{I_a}{I^*_a}} & \leq 0, \\
2 - \frac{S^*_h - S_h}{S^*_h} & \leq 0, \\
3 - \frac{S^*_h - E_h I^*_h}{E^*_h I^*_h - \frac{I_h}{I^*_h}} & \leq 0, \\
\left( \frac{S_a E^*_a}{S^*_a E^*_a} - 1 \right) & \leq 0, \\
\left( \frac{S_h E^*_h}{S^*_h E^*_h} - 1 \right) & \leq 0.
\end{align*}
\]

(57)

Thus, the condition (47) ensures that \( V'(t) \leq 0 \) for all \((S_a, E_a, I_a, S_h, E_h, I_h) \in \Omega \), and the strict equality \( V'(t) = 0 \) holds only for \( S_a = S^*_a, E_a = E^*_a, I_a = I^*_a, S_h = S^*_h, E_h = E^*_h \) and \( I_h = I^*_h \). Then, the equilibrium state \( E_1 \) is the only positively invariant set of the system (1) contained entirely in \( \Omega = (S_a, E_a, I_a, S_h, E_h, I_h), \)

\( S_a = S^*_a, E_a = E^*_a, I_a = I^*_a, S_h = S^*_h, E_h = E^*_h \) and \( I_h = I^*_h \). and hence by the asymptotic stability theorem [29], the positive endemic equilibrium state \( E_1 \) is globally asymptotically stable on \( \Omega \).
Figure 2. The behavior of infected individuals $I_h$ when $R_0 > 1$.
Figure 2(a): $\alpha = m = 0$, Figure 2(b): $\alpha = m = 0.001$.

Figure 3. The behavior of infected individuals $I_h$ and $R_0 > 1$: Figure 3(a) when $\alpha = 0.001, 0.001, 0.01$ and $m = 0.001$ fixed. Figure 3(b) when $m = 0.001, 0.001, 0.01$ and $\alpha = 0.001$ fixed.

Figure 4. The behavior of infected individuals $I_h$ and $R_0 < 1$: Figure 4(a) when $\alpha = 0.001, 0.001, 0.01$ and $m = 0.001$ fixed. Figure 3(b) when $m = 0.001, 0.001, 0.01$ and $\alpha = 0.001$ fixed.
5. **Numerical results.** In this section, we carried out the numerical results for the avian influenza model (1). In numerical simulation, we fix the parameters as follows, $\Lambda_a = 350$, $\Lambda_h = 100$, $\delta_a = 0.3$, $\delta_h = 0.2$, $\gamma_a = 0.01$, $\gamma_h = 3.91 \times 10^{-3}$, $\mu_a = 0.05$, $\theta_h = 0.03$, $\psi_h = 0.01$, and varying $\alpha$, $m$, $\beta_a$ and $\beta_h$. The baseline (initial values) for the variables are $S_a(0) = 200000$, $E_a(0) = 100$, $I_a(0) = 1$, $S_h(0) = 200000$, $E_h(0) = 100$, $I_h(0) = 1$ and $R_h(0) = 0$. Initially, we study the effect of $\beta_a$ and $\beta_h$ on avian influenza model. The parameter $\beta_a$ which actually affects the population of human as its contact size is larger. Figure 1 is obtained by taking, $\beta_a = 1.772 \times 10^{-6}$, $\alpha = m = 0.01$ and the rest of the parameters are keep fixed, then $R_0 = 1$. In Figure 1(a), we varies $\beta_a = (1 \times 10^{-6}, 1.772 \times 10^{-6}, 3 \times 10^{-6})$ and $\beta_h = 8 \times 10^{-7}$ is keep fixed. If $\beta_a \leq \beta_a^*$, the disease vanish from the community, and $I_h(t)$ converges to the disease free equilibrium and stable locally asymptotically. In Figure 1(b), we varies $\beta_h = 8 \times 10^{-5}, 8 \times 10^{-6}, 8 \times 10^{-7}$ and fixed $\beta_a = 3 \times 10^{-6}$, then $R_0 = 1.69355 > 1$. If $\beta_a > \beta_a^*$, then the $I_h(t)$ converges to the endemic equilibrium which is stable locally asymptotically. Figure 2, is obtained by choosing $\alpha = m = 0$ and $\alpha = m = 0.01$. The subplot in Figure 2(a) is obtained by setting $\alpha = m = 0$ and Figure 2(b) is obtained by $\alpha = m = 0.01$. From Figure 2, one can see that the parameters $\alpha$ and $m$ reduce the number of infective individuals. Figure 3(a) is obtained by setting $m = 0.01$ and varying $\alpha$ from 0.0001, 0.001, 0.01. In Figure 3(b), we fix $\alpha = 0.001$ and varying $m = 0.001, 0.01, 0.1$. In Figure 3(a), keeping $\alpha = 0.01$ and varying $m$ see figure 3(b). If $\beta_a = 1 \times 10^{-6}$ then $R_0 = 0.564516$, the disease disappear from the population and converge to the disease free equilibrium,
which is stable locally asymptotically. Further, we fix the parameter $m = 0.001$ and varying $\alpha = 0.001, 0.01, 0.1$ we obtain Figure 4(a) and Figure 4(b) is obtained by fixing $\alpha = 0.001$ and varying $m = 0.001, 0.01, 0.1$. If we varying both the parameters $\alpha$ and $m$, we obtain Figure 5.

6. Discussion and conclusion. We proposed a mathematical model on avian influenza and presented the detailed analysis. We basically constructed two models one for birds and the other one for human. Initially, we presented a brief mathematical results for birds only model. The results obtained show that the birds only model is stable both locally and globally. The stability results for disease free equilibrium is obtained when $R_{0b} < 1$. If $R_{0b} > 1$, we proved that the endemic equilibrium of birds only model is stable both locally and globally. Then, we analyzed the full model and presented the local as well as the global asymptotic stability. The stability results for the disease free case (locally and globally) are presented when $R_0 < 1$. Further, when $R_0 > 1$ it is proven that the endemic equilibrium of the model is stable locally and globally.

The numerical results of the model is obtained and is given in Figure 1-5. The numerical results validate that the transmission dynamics of the avian influenza which is determined by force of infections in birds. It is observed that the parameter $\alpha$, $m$ and $\beta_h$, respectively show, the saturation effect, the psychological effect and the contact between infective birds to susceptible humans do not change the stability of the equilibria and so that the outbreaks, as the infected humans do not spread the virus further. However, the numerical results shows that making an increase in parameters $\alpha$ and $m$ and reducing the parameter $\beta_h$ should decrease the infected humans and can help to control the disease. Also, reducing new recruitment, newborns of domestic birds, reducing contact between susceptible and infective birds, and shortening the lifetime would be used as a optimal control strategy. In outbreak case, an effective control strategy should be enhancing the intensity of media coverage and avoiding contact with the infective avian population. Further, the fastest control of the disease is the closing of the retail live-poultry markets.

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