ANNALS OF COMMUNICATIONS IN MATHEMATICS
Volume 7, Number 4 (2024), 328-353
DOI: 10.62072/acm.2024.070402
ISSN: 2582-0818

A MATHEMATICAL MODEL FOR TRANSMISSION DYNAMICS OF AN AVIAN INFLUENZA DISEASE IN A HUMAN POPULATION

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ABSTRACT. Avian influenza is known as one of the respiratory diseases that causes high morbidity and mortality rate predominately among the immunodeficiency persons worldwide. Treatment and vaccination remain the optimal strategies in curbing the spread of avian infuenza infection.In this work, a mathematical model of the dynamics of influenza infection is formulated and was computed analytically and numerically. The analytic computation of the model is given in terms of the basic reproduction number, equilibria points and their stabilities. Thus, the disease dies out whenever the basic reproduction number is less than one. The disease free equilibrium (DFE) is locally asymptotically stable provided $R_0 < 1$ and unstable if otherwise. The endemic equilibrium only occurs whenever the disease threshold is greater than a unit. The endemic equilibrium, is locally, globally asymptotically stable under certain conditions. Numerical solution shows that vaccination and treatment of the susceptible and the infected individuals respectively have high impact for eradicating the disease. The non-linear incidence as a force of infection with parameter, θ,Ψ_1, u_1 and u_2 have great impact for reducing the pandemic of influenza disease. In conclusion, vaccination of susceptible individuals, isolation of exposed individuals and treatment of infected individuals are imperative for curbing the spread of an avian influenza infection. Modelling style or structure especially, the type of force of infection adopted for modelling an avian influenza disease depends on whether the disease, can easily be put under control.

1. INTRODUCTION

Avian influenza (AI) is a highly contagious viral disease affecting several species of food producing birds (chickens, turkeys, quails, guinea fowl to mention a few) as well as pet birds and wild birds. Occasionally mammals including humans may contract avian influenza (WAOH, 2020) [26].

The case and circulation of avian influenza (AI) virus is not a new phenomenon. There are many descriptions of historical outbreaks of avian influenza disseminating within domestic poultry flocks in the literatures (CDC, 2017 [4]; CDC, 2023 [5] WHO, 2018 [24]; WAOH, 2020 [26]; WHO, 2023 [25]). Avian Influenza (AI) occurs worldwide and different strains are more prevalent in certain areas of the world than others (WAOH, 2020)[26].

2020 *Mathematics Subject Classification.* 90B99, 62P10, 92B05.
Key words and phrases. Mathematical model; avian influenza; transmission dynamics, stability.
Received: September 21, 2024. Accepted: November 25, 2024. Published: December 31, 2024.
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It is less frequent in mammalian species, including rats, mice, weasels, ferrets, pigs, cats, tigers, dogs and horses, as well as humans. AI of the flu is a common respiratory disease caused by influenza virus (Cox and Subbarao, 1999 [6]; Shek and Lee, 2003 [22]; Wang *et al.*, 2015 [23]).

Avian influenza A virus can spread from infected birds to susceptible humans and reports from such transmission are documented in the Centers for Disease Control and Prevention's website. Humans infected by avian influenza infection exhibit symptoms such as fever, cough, sore throat, acute respiratory distress and sometimes respiratory failure. The most destructive outbreak of influenza in history happened in 1918-1919. Avian influenza is considered as one of the most severe endemic outbreaks the world has ever witnessed and encountered. The epidemic is believed to have resulted in over 50 million to 100 million deaths globally. In Nigeria, a high pathogenic outbreak of avian influenza (HPAI) H5N1 in 30 poultry farms in seven states. The affected states include Kano, Plateau, Bauchi, Gombe, Nasarawa, Kaduna and Niger state.

The outbreak of AI has become a major concern. Various health bodies and organizations throughout the world continue to seek for solutions that could bring the pandemic under control. Modnak (2017) [15] accounts for the nonlinear incidence rate as our force of infection in inhibiting the disease transmission. The control measures are built into the model to help develop strategies for the control of the disease. In this study, the transmission dynamics and control of AI was done by modifying the model of Modnak (2017) [15].

It has become possible and imperative to mathematically model the progress of infectious diseases to uncover the likely outcome of an epidemic or to help manage them by different control programmes. In the early 20th century, mathematical models were introduced for epidemiology by Ross (1916)[21] and others (Chong and Tchuenche, 2014[6]; Hancioglu, 2007 [9]; Henaux *et al.*, 2013 [10]; Liu *et al.*, 2015 [14]; Mohler *et al.*, 2015 [16]; Taubenberger and Morens, 2006, 2008 [19, 20] Parvin *et al.*, 2019 [17]). Even though the actual data needed for the models might not be accurate or ever available (Aid *et al.*, 2015 [1]), modelling is vital in investigating how changes in the various assumptions and parameter values affect the course of the epidemic. In order to gain more insight into the dynamics of an avian influenza disease, we shall review related models that have been studied by many authors as discussed below.

Putri *et al.* (2016) [18] studied a mathematical model of influenza dynamics and compared the incubation period and control in Thailand. The data from the annual number of cases reported by the Division of Epidemiology, Ministry of Public Health during the period 1997 – 2013 were analyzed. They presented two models using the system of nonlinear differential equations for the two models. The standard dynamical modeling methods are applied to determine the behaviours of solutions to each model. The conditions required of the parameters for the disease free and endemic equilibrium state to be local asymptotically stable is obtained. Their numerical simulations are seen to support the theoretical predictions.

Jagan and Kartheek (2016) [11] carried out research on the epidemic analysis and mathematical modeling of H1N1 (A) with vaccination. They consider the infected individuals using rate coefficients such as transmission, progression, recovery and vaccination. The fact that the dynamics analysis is completely determined by the basic reproduction number is established. More specifically, local and global stabilities of the disease free equilibrium and the endemic equilibrium are proved under certain parameter values when the basic

reproduction number is below or above unity. The numerical simulations review that vaccination is one of the best effective strategies or solutions that is to be adopted so as to have the spread or if possible, avoid poultry farms, contact with animals in live poultry markets, entering areas where poultry may be slaughtered, and contact with any surfaces that appear to be contaminated with feaces from poultry or other animals. Good food safety and food hygiene practices e.g. hands washing with soap and water should be followed. Travellers returning from affected regions should report to local health services if respiratory symptoms suspecting zoonotic influenza virus infection.

Caroline *et al.* (2019) [13] studied mathematical analysis of influenza A dynamics in the emergence of drug resistance. The qualitative analysis of the model is given in terms of the control reproduction number, R_c. The model equilibria are computed and stability analysis was carried out. The model is found to exhibit backward bifurcation prompting the need to lower R_c to a critical value R_c for effective disease control. Sensitivity analysis results reveal that vaccine efficacy is the parameter with the most control over the spread of influenza. Numerical simulations reveal that despite vaccination reducing the reproduction number below unity, influenza still persists in the population. Hence, it is essential, in addition to vaccination, to apply other strategies to curb the spread of influenza.

Khan *et al.* (2019)[12] modelled the transmission dynamics of avian influenza with saturation and psychological effect. They show that the birds only model is stable both locally and globally. The stability results for disease free equilibrium is obtain when the disease threshold parameter, $R_0 < 1$. They also proved that $R_0 > 1$. The analysis of their full model shows that the disease free case (locally and globally) when $R_0 < 1$. More so, when $R_0 > 1$, it was proved that the endemic equilibrium of the model is stable locally and globally. Their numerical analysis review that the transmission dynamics of the avian influenza is determined by the force of infection in birds. It is observed that the parameter α, m and β_m which represent 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 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 an optimal control strategy.

The organisation of this paper is as follows. The model is formulated in Section 2. The analyses of the formulated model and its numerical simulations are carried out in Section 3 and Section 4 respectively. Discussion and conclusion are presented in Section 5.

2. Model Formulation

A simplified dynamical model of immune response to uncomplicated influenza virus infection is presented which put into consideration on the control of the infection. Most influenza disease mathematical model follow $SVEIR$ model for human disease transmission. On the other hand, the carrier (bird) population has SIR model dynamics. It should be noted that influenza model sometimes capture human population only without explicitly show the carrier model (Modnak, 2017)[15].

2.1. **Formulation of Existing Model.**

In this section, we shall present the model by Modnak (2017) [15] by putting into consideration the assumptions, variables and parameters of the model

2.2. **Assumptions of the existing model.** The following assumptions were made by Modnak (2017) [15] for formulating avian influenza virus model.

- Influenza Avian virus can spread from infected birds to susceptible humans;
- Recovered or vaccinated class(es) of humans may lose immunity over time and re-enter the susceptible population and become re-infected individuals;
- Individuals are born and die at an average rate, μ;
- Susceptible individuals are vaccinated.

The variables and parameters of the existing model are given in Table 1 while the flow diagram of the model is presented in Figure 1.

TABLE 1. Description and Interpretation of Variables of the Existing Model Equations

Variable	**Interpretation**
$S(t)$	Number of susceptible human at time t
$V(t)$	Number of vaccinated human at time t
$E(t)$	Number of exposed human at time t
$I(t)$	Number of infected human at time t
$R(t)$	Number of recovered human at time t
μN	Birth and death rate of the population
α	Disease induced death rate
$\phi_1(t)$	Vaccination rate of the susceptible individual
$\sigma = (1-\xi)$	Degree of protection, where, ξ is the vaccine efficacy
$\phi_2(t)$	Treatment of the infected individual rate
γ	Natural recovery rate of the infected individuals
k	Progression rate from exposed to infectious
δ	Rate at which recovered individual loss immunity and become susceptible
$1+\theta I(t)$	Inhibition effect from the behavioural increase
$\lambda(t)$	The force of infection which represent the transmission rate of the disease

2.3. **Equations of the existing model.** The equations of the existing model is given by Equations (1) - (5)

$$\frac{dS}{dt} = \mu - \beta IS(t) - (\psi_1(t) + \mu)S(t) + \delta R(t), \tag{1}$$

$$\frac{dV}{dt} = \psi_1(t)S(t) - \sigma\beta I(t)V(t) - \mu V(t), \tag{2}$$

$$\frac{dE}{dt} = \sigma\beta I(t)V(t) + \beta I(t)S(t) - kE(t) - \mu E(t), \tag{3}$$

$$\frac{dI}{dt} = kE(t) - (\alpha + \mu + \gamma + \psi_2(t))I(t), \tag{4}$$

$$\frac{dR}{dt} = \psi_2(t)I(t) + \gamma I(t) - \mu R(t) - \delta R(t). \tag{5}$$

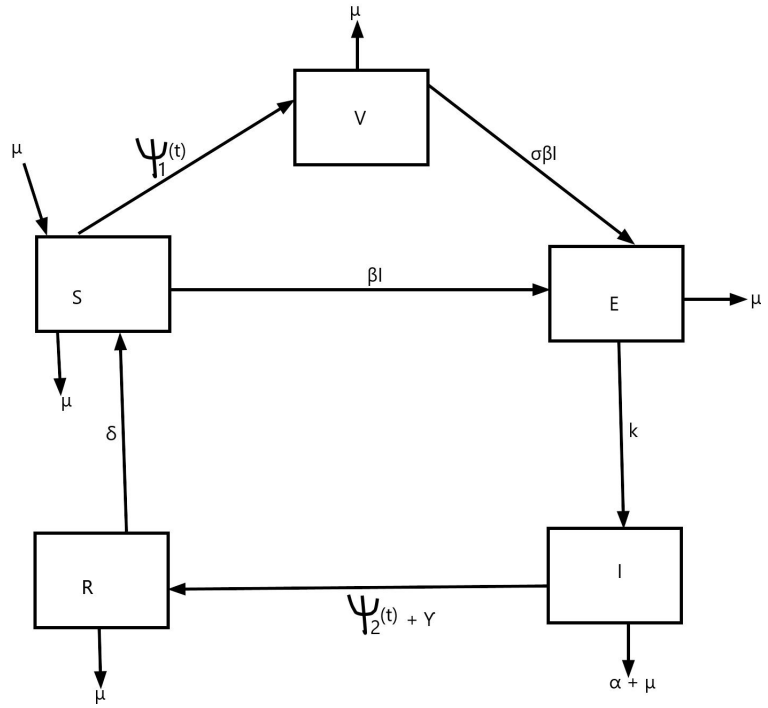

FIGURE 1. **Flow Diagram of the Existing Model (Modnak, 2017)** [15]

where $\psi_1(t)$ and $\psi_2(t)$ are function of t, representing non-uniform and time dependent controls.

2.4. **The Modified Model Equations.** We modified the model by Modnak (2017) [15] by converting the model from autonomous version to a non-autonomous version. We also considered the force of infection to be incidence rate. Lastly, we employed the use of constant recruitment rate into the population.

2.5. **Assumptions of the modified model.** In addition to the assumptions of the existing model, we make the following assumptions:

- H_1 The recruitment into the susceptible is considered for a varying population;
- H_2 The force of infection follows saturated rate due to the disease inhibition;
- H_3 A non-autonomous model is considered due to the time dependence of each state variable;
- H_4 Two controls which represent the reduction of force of infection and the isolation of some exposed individuals that had contact with the infected individuals respectively are introduced;
- H_5 Parameters used in the model are not time dependent as opposed to the existing model.

The variables and parameters of the modified model are given in Table 2. While the schematic diagram for the modified model is represented by Figure 2.

TABLE 2. Description and Interpretation of Variables of the Model Equations

Variable	Interpretation
$S(t)$	Number of susceptible human at time t
$V(t)$	Number of vaccinated human at time t
$E(t)$	Number of exposed human at time t
$I(t)$	Number of infected human at time t
$R(t)$	Number of recovered human at time t
Λ	Recruitment rate of human population
μ	Natural death rate
u_1, u_2	Infection control parameters
δ	Disease induced death rate
ψ_1	Vaccination rate of the susceptible individual
$\sigma = (1 - \xi)$	Degree of protection where ξ is the vaccine efficacy
ψ_2	Treatment of the infected individual
γ	Natural recovery rate of the infected individuals
k	Progression rate from exposed to infectious
δ	Rate at which recovered individual loss immunity and become susceptible
$1 + \theta I(t)$	Inhibition effect from the behavioural increase
$\lambda(t)$	The force of infection which represent the transmission rate of the disease

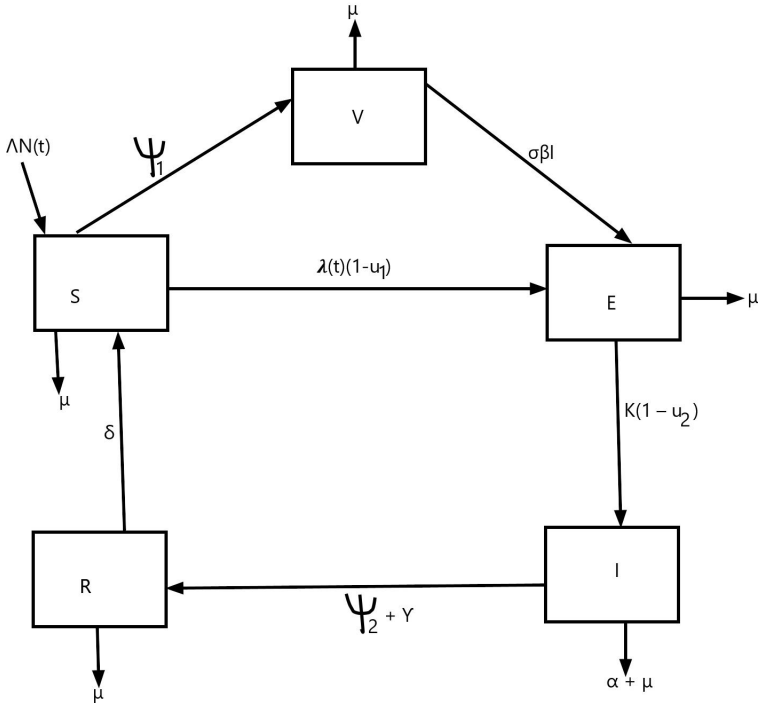

FIGURE 2. Flow Diagram of the Modified Model

2.6. **Governing equations of the modified model.** Putting into consideration the variables, parameters and the assumptions stated above we arrived at our modified model as seen in Equations $(6)-(11)$ below.

$$\frac{dS}{dt} = \Lambda N(t) - \lambda(t)(1-u_1)S(t) - (\psi_1+\mu)S(t) + \delta R(t), \tag{6}$$

$$\frac{dV}{dt} = \psi_1 S(t) - \sigma\beta I(t)V(t) - \mu V(t), \tag{7}$$

$$\frac{dE}{dt} = \sigma\beta I(t)V(t) + \lambda(t)(1-u_1)S(t) - k(1-u_2)E(t) - \mu E(t), \tag{8}$$

$$\frac{dI}{dt} = k(1-u_2)E(t) - (\alpha+\mu+\gamma+\psi_2)I(t), \tag{9}$$

$$\frac{dR}{dt} = \psi_2 I(t) + \gamma I(t) - (\delta+\mu)R(t), \tag{10}$$

where

$$\lambda(t) = \frac{\beta I(t)}{(1+\theta I(t))}, N(t) = S(t)+V(t)+E(t)+I(t)+R(t). \tag{11}$$

3. Model Analysis

3.1. **Positive invariant region.** In this sub-section, we show the positive invariant of the five state variables in model Equations $(6)-(10)$.

$$\begin{aligned}\frac{dN(t)}{dt} &= \frac{dS(t)}{dt} + \frac{dV(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI(t)}{dt} + \frac{dR(t)}{dt} \\ &= \Lambda - \mu N(t) - \alpha I(t)\end{aligned} \tag{12}$$

When the disease no longer kills. Thus, $\frac{dN(t)}{dt} \leq \Lambda - \mu N(t)$ for $\alpha = 0$

At $\frac{dN(t)}{dt} = 0$, we have

$$\Lambda - \mu N(t) \leq 0 \tag{13}$$

therefore,

$$N(t) \leq \frac{\Lambda}{\mu}$$

Proposition 1: The solution of the model $(6)-(10)$ is feasible for all $t > 0$ if they enter the invariant region.

$$D = \{(S(t),V(t),E(t),I(t),R(t)) \in \Re^{5+} : S(t) > 0, V(t) \geq 0, E(t) \geq 0, I(t) \geq 0, R(t) \geq 0 | N(t) \leq \frac{\Lambda}{\mu}\} \tag{14}$$

Theorem 3.1. The closed set $D = \{(S(t),V(t),E(t),I(t),R(t)) \in \Re^5_+ : N(t) \leq \frac{\Lambda}{\mu}\}$ is positively invariant and attract all positive solutions of the model equations.

Remark 1. In the theorem, we think of the solution space as having five dimensions, so that at any point in time t, we have a vector of solutions with five elements (standing for the state variables) real and positive solutions, hence the plus sign in $\Re^{5+}$. We therefore prove Theorem $(6-10)$. Generally, it implies thus:

Proof:

$$\frac{dN}{dt} = \Lambda - \mu N(t) - \alpha I(t), \tag{15}$$

By a standard comparison theorem [Lakshmikantham *et al.,*1989], we see that

$$\frac{dN}{dt} = \Lambda - \mu N(t), \tag{16}$$

which yields (by the method of integrating factor)

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}). \tag{17}$$

To be specific, if $N(0) \leq \frac{\Lambda}{\mu}$, then $N(t) \leq \frac{\Lambda}{\mu}$. Hence, D is positively invariant and an attractor so that no solution path leaves through any boundary of D.

3.2. **Disease Free Equilibrium (DFE) Analysis.** Under this sub-section, we carry out the equilibrium state of our model Equations $(6) - (10)$ when there is no disease in the population of interest. To investigate this, we simply substitute $E(t) = I(t) = R(t) = 0$ and $\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$ in model Equations $(6) - (10)$ and obtain

$$\Lambda - (\psi_1 + \mu)S(t) = 0, \tag{18}$$

$$\psi_1 S(t) - \mu V(t) = 0. \tag{19}$$

Solving Equations (18) and (19) simultaneously, we have

$$S(t) = \frac{\Lambda}{\psi_1 + \mu}, \tag{20}$$

and

$$V(t) = \frac{\psi_1 \Lambda}{\mu(\psi_1 + \mu)}, \tag{21}$$

Thus, the DFE of the model $(6) - (10)$ is given by

$$\begin{aligned} E_1 &= (S(t)^*, V(t)^*, E(t)^*, I(t)^*, R(t)^*), \\ &= (\frac{\Lambda}{\psi_1 + \mu}, \frac{\psi_1 \Lambda}{\mu(\psi_1 + \mu)}, 0, 0, 0). \end{aligned} \tag{22}$$

3.3. **Analysis of Basic Reproduction Number** (R_0)**.** To calculate the basic reproduction number, we divide model Equations $(6) - (10)$ into appearance of infection and transfer of infection as matrix $F_i(x)$ and $V_i(x)$ (Driessche and Watmough, 2002)[8] where $F_i(x)$ be the rate of appearance of new infections in compartment i and $V_i(x)$ be the difference between the transfer rate of individuals out of compartment i by all other means. Thus, the $F_i(x)$ and $V_i(x)$ of model Equations $(6) - (10)$ is shown below: Let x_0 be the DFE of model Equations $(6) - (10)$. Thus, we have the following partitioned derivatives,

$$DF(x_0) = \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix},$$

$$DV(x_o) = \begin{pmatrix} V & 0 \\ J_3 & J_4 \end{pmatrix},$$

where F and V are $M \times N$ matrices defined by

$$F = \left(\frac{\partial F_i}{\partial x_j(x_o)} \right), \tag{23}$$

and

$$V = \left(\frac{\partial V_i}{\partial x_j(x_o)} \right), \tag{24}$$

So that

$$V^{-1} = \frac{1}{\text{determinant}} \left(\frac{\partial V_i}{\partial x_j(x_o)} \right), \tag{25}$$

$1 \leq 1, j \leq m$.
Here,the partial derivatives of F_i and V_i are with respect to the infected classes only. The basic reproduction number is defined as

$$R_o = \rho(FV^{-1}), \tag{26}$$

where ρ is the spectral radius of FV^{-1}(by spectral radius we mean the maximum eigenvalue of FV^{-1}.

Theorem 3.2. Consider the disease transmission model (2) with $f(x)$ satisfying the stability conditions if x_0 is a DFE of the model,the x_o is locally asymptotically stable if $R_0 < 1$, but unstable if $R_0 > 1$.

For the purpose of easy access and clarity, we shall rewrite model Equations $(6) - (10)$ as follows:

$$\frac{dS}{dt} = \Lambda N(t) - \lambda(t)(1-u_1)S(t) - (\phi_1 + \mu)S(t) + \delta R(t), \tag{27}$$

$$\frac{dV}{dt} = \phi_1 S(t) - \sigma\beta I(t)V(t) - \mu V(t), \tag{28}$$

$$\frac{dE}{dt} = \sigma\beta I(t)V(t) + \lambda(t)(1-u_1)S(t) - k(1-u_2)E(t) - \mu E(t), \tag{29}$$

$$\frac{dI}{dt} = k(1-u_2)E(t) - (\alpha + \mu + \gamma + \phi_2)I(t), \tag{30}$$

$$\frac{dR}{dt} = \phi_2 I(t) + \gamma I(t) - (\delta + \mu)R(t), \tag{31}$$

where

$$\lambda(t) = \frac{\beta I(t)}{(1 + \theta I(t))}, N(t) = S(t) + V(t) + E(t) + I(t) + R(t). \tag{32}$$

Thus, F and V are given as

$$F = \begin{pmatrix} \sigma\beta I(t)V(t) + \frac{\beta I(t)S(t)(1-u_1)}{1+\theta I(t)} \\ 0 \end{pmatrix}, \tag{33}$$

and

$$V = \begin{pmatrix} k(1-u_2)E(t) + \mu E(t) \\ -k(1-u_2)E(t) + (\alpha + \mu + \gamma + \psi_2)I(t) \end{pmatrix}. \tag{34}$$

Since we have only two infected classes, $E(t)$ and $I(t)$. It follows that $m = 2$.
We should note that at the DFE, $E(t)^* = I(t)^* = R(t)^* = 0$, $X \in (S(t), V(t), E(t), I(t), R(t))$

$$S(t)^* = \frac{\Lambda N(t)}{\psi_1 + \mu}. V(t)^* = \frac{\psi_1 \Lambda N(t)}{\mu(\psi_1(t) + \mu)}, \tag{35}$$

Putting these into consideration, we have

$$M = \frac{\partial F}{\partial X}(E_1) = \begin{pmatrix} 0 & \sigma\beta V(t) + \beta S(t) \\ 0 & 0 \end{pmatrix}, \tag{36}$$

$$N = \begin{pmatrix} k(1-u_2)+\mu & 0 \\ -k(1-u_2) & \alpha+\gamma+\mu+\psi_2 \end{pmatrix},$$

and

$$N^{-1} = \begin{pmatrix} \frac{1}{k(1-u_2)+\mu} & 0 \\ \frac{k(1-u_2)}{(k(1-u_2)+\mu)(\alpha+\gamma+\mu+\psi_2)} & \frac{1}{\alpha+\gamma+\mu+\psi_2} \end{pmatrix}. \tag{37}$$

Therefore,

$$MN^{-1} = \begin{pmatrix} \frac{\beta k\Lambda N(t)\sigma(1-u_2)\phi_1}{\mu(\mu+\phi_1)(k(1-u_2)+\mu)(\alpha+\gamma+\mu+\phi_2)} & \frac{\beta\Lambda N(t)\sigma\phi_1}{\mu(\mu+\phi_1)(\alpha+\gamma+\mu+\phi_2)} \\ 0 & 0 \end{pmatrix}. \tag{38}$$

$$\left|MN^{-1} - xI\right| = \begin{vmatrix} \frac{\beta k\Lambda\sigma(1-u_2)\psi_1}{\mu(\mu+\psi_1)(k(1-u_2)+\mu)(\alpha+\gamma+\mu+\psi_2)} - x & \frac{\beta\Lambda\sigma\psi_1}{\mu(\mu+\psi_1)(\alpha+\gamma+\mu+\psi_2)} \\ 0 & 0-x \end{vmatrix} = 0 \tag{39}$$

Thus, calculating the eigenvalues of equation (39), we have,

$$x_1 = 0,$$

$$x_2 = \frac{\beta k\Lambda\sigma(1-u_2)\psi_1}{\mu(\mu+\psi_1)(k(1-u_2)+\mu)(\alpha+\gamma+\mu+\psi_2)}.$$

More so, the dominant eigenvalue is the basic reproduction number. Therefore,

$$R_0 = \frac{\beta k\Lambda\sigma(1-u_2)\psi_1}{\mu(\mu+\psi_1)(k(1-u_2)+\mu)(\alpha+\gamma+\mu+\psi_2)}.$$

The biological interpretation of this R_0 is that, the avian influenza disease will die off whenever its numerical value is less one. It becomes endemic whenever the R_0 is greater than one. It becomes inconclusive whenever R_0 equals 0 or 1. In such case backward or forward bifurcation analysis will be required.

3.4. **Local Asymptotic Stability of the Disease Free Equilibrium.** In this sub-section, we investigate the local asymptotic stability (LAS) of our model Equations $(6)-(10)$ in the case where there is no disease in a population of interest (Asquith and Bangham, 2003)[2]. To do this, we linearized our model equations with the corresponding equilibrium, E_1. We first rewrite model Equations $(6)-(10)$ as follows.

$$f_1 = \Lambda N(t) - \lambda(t)(1-u_1)S(t) - (\psi_1+\mu)S(t) + \delta R(t), \tag{40}$$

$$f_2 = \psi_1 S(t) - \sigma\beta I(t)V(t) - \mu V(t), \tag{41}$$

$$f_3 = \sigma\beta I(t)V(t) + \lambda(t)(1-u_1)S(t) - k(1-u_2)E(t) - \mu E(t), \tag{42}$$

$$f_4 = k(1-u_2)E(t) - (\alpha+\mu+\gamma+\psi_2)I(t), \tag{43}$$

$$f_5 = \psi_2 I(t) + \gamma I(t) - (\delta+\mu)R(t). \tag{44}$$

Differentiating $(40)-(44)$ with respect to $S(t), V(t), E(t), I(t)$ and $R(t)$, which yielded Equations $(45), (46), (47), (48)$ and (49) respectively

$$\frac{\partial f_1}{\partial S} = \frac{\beta I(t)(1-u_1)}{1+\theta I(t)} - \mu - \psi_1, \frac{\partial f_1}{\partial V} = 0, \frac{\partial f_1}{\partial E} = 0, \frac{\partial f_1}{\partial I} = -\frac{S(t)I(t)\beta\theta}{(1+\theta I(t))^2} - \frac{S(t)\beta}{1+\theta I(t)}, \tag{45}$$

$$\frac{\partial f_1}{\partial R} = \delta, \frac{\partial f_2}{\partial S} = \psi_1, \frac{\partial f_2}{\partial V} = -\mu - I(t)\beta\sigma, \frac{\partial f_2}{\partial E} = 0, \frac{\partial f_2}{\partial I} = -\sigma(t), \frac{\partial f_2}{\partial R} = 0, \tag{46}$$

$$\frac{\partial f_3}{\partial S} = \frac{(t)(1-u_1)}{1+\theta I(t)}, \frac{\partial f_2}{\partial V} = \sigma\beta I(t), \frac{\partial f_3}{\partial E} = -k(1-u_2) - \mu, \frac{\partial f_3}{\partial I} = \sigma\beta V(t) + \frac{S(t)I(t)\beta\theta}{(1+\theta I(t))^2} + \frac{S(t)\beta}{1+\theta I(t)}, \tag{47}$$

$$\frac{\partial f_3}{\partial R} = 0, \frac{\partial f_4}{\partial S} = 0, \frac{\partial f_4}{\partial V} = 0, \frac{\partial f_4}{\partial E} = k(1-u_2), \frac{\partial f_4}{\partial I} = -(\alpha+\mu+\gamma+\psi_2), \frac{\partial f_4}{\partial R} = 0, \tag{48}$$

$$\frac{\partial f_5}{\partial S} = 0, \frac{\partial f_5}{\partial V} = 0, \frac{\partial f_5}{\partial E} = 0, \frac{\partial f_5}{\partial I} = \psi_2 + \gamma, \frac{\partial f_5}{\partial R} = -(\mu+\delta). \tag{49}$$

Using the above linearized system, we obtain the general Jacobian Matrix of the model $(6)-(10)$ and is given by

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial V} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial V} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial V} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial V} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial R} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial V} & \frac{\partial f_5}{\partial E} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial R} \end{pmatrix}, \tag{50}$$

Now, at the equilibrium E_1, we have

$$J(E_1) = \begin{pmatrix} -\mu-\psi_1 & 0 & 0 & 0 & \delta \\ \psi_1 & -\mu & 0 & -\frac{\beta\Lambda\sigma\psi_1}{\mu(\mu+\psi_1)} & 0 \\ 0 & 0 & -k(1-u_2)-\mu & \frac{\beta\Lambda\sigma\psi_1}{\mu(\mu+\psi_1)} & 0 \\ 0 & 0 & k(1-u_2) & -\alpha-\gamma-\mu-\psi_2 & 0 \\ 0 & 0 & 0 & \gamma+\psi_2 & -\delta-\mu \end{pmatrix}, \tag{51}$$

$$J(E_1)-xI = \begin{pmatrix} -\mu-x-\psi_1 & 0 & 0 & 0 & \delta \\ \psi_1 & -\mu-x & 0 & -\frac{\beta\Lambda\sigma\psi_1}{\mu(\mu+\psi_1)} & 0 \\ 0 & 0 & -k(1-u_2)-\mu-x & \frac{\beta\Lambda\sigma\psi_1}{\mu(\mu+\psi_1)} & 0 \\ 0 & 0 & k(1-u_2) & -\alpha-\gamma-\mu-x-\psi_2 & 0 \\ 0 & 0 & 0 & \gamma+\psi_2 & -\delta-\mu-x \end{pmatrix}, \tag{52}$$

The eigenvalues of equation (52) was obtained using Mathematica Software with the command in Appendix A.

$$a_1x^2 + a_2x + a_3 = 0, \tag{53}$$

where

$$a_1 = 1,$$
$$a_2 = \frac{A-B}{\mu^2+\mu\psi_1},$$
$$a_3 = \frac{T}{\mu^2+\mu\psi_1},$$

with

$$A = \alpha\mu^2 + \alpha\mu\psi_1 + \gamma\mu^2 + \gamma\mu\psi_1 + k\mu^2 + k\mu\psi_1 + 2\mu^3 + 2\mu^2\psi_1 + \mu^2\psi_2 + \mu\psi_1\psi_2,$$

$B = k\mu^2u_2 + k\mu u_2\psi_1,$
$T = \alpha k\mu^2 + \gamma k\mu^2 + k\mu^3 + \alpha\mu^3 + \gamma\mu^3 + \mu^4 - \alpha k\mu^2u_2 - \gamma k\mu^2u_2 - k\mu^3u_2 + \alpha k\mu\psi_1 + \gamma k\mu\psi_1 + k\mu^2\psi_1 + \alpha\mu^2\psi_1 + \gamma\mu^2\psi_1 + \mu^3\psi_1 - \beta k\Lambda\sigma\psi_1 - \alpha k\mu u_2\psi_1 - \gamma k\mu u_2\psi_1 - k\mu^2u_2\psi_1 + \beta k\Lambda\sigma u_2\psi_1 + k\mu^2\psi_2 + k\mu\psi_1\psi_2 - k\mu^2u_2\psi_2 - k\mu u_2\psi_1\psi_2 - \mu^3\psi_2 + \mu^2\psi_1\psi_2.$

Remark 2. The model $(6) - (10)$ is locally asymptotically stable provided $R_0 < 1$ and unstable if otherwise.

3.5. **Disease Endemic Equilibrium.** Disease endemic equilibrium (DEE) only occurs when the basic reproduction number R_0 is greater than a unit. Whenever this occurred, the disease evade a population and persist for a long time. We therefore seek to investigate the DEE of model $(6) - (10)$,
$E_2 = (S(t)^{**}, V(t)^{**}, E(t)^{**}, I(t)^{**}, R(t)^{**})$ by equating the left hand sides of the model $(6) - (10)$ to zero and then solve for each state variable as follows:

$$\begin{aligned}
&\Lambda - \lambda(t)(1-u_1)S(t) - (\psi_1+\mu)S(t) + \delta R(t) = 0,\\
&\psi_1 S(t) - \sigma\beta I(t)V(t) - \mu V(t) = 0,\\
&\sigma\beta I(t)V(t) + \lambda(t)(1-u_1)S(t) - k(1-u_2)E(t) - \mu E(t) = 0,\\
&k(1-u_2)E(t) - (\alpha+\mu+\gamma+\psi_2)I(t) = 0,\\
&\psi_2 I(t) + \gamma I(t) - (\delta+\mu)R(t) = 0.
\end{aligned} \tag{54}$$

We solve for each state variable of Equations (15) and we obtain

$$\begin{aligned}
S(t)^{**} &= \frac{K_1}{K_2},\\
V(t)^{**} &= \frac{L_1}{L_2},\\
E(t)^{**} &= \frac{\Phi_1}{\Phi_2},\\
I(t)^{**} &= \frac{k\,(1-u_2)}{\alpha+\gamma+\mu+\psi_2},\\
R(t)^{**} &= \frac{k\,(1-u_2)\,(\gamma+\psi_2)}{(\delta+\mu)\,(\alpha+\gamma+\mu+\psi_2)},
\end{aligned}$$

where $\Phi_1, \Phi_2, L_1, L_2, K_1, K_2$ are presented in D.

3.6. **Local Asymptotic Stability of the Disease Endemic Equilibrium.** Here, we seek to analyze the local asymptotic stability of the disease endemic equilibrium (DEE). To do this we claim the following result

$$J(E_2) = \begin{pmatrix}
P_1 - u_1 - \psi_1 & 0 & 0 & P_2 & \delta\\
\psi_1 & -\mu - P_3 & 0 & -\beta H^*\sigma & 0\\
P_4 & P_5 & -k\,(1-u_2) - \mu & \beta H^*\sigma + P_6 & 0\\
0 & 0 & k\,(1-u_2) & -\alpha-\gamma-\mu-\psi_2 & 0\\
0 & 0 & 0 & \gamma+\psi_2 & -\delta-\mu
\end{pmatrix}, \tag{55}$$

where
$P_1 = -\frac{\beta I(t)(1-u_1)}{1+\theta I(t)}, P_2 = -\frac{(1+\theta I(t)\beta S(t))-\beta I(t)S(t)\theta}{(1+\theta I(t))^2}, P_3 = \sigma\beta I(t), P_4 = \frac{\beta I(t)(1-u_1)}{1+\theta I(t)}$,
$P_5 = \sigma\beta V(t), P_6 = \frac{(1+\theta I(t)\beta S(t))-\beta I(t)S(t)\theta}{(1+\theta I(t))^2}, H = V(t)^{**}$.

$$J(E_2)-Iy = \begin{pmatrix} P_1 - u_1 - \psi_1 - y & 0 & 0 & P_2 & \delta \\ \psi_1 & -\mu - P_3 - y & 0 & -\beta H^*\sigma & 0 \\ P_4 & P_5 & c_1 - y & \beta H^*\sigma + P_6 & 0 \\ 0 & 0 & k\,(1-u_2) & c_2 - y & 0 \\ 0 & 0 & 0 & \gamma + \psi_2 & -\delta - \mu - y \end{pmatrix}, \tag{56}$$

where $c_1 = -k\,(1-u_2) - \mu, c_2 = -\alpha - \gamma - \mu - \phi_2$.

The characteristic equation corresponding to the above matrix is given by

$$y^5 + a_1y^4 + a_2y^3 + a_3y^2 + a_4y + a_5 = 0, \tag{57}$$

where a_1, a_2, a_3, a_4, a_5 are obtained using Mathematica Software command in Appendix B and the coefficients of equation (57) are stated in Appendix E.

Theorem 3.3. The equilibrium, E^2 is locally asymptotically stable provided the coefficients of the characteristic equation (19) satisfies the following Routh Hurwitz stability conditions:

(1) $a_i(i = 1, 2, ..., 5) > 0$,
(2) $a_1a_2 > a_3$,
(3) $a_1a_2a_3 > a_3^2 + a_1^2a_4$,
(4) $a_1a_2a_3a_4 + a_1a_{2a5} + 2a_1a_4a_5 > a_3^2a_4 + a_1^2a_4^2 + a_1b_2^2a_5 + a_5^2$.

Proof: The prove of Theorem 3.5 is omitted due to the size of the polynomial coefficients $(a_1, a_2, a_3, a_4, a_5)$. However, in application, we will simply use numerical values as tabulated in Table 3 to check.

3.7. **Global Stability Analysis of the Disease Endemic Equilibrium.** In this section, we present the global stability of the model (2) at E_2^* equilibrium. We assume that the drug efficacy $\xi \to 1$ and then $\sigma \to 0$. We give the following theorem by following (Li *et al.*, 2018). [18]

The global stability analysis of the endemic equilibrium, E_2^* of model (2) becomes imperative when $R_0 > 1$. To investigate the stability of the E_2^* equilibrium, we first of all consider the compartments with the disease force of infection and the infected classes only as follow.

$$\Lambda = \frac{\beta I(t)^{**}}{(1+\theta I(t)^{**})}(1-u_1)S(t)^{**} + (\psi_1+\mu)S(t) - \delta R(t)^{**},$$

$$k(1-u_2)+\mu = \frac{\sigma\beta I(t)^{**}V(t)^{**} + \frac{\beta I(t)^{**}}{(1+\theta I(t)^{**})}(1-u_1)S(t)}{E(t)^{**}},$$

$$\frac{\alpha+\mu+\gamma+\psi_2}{k(1-u_2)} = \frac{I(t)^{**}}{E(t)^{**}}.$$

Thus, we construct a Lyapunov function

$$Y(t) = (S(t) - S^{**}(t) - S^{**}(t)log\frac{S(t)}{S^{**}(t))} + (E(t) - E^{**}(t) - E^{**}(t)log\frac{E(t)}{E^{**}(t)} + \frac{k+\mu}{k}(I(t) - I^{**}(t) - I^{**}(t)log\frac{I(t)}{I^{**}(t)}. \tag{58}$$

Taking the time derivative of equation (58) along the solution of the Equations $(6)-(10)$, we have

$$\dot{Y} = (1 - \frac{S^{**}(t)}{S(t)})\dot{S} + (1 - \frac{E^{**}(t)}{E(t)})\dot{E} + (\frac{k+\mu}{k})(1 - \frac{I^{**}(t)}{I(t)})\dot{I}. \tag{59}$$

By direct substitution of Equations $(6)-(10)$ in (59), we obtain

$$\begin{aligned}
(1 - \frac{S^{**}(t)}{S(t)})\dot{S} &= (1 - \frac{S^{**}(t)}{S(t)})[\Lambda - \frac{\beta I(t)^{**}}{(1+\theta I(t)^{**})}(1-u_1)S(t)^{**} - (\psi_1+\mu)S(t)^{**} + \delta R(t)^{**}] \\
&= (1 - \frac{S^{**}(t)}{S(t)})[\frac{\beta I^{**}(t)}{(1+\theta I^{**}(t))}S^{**}(t) + (\psi_1(t)+\mu)S^{**}(t) \\
&\quad - \delta R^{**}(t)] - \frac{\beta I(t)^{**}}{(1+\theta I(t)^{**})}(1-u_1)S(t)^{**} + (\psi_1+\mu)S(t) - \delta R(t)^{**}] \\
&= \frac{\beta I^{**}(t)S^{**}(t)}{1+\theta I^{**}(t)}(1-u_1)(1 - \frac{S^{**}(t)}{S(t)}) + (\psi_1+\mu)S^{**}(t)(1 - \frac{S^{**}(t)}{S(t)}) \\
&\quad - \delta R^{**}(t)(1 - \frac{S^{**}(t)}{S(t)}) - \frac{\beta I(t)S(t)}{1+\theta I(t)}(1-u_1)(1 - \frac{S^{**}(t)}{S(t)})
\end{aligned} \tag{60}$$

$$\begin{aligned}
&- (\psi_1(t)+\mu)S(t)(1 - \frac{S^{**}(t)}{S(t)}) \\
&- \delta R(t)(1 - \frac{S^{**}(t)}{S(t)}) \\
&= (\psi_1(t)+\mu)S^{**}(t)(2 - \frac{S^{**}(t)}{S(t)} - \frac{S(t)}{S^{**}(t)}) + (1 - \frac{S^{**}(t)}{S(t)})(\frac{\beta I^{**}(t)S^{**}(t)}{1+\theta I^{**}(t)} \\
&(1-u_1) - \frac{\beta I(t)S(t)}{1+\theta I(t)}(1-u_1))
\end{aligned}$$

$$\begin{aligned}
(1 - \frac{E^{**}(t)}{E(t)})\dot{E} &= (1 - \frac{E^{**}(t)}{E(t)})[\frac{\beta I(t)S(t)}{1+\theta I(t)}(1-u_1) - (k+\mu)E(t)] \\
&= (1 - \frac{E^{**}(t)}{E(t)})[\frac{\beta I(t)S(t)}{1+\theta I(t)}(1-u_1) - \frac{\beta I^{**}(t)S^{**}(t)}{1+\theta I^{**}(t)}(1-u_1)\frac{E(t)}{E^{**}(t)}] \\
&= \frac{\beta I(t)S(t)}{1+\theta I(t)}(1-u_1) - \frac{\beta I^{**}(t)S^{**}(t)}{1+\theta I^{**}(t)}(1-u_1)\frac{E(t)}{E^{**}(t)} \\
&\quad - \frac{\beta I(t)S(t)}{1+\theta I(t)}(1-u_1)\frac{E(t)}{E^{**}(t)} + \frac{\beta I^{**}(t)S^{**}(t)}{1+\theta I^{**}(t)}(1-u_1).
\end{aligned} \tag{61}$$

$$
\begin{aligned}
&(\frac{k+\mu}{k})(1-\frac{I^{**}(t)}{I(t)})\dot{I} = (\frac{k+\mu}{k})(1-\frac{I^{**}(t)}{I(t)})[k(1-u_2)E(t)-(\alpha+\mu+\gamma+\psi_2)I(t)]\\
&= (k(1-u_2)+\mu)(1-\frac{I^{**}(t)}{I(t)})E(t) - \frac{k+\mu}{k(1-u_2)}(\alpha+\mu+\gamma+\psi_2(1-\frac{I^{**}(t)}{I(t)})I(t)\\
&= \frac{\beta I^{**}(t)S^{**}(t)}{1+\theta I^{**}(t)}(1-u_1)\frac{E(t)}{E^{**}(t)}(1-\frac{I^{**}(t)}{I(t)}) - \frac{\beta I^{**}(t)S^{**}(t)}{1+\theta I^{**}(t)}(1-u_1)\frac{I(t)}{I^{**}(t)}(1-\frac{I^{**}(t)}{I(t)})\\
&= \frac{\beta I^{**}(t)S^{**}(t)}{1+\theta I^{**}(t)}\frac{E(t)}{E^{**}(t)} - \frac{\beta I^{**}(t)S^{**}(t)}{1+\theta I^{**}(t)}\frac{E(t)}{E^{**}(t)}\frac{I^{**}(t)}{I(t)}\\
&- \frac{\beta I^{**}(t)S^{**}(t)}{1+\theta I^{**}(t)}\frac{I(t)}{I^{**}(t)} + \frac{\beta I^{**}(t)S^{**}(t)}{1+\theta I^{**}(t)}
\end{aligned}
\tag{62}
$$

It follows from $(60-62)$ that

$$
\begin{aligned}
\dot{Y} = (\psi_1+\mu)S^{**}(t)&\left(2-\frac{S^{**}(t)}{S(t)}-\frac{S(t)}{S^{**}(t)}\right)\\
&+\frac{\beta I^{**}(t)S^{**}(t)}{1+\theta I^{**}(t)}(1-u_1)\left(3-\frac{S^{**}(t)}{S(t)}-\frac{E(t)I^{**}(t)}{E^{**}I(t)}-\frac{I(t)}{I^{**}(t)}\right)\\
&+\frac{\beta I(t)S^{**}(t)}{1+\theta I(t)}(1-u_1)\left(1-\frac{S(t)E^{**}(t)}{S^{**}(t)E(t)}\right)
\end{aligned}
\tag{63}
$$

From equation (63), we have

$$
\begin{aligned}
\left(2-\frac{S^{**}(t)}{S(t)}-\frac{S(t)}{S^{**}(t)}\right)&\leq 0,\\
\left(3-\frac{S^{**}(t)}{S(t)}-\frac{E(t)I^{**}(t)}{E^{**}I(t)}-\frac{I(t)}{I^{**}(t)}\right)&\leq 0,\\
\left(1-\frac{S(t)E^{**}(t)}{S^{**}(t)E(t)}\right)&\leq 0.
\end{aligned}
\tag{64}
$$

Thus, the condition (64) implies that $\dot{Y}\leq 0$, for $(S(t),V(t),E(t),I(t),R(t))\in\Omega$. Then, the equilibrium, E_2^* is globally asymptotically stable on Ω.

Remark 3. The occurrence of this condition implies that the influenza disease becomes endemic globally. When this happens, it will claim many lives and may also put the world in recession as similar to the novel corona virus disease 2019 (COVID 19 Pandemic).

3.8. **Sensitivity Analysis.** In this section, we performed the sensitivity analysis in order to determine the relative importance of the model parameters on disease transmission. The analysis will enable us to find out parameters that have high impact on the basic reproduction number and which should be targeted for intervention strategies. We perform sensitivity analysis by calculating the sensitivity indices of the effective reproduction number R_0 since our major emphasis is on the invasive plants in order to determine whether malaria fever can be controlled or not. These indices tell us how crucial each parameter is on the transmission of malaria fever. The sensitivity index of R_{01} to a parameter say τ, where τ is any of the parameters in Table 3 is given by

$$
\Gamma_{\tau}^{R_0} = \frac{\partial R_0}{\partial \tau}\times\frac{\tau}{R_0},
$$

where

$$\Gamma_{\beta}^{R_0} = \Gamma_{\Lambda}^{R_0} 1 = \Gamma_{\sigma}^{R_0} = 1,$$
$$\Gamma_{k}^{R_0} = \frac{\mu}{k\left(-u_2\right) + k + \mu},$$
$$\Gamma_{u_2}^{R_0} = \frac{\mu u_2}{\left(u_2 - 1\right)\left(k\left(-u_2\right) + k + \mu\right)},$$
$$\Gamma_{\Psi_1}^{R_0} = \frac{\mu}{\mu + \psi_1},$$
$$\Gamma_{\mu}^{R_0} = \frac{d_1}{d_2},$$
$$\Gamma_{\Psi_2}^{R_0} = -\frac{\psi_2}{\alpha + \gamma + \mu + \psi_2},$$
$$\Gamma_{\alpha}^{R_0} = -\frac{\alpha}{\alpha + \gamma + \mu + \psi_2},$$
$$\Gamma_{\gamma}^{R_0} = -\frac{\gamma}{\alpha + \gamma + \mu + \psi_2}.$$

where

$$d_1 = \psi_1\left(\mu(2\alpha + 2\gamma + 3\mu) + k(\alpha + \gamma + 2\mu) + \psi_2(k + 2\mu)\right) + \mu\left(\mu(3\alpha + 3\gamma + 4\mu)\right.$$
$$+ k(2\alpha + 2\gamma + 3\mu) + \psi_2(2k + 3\mu) - ku_2\left(\psi_1\left(\alpha + \gamma + 2\mu + \psi_2\right).\right.$$
$$\left.+ \mu\left(2\alpha + 2\gamma + 3\mu + 2\psi_2\right)\right.$$
$$d_2 = \left(\mu + \psi_1\right)\left(k\left(-u_2\right) + k + \mu\right)\left(\alpha + \gamma + \mu + \psi_2\right).$$

4. Model Simulation

In this section, we use the inbuilt MATLAB function ode 45 to solve the modified model equations above. The graphical user interface in the MATLAB version 7.5 was used for the solution method, simulation and visualization on graphs. We made use of the parameters of the model and their values from related literature and we assumed some parameters values that are not found in literature. The values used here are as contained in Table 4. The Matlab code used is in Appendix C.

TABLE 3. **Parameter values of the Model Equation**

Parameter	**Value**	**References**
Λ_h	100	Assumed
β	0.005/day	Modnak (2017)[15]
σ	0.699/day	Modnak (2017)[15]
k	0.00015(0-0.8)	Varied
u_2	0.2(0-0.8)	Varied
ψ_1	0.7	Modnak (2017)[15]
μ	0.00197	Assumed
ψ_2	0.7	Assumed
α	0.03	Modnak (2017)[15]
γ	0.36	Modnak (2017)[15]

4.1. **Results from the Analysis of the Model Equations.** The modified model equations positive region was proved to exist within the domain, $D = \{(S(t), V(t), E(t), I(t), R(t)) \in \Re^{5+} : S(t) > 0, V(t) \geq 0, E(t) \geq 0, I(t) \geq 0, R(t) \geq 0 : N(t) \leq \frac{\Lambda}{\mu}$. The model has two equilibria namely, the disease-free equilibrium and the disease endemic equilibrium denoted as E_1 and E_2 respectively. In the case of E_1, the susceptible and the vaccinated humans or populations exist with intake of the vaccines. In the case of E_2 equilibrium, all the sub-populations coexist.

The stability analysis of the E_1 equilibrium shows that the system is locally asymptotically stable provided $R_0 < 1$ and unstable if otherwise. The E_2 equilibrium is locally asymptotically stable provided the given Routh Hurwitz stability conditions stated in Theorem 3.5 is satisfied. The model global stability was proved by the use of Lyapunov function.

We also carried out the sensitivity analysis of the modified model by taking partial derivatives of the R_0 with respect to each of the model parameters. We found that $\beta, k, \sigma, \Lambda$ and ψ_1 are the most sensitive parameters. This implies that any increase in any of these parameters' value will lead to increase of the model basic reproduction number R_0, and thereby trigger the disease infection.

TABLE 4. **Parameter values of the model equations**

Parameter	**Value**	**Sensitivity Index**
Λ	100	+1.0000
β	0.005/day	+1.0000
σ	0.699/day	+1.0000
k	0.00015(0-0.8)	+0.942584
u_2	0.2(0-0.8)	0.0028064
ψ_1	0.7	+0.0001343
μ	0.00197	-0.2356460
ψ_2	0.7	-0.6410430
α	0.03	-0.0274730
γ	0.36	-0.3296790

4.2. **Results from the Sensitivity Analysis.** The most sensitive parameters are $\lambda_h, \beta, \sigma, k, \psi_1$ and μ. Any increment in the values of the parameters with negative indices would reduce the value of the basic reproduction number, also any increment in the value of the parameters having positive indices will increase the endemicity of the disease. The basic reproduction number increases in all cases as all these parameters increases, which means that all these parameters have to be kept under control in order to have a complete eradication of avian influenza.

4.3. **Numerical Simulation.** In this section, we shall use the parameters values given in Table 4 to carryout our numerical simulation as follows.

5. CONCLUSIONS AND DISCUSSIONS

We present a mathematical model on avian influenza with detailed analysis. We basically considered human population only and ignored the bird population as a result of our interest in the dynamics of avian influenza on human population. Initially, we presented a detailed mathematical results of the model equations. The results obtained show that

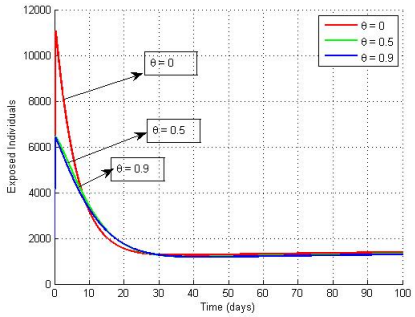

FIGURE 3. Plots of Exposed Individuals over time at different values of inhibition rate, θ

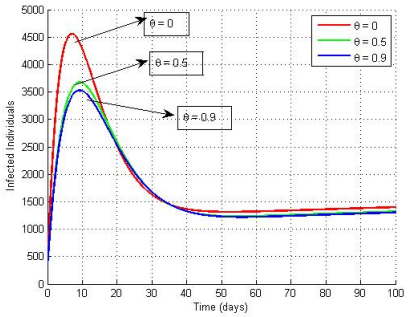

FIGURE 4. Plots of Infected Individuals over time at different values of Inhibition Rate, θ

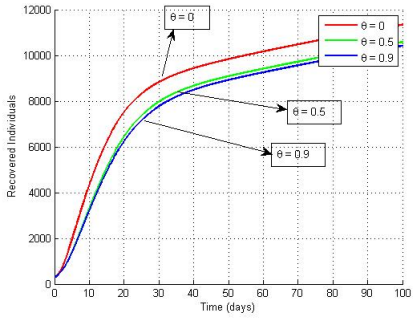

FIGURE 5. Plots of Recovered Individuals over time at different values of Inhibition Rate, θ

the model is stable both locally and globally under some certain conditions. The stability results for disease free equilibrium is obtained when $R_0 < 1$. If $R_0 > 1$, we proved that the endemic equilibrium of the model is both locally, globally asymptotically stable under some certain conditions.

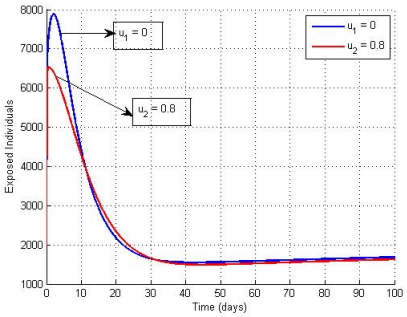

FIGURE 6. Plots of Exposed Individuals over time at different values of Control Rate, u_1

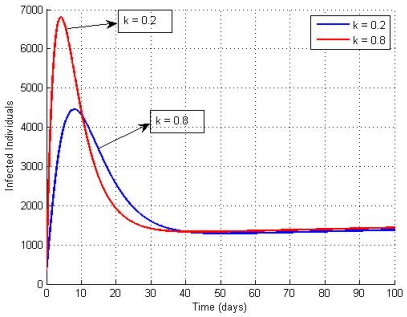

FIGURE 7. Plots of Infected Individuals over time at different values of Treatment Rate, k

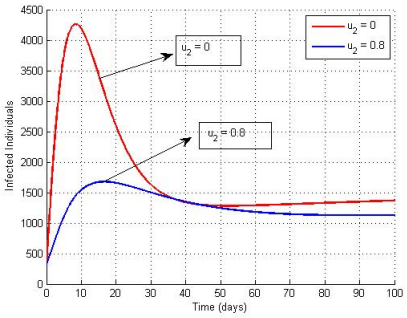

FIGURE 8. Plots of Infected Individuals over time at different values of Control Rate, u_2

The numerical results of the model is obtained and is given in Figure 3 - 9. The numerical results validate that the transmission dynamics of the avian influenza which is determined by force of infections. It is observed that the parameter the saturation effect, θ and the contact between infected humans to susceptible humans do not change the stability

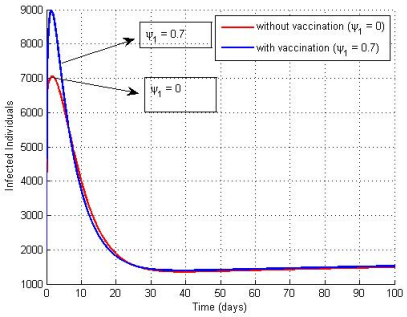

FIGURE 9. Plots of Exposed Individuals over time at different values of Vaccination Rate, ψ_1

of the equilibria and so that the outbreaks, as the infected humans do not spread the virus further. Figure 3 shows the dynamics of avian influenza disease with respect to exposed individual by varying the inhibition rate parameter, θ. The exposed individuals population decreases with increase in θ and vice versa within the first ten (10) days.

In Figure 4, we show the dynamics of the disease with respect to infected population by varying the inhibition rate, θ. The infected population decreases with the increase in θ value and vice versa for 20 - 30 days. However, it is interesting to know here that the inhibition rate, θ reduced the infected population from 4,500 to about 1,250 (i.e. decline or reduction of infected population by 1, 350).

In Figure 5, we plot the recovered population over time. We observed that the recovered population increases with decrease of the inhibition rate, θ. This is expected to happen since the waiting time in exposed and infected compartment is ignored or reduced.

Figure 6 shows the plot of exposed individuals over time by varying the control parameter value, u_1 which represents the control of susceptible individuals from getting or moving into the exposed compartment. It was found that the higher the control rate the lower the exposed population becomes over time.

Figure 7 describes the plot of infected individuals over time with respect to varying the parameter, k which represents the progression rate of exposed individuals to infected compartment. It was found that the higher the progression rate value the higher the growth of the infected individuals population over time and vice versa.

In Figure 8, we vary u_2 which represents the control of exposed individuals from becoming infected. The control was achieved significantly from day 0 to day 35. An equilibrium point is observed from day 35 to day 50.

Figure 9 shows variation of the vaccination parameter, ψ_1. With vaccination, the exposed population declined drastically. It was found that vaccination of individuals is paramount at the early stage of the disease occurrence.

Following the numerical results obtained from this study, we found out that the results are in agreement with the works of Jagan and Kartheek (2016) [11], Caroline *et al.* (2018)[3], and Khan *et al.* (2019)[12] who observed that vaccination, force of infection and contact with infected birds are the major ways the disease can be eliminated. However, Caroline *et al.* (2018)observed that vaccination alone cannot eliminate the disease despite the numerical analysis shows that the basic reproduction number was reduced to less than unity.

In conclusion, the numerical results show that increasing the vaccination parameter and the treatment parameter decrease the exposed humans and the infected humans respectively and can help to control the disease.

In this study we have formulated a non-autonomous mathematical model that study the dynamics of an avian influenza with saturated incidence rate by modifying Modnak (2017)[15]. The model exhibits two equilibria, namely, the disease free equilibrium (DFE) and the disease endemic equilib- rium (DEE). The disease threshold that is, the basic reproduction number was computed using the next generation matrix method. The DFE is locally asymptotically stable when $R_0 < 1$ and the endemic equilibrium is both locally and globally stable when $R_0 > 1$. The disease can be eradicated provided the efficacy of the vaccine is high and the administration is up to 80% - 90% of the susceptible individuals and the treatment of the infected individuals are seriously observed.

6. ACKNOWLEDGEMENTS

The authors acknowledged the Department of Mathematics, College Physical Sciences, Joseph Sarwuan Tarka University, Makurdi, for providing enabling environment for the success of this research work.

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APPENDICES

Appendix A. $(J(E_1)-xI) = \{\{-\mu-x, 0, 0, 0, \delta\}, \{\psi_1, -\mu, 0, -\frac{\beta\Lambda\sigma\psi_1}{\mu(\mu+\psi_1)}, 0\}, \{0, 0, -k(1-u_2)$
$-\mu-x, \frac{\beta\Lambda\sigma\psi_1}{\mu(\mu+\psi_1)}, 0\}, \{0, 0, k(1-u_2), -\alpha-\gamma-\mu-x-_2, 0\}, \{0, 0, 0, \gamma+\psi_2, -\delta-\mu-x\}\}$

$characteristicPolynomial[\%1, x]$

$Collect[\%2, x]$

Appendix B. $(J(E_2)-Iy) = \{\{P_1-u_1-\psi_1-y, 0, 0, P_2, \delta\}, \{\psi_1, -\mu-P_3-y, 0, -\beta H^*\sigma, 0\}, \{P_4, P_5, c_1-y, \beta H^*\sigma + P_6, 0\}, \{0, 0, k(1-u_2), c_2-y, 0\}, \{0, 0, 0, \gamma+\psi_2, -\delta-\mu-y\}\}$

$characteristicPolynomial[\%3, y]$

$Collect[\%4, y]$

Appendix C. $functiond_y = umoru(t, y)$
$d_y = zeros(5, 1);$
$Lambda = 100; mu = 0.001917; beta = 0.04; theta = 0.9; gamma = 0.036;$
$delta = 0.01; xi = 0.05; sigma = (1-xi); k = 0.15; alpha = 0.03;$
$psi_1 = 0.09; psi_2 = 0.08;$

u1 = 0.2; u2 =0; lambda = beta*y(4)/(1+theta*y(4));
dy(1)= Lambda - lambda*(1-u1)*y(1)-(psi$_1$ + mu) $* y(1) + delta * y(5);$
$dy(2) = psi_1 * y(1) - sigma * beta * y(4) * y(2) - mu * y(2);$
$dy(3) = sigma*beta*y(4)*y(2)+lambda*(1-u1)*y(1)-(k*(1-u2)+mu)*y(3);$
$dy(4) = k * (1-u2) * y(3) - (alpha + mu + gamma + psi_2) * y(4);$
$dy(5) = psi_2 * y(4) + gamma * y(4) - (delta + mu) * y(5);$

```
T,Y
= ode45('umoru', [0, 100], [500045002000340300]);
y1 = Y(:, 1);
y2 = Y(:, 2);
y3 = Y(:, 3);
y4 = Y(:, 4);
y5 = Y(:, 5);

gridon
holdon
plot(T, y3,' r',' Linewidth', 2)
xlabel('Time(days)')
ylabel('ExposedIndividuals')
legend('θ = 0',' θ = 0.5',' θ = 0.9')
subplot(2, 3, 1); plot(y4, y1,' Linewidth', 1)
subplot(2, 3, 1); xlabel('Time(hour)')
subplot(2, 3, 1); ylabel('FishPopulation')
subplot(2, 3, 2); plot(T, y2,' Linewidth', 1)
subplot(2, 3, 2); xlabel('Time(hour)')
subplot(2, 3, 2); ylabel('Pesticidesinwater')
subplot(2, 3, 3); plot(T, y3,' Linewidth', 1)
subplot(2, 3, 3); xlabel('Time(hour)')
subplot(2, 3, 3); ylabel('Pesticidesinsediment')
subplot(2, 3, 4); plot(T, y4,' Linewidth', 1)
subplot(2, 3, 4); xlabel('Time(hour)')
subplot(2, 3, 4); ylabel('Uptakeconcentration')
subplot(2, 3, 5); plot(T, y5,' Linewidth', 1)
subplot(2, 3, 5); xlabel('Time(hour)')
subplot(2, 3, 5); ylabel('Uptakeconcentration')
```

APPENDIX D

$$
\begin{aligned}
\Phi_1 = {} & \beta k\left(u_2-1\right)\left(\alpha+\gamma+\theta k-\theta k u_2+\mu+\psi_2\right)\left(\alpha\delta\Lambda+\alpha\Lambda\mu+\gamma\delta\Lambda+\gamma\Lambda\mu\right. \\
& +\delta\Lambda\mu+\delta\Lambda\psi_2+\gamma\delta k+\delta k\psi_2-\gamma\delta k u_2-\delta k u_2\psi_2+\Lambda\mu^2+\Lambda\mu\psi_2 \\
& (-\alpha\mu-\alpha\sigma\psi_1-\gamma\mu-\gamma\sigma\psi_1-\beta k\sigma-\theta k\sigma\psi_1+\beta k\sigma u_1+\beta k\sigma u_2 \\
& -\beta k\sigma u_1u_2+\theta k\sigma u_2\psi_1-\mu^2-\mu\sigma\psi_1-\mu\psi_2-\sigma\psi_1\psi_2+\alpha\mu u_1+\gamma\mu u_1+\mu^2u_1+\mu u_1\psi_2 \\
\Phi_2 = {} & (\delta+\mu)\left(k\left(-u_2\right)+k+\mu\right)\left(\alpha+\gamma+\mu+\psi_2\right)^2\left(\alpha\psi_1+\gamma\psi_1+\beta k+\theta k\psi_1\right. \\
& -\beta k u_1-\beta k u_2+\beta k u_1u_2-\theta k u_2\psi_1+\theta k u_1-\theta k u_1u_2+\mu\psi_1+\alpha u_1+\gamma u_1+\mu u_1+u_1\psi_2 \\
& +\psi_1\psi_2\left(\alpha\mu+\gamma\mu+\beta k\sigma-\beta k\sigma u_2+\mu^2+\mu\psi_2\right) \\
& L_1 = \phi_1\left(\alpha+\gamma+\theta k-\theta k u_2+\mu+\psi_2\right)\left(\alpha\delta\Lambda+\alpha\Lambda\mu+\gamma\delta\Lambda+\gamma\Lambda\mu\right. \\
& +\delta\Lambda\mu+\delta\Lambda\psi_2+\gamma\delta k+\delta k\psi_2-\gamma\delta k u_2-\delta k u_2\psi_2+\Lambda\mu^2+\Lambda\mu\psi_2
\end{aligned}
$$

$$L_2 = (\delta+\mu)(\alpha\psi_1+\gamma\psi_1+\beta k+\theta k\psi_1-\beta k u_1-\beta k u_2+\beta k u_1u_2-\theta k u_2\psi_1 + \theta k u_1-\theta k u_1u_2+\mu\psi_1+\alpha u_1+\gamma u_1+\mu u_1+u_1\psi_2+\psi_1\psi_2(\alpha\mu+\gamma\mu + \beta k\sigma-\beta k\sigma u_2+\mu^2+\mu\psi_2)$$

$$K_1 = (\alpha+\gamma+\theta k-\theta k u_2+\mu+\psi_2)(\alpha\delta\Lambda+\alpha\Lambda\mu+\gamma\delta\Lambda+\gamma\Lambda\mu+\delta\Lambda\mu+\delta\Lambda\psi_2 + \gamma\delta k+\delta k\psi_2-\gamma\delta k u_2-\delta k u_2\psi_2+\Lambda\mu^2+\Lambda\mu\psi_2$$

$$K_2 = (\delta+\mu)(\alpha+\gamma+\mu+\psi_2)(\alpha\psi_1+\gamma\psi_1+\beta k+\theta k\psi_1-\beta k u_1-\beta k u_2+\beta k u_1u_2 - \theta k u_2\psi_1+\theta k u_1-\theta k u_1u_2+\mu\psi_1+\alpha u_1+\gamma u_1+\mu u_1+u_1\psi_2+\psi_1\psi_2$$

Appendix E.

$$a_1 = \alpha+\gamma+\delta+k(1-u_2)+4\mu+P_1+P_3+u_1+\psi_1+\psi_2$$

$$a_2 = -(\alpha\delta-3\alpha\mu-\alpha\psi_1-\gamma\delta-3\gamma\mu-\gamma\psi_1-3\delta\mu-\delta\psi_1-\delta\psi_2+k(1-u_2)(\beta H^*\sigma+P_6)+ kP_1(1-u_2)-kP_3(1-u_2)-\alpha k(1-u_2)-\gamma k(1-u_2)-\delta k(1-u_2)-3k\mu(1-u_2)- k(1-u_2)\psi_1-k(1-u_2)\psi_2-ku_1(1-u_2)-6\mu^2-4\mu\psi_1-3\mu\psi_2+\alpha P_1-\alpha P_3+ \gamma P_1-\gamma P_3+\delta P_1-\delta P_3+4\mu P_1-3\mu P_3-P_3u_1-P_3\psi_1+P_1\psi_2-P_3\psi_2+P_1P_3-\alpha u_1- \gamma u_1-\delta u_1-4\mu u_1-u_1\psi_2-\psi_1\psi_2)$$

$$a_3 = -(4\mu^3-3\alpha\mu^2-3\gamma\mu^2-3\delta\mu^2+6P_1\mu^2-3P_3\mu^2-6u_1\mu^2-3k(1-u_2)\mu^2-6\psi_1\mu^2- 3\psi_2\mu^2-2\alpha\delta\mu-2\gamma\delta\mu+3\alpha P_1\mu+3\gamma P_1\mu+3\delta P_1\mu-2\alpha P_3\mu-2\gamma P_3\mu-2\delta P_3\mu+3P_1P_3\mu- 3\alpha u_1\mu-3\gamma u_1\mu-3\delta u_1\mu-3P_3u_1\mu-2k\alpha(1-u_2)\mu-2k\gamma(1-u_2)\mu-2k\delta(1-u_2)\mu+ 3kP_1(1-u_2)\mu-2kP_3(1-u_2)\mu-3ku_1(1-u_2)\mu-3\alpha\psi_1\mu-3\gamma\psi_1\mu-3\delta\psi_1\mu- 3P_3\psi_1\mu-3k(1-u_2)\psi_1\mu-2\delta\psi_2\mu+3P_1\psi_2\mu-2P_3\psi_2\mu-3u_1\psi_2\mu-2k(1-u_2)\psi_2\mu- 3\psi_1\psi_2\mu+2k(1-u_2)(P_6+\beta\sigma H^*)\mu+\alpha\delta P_1+\gamma\delta P_1-\alpha\delta P_3-\gamma\delta P_3+\alpha P_1P_3+\gamma P_1P_3+ \delta P_1P_3-\alpha\delta u_1-\gamma\delta u_1-\alpha P_3u_1-\gamma P_3u_1-\delta P_3u_1-k\alpha\delta(1-u_2)-k\gamma\delta(1-u_2)+ k\alpha P_1(1-u_2)+k\gamma P_1(1-u_2)+k\delta P_1(1-u_2)-k\alpha P_3(1-u_2)-k\gamma P_3(1-u_2)- k\delta P_3(1-u_2)+kP_1P_3(1-u_2)+kP_2P_4(1-u_2)-k\alpha u_1(1-u_2)-k\gamma u_1(1-u_2)- k\delta u_1(1-u_2)-kP_3u_1(1-u_2)-\alpha\delta\psi_1-\gamma\delta\psi_1-\alpha P_3\psi_1-\gamma P_3\psi_1-\delta P_3\psi_1-k\alpha(1-u_2)\psi_1- k\gamma(1-u_2)\psi_1-k\delta(1-u_2)\psi_1-kP_3(1-u_2)\psi_1+\delta P_1\psi_2-\delta P_3\psi_2+P_1P_3\psi_2-\delta u_1\psi_2- P_3u_1\psi_2-k\delta(1-u_2)\psi_2+kP_1(1-u_2)\psi_2-kP_3(1-u_2)\psi_2-ku_1(1-u_2)\psi_2- \delta\psi_1\psi_2-P_3\psi_1\psi_2-k(1-u_2)\psi_1\psi_2-k\beta\sigma P_5(1-u_2)H^*+ k\delta(1-u_2)(P_6+\beta\sigma H^*)-kP_1(1-u_2)(P_6+\beta\sigma H^*)+kP_3(1-u_2)(P_6+\beta\sigma H^*)+ ku_1(1-u_2) (P_6+\beta\sigma H^*)+k(1-u_2)\psi_1(P_6+\beta\sigma H^*)$$

$$a_4 = -(\mu^4-\alpha\mu^3-\gamma\mu^3-\delta\mu^3+4P_1\mu^3-P_3\mu^3-4u_1\mu^3-k(1-u_2)\mu^3-4\psi_1\mu^3- \psi_2\mu^3-\alpha\delta\mu^2-\gamma\delta\mu^2+3\alpha P_1\mu^2+3\gamma P_1\mu^2+3\delta P_1\mu^2-\alpha P_3\mu^2-\gamma P_3\mu^2-\delta P_3\mu^2+ 3P_1P_3\mu^2-3\alpha u_1\mu^2-3\gamma u_1\mu^2-3\delta u_1\mu^2-3P_3u_1\mu^2-k\alpha(1-u_2)\mu^2-k\gamma(1-u_2)\mu^2- k\delta(1-u_2)\mu^2+3kP_1(1-u_2)\mu^2-kP_3(1-u_2)\mu^2-3ku_1(1-u_2)\mu^2-3\alpha\psi_1\mu^2- 3\gamma\psi_1\mu^2-3\delta\psi_1\mu^2-3P_3\psi_1\mu^2-3k(1-u_2)\psi_1\mu^2-\delta\psi_2\mu^2+3P_1\psi_2\mu^2-P_3\psi_2\mu^2- 3u_1\psi_2\mu^2-k(1-u_2)\psi_2\mu^2-3\psi_1\psi_2\mu^2+k(1-u_2)(P_6+\beta\sigma H^*)\mu^2+2\alpha\delta P_1\mu+2\gamma\delta P_1\mu- \alpha\delta P_3\mu-\gamma\delta P_3\mu+2\alpha P_1P_3\mu+2\gamma P_1P_3\mu+2\delta P_1P_3\mu-2\alpha\delta u_1\mu-2\gamma\delta u_1\mu-2\alpha P_3u_1\mu- 2\gamma P_3u_1\mu-2\delta P_3u_1\mu-k\alpha\delta(1-u_2)\mu-k\gamma\delta(1-u_2)\mu+2k\alpha P_1(1-u_2)\mu+2k\gamma P_1(1-u_2)\mu+ 2k\delta P_1(1-u_2)\mu-k\alpha P_3(1-u_2)\mu-k\gamma P_3(1-u_2)\mu-k\delta P_3(1-u_2)\mu+2kP_1P_3(1-u_2)\mu+ 2kP_2P_4(1-u_2)\mu-2k\alpha u_1(1-u_2)\mu-2k\gamma u_1(1-u_2)\mu-2k\delta u_1(1-u_2)\mu-2kP_3u_1(1-u_2)\mu- 2\alpha\delta\psi_1\mu-2\gamma\delta\psi_1\mu-2\alpha P_3\psi_1\mu-2\gamma P_3\psi_1\mu-2\delta P_3\psi_1\mu-2k\alpha(1-u_2)\psi_1\mu-2k\gamma(1-u_2)\psi_1\mu- 2k\delta(1-u_2)\psi_1\mu-2kP_3(1-u_2)\psi_1\mu+2\delta P_1\psi_2\mu-\delta P_3\psi_2\mu+2P_1P_3\psi_2\mu-2\delta u_1\psi_2\mu- 2P_3u_1\psi_2\mu-k\delta(1-u_2)\psi_2\mu+2kP_1(1-u_2)\psi_2\mu-kP_3(1-u_2)\psi_2\mu-2ku_1(1-u_2)\psi_2\mu-$$

$2\delta\psi_1\psi_2\mu-2P_3\psi_1\psi_2\mu-2k\left(1-u_2\right)\psi_1\psi_2\mu-k\beta\sigma P_5\left(1-u_2\right)H^*\mu+k\delta\left(1-u_2\right)\left(P_6+\beta\sigma H^*\right)\mu-2kP_1\left(1-u_2\right)\left(P_6+\beta\sigma H^*\right)\mu+kP_3\left(1-u_2\right)\left(P_6+\beta\sigma H^*\right)\mu+2ku_1\left(1-u_2\right)\left(P_6+\beta\sigma H^*\right)\mu+2k\left(1-u_2\right)\psi_1\left(P_6+\beta\sigma H^*\right)\mu+\alpha\delta P_1P_3+\gamma\delta P_1P_3-\alpha\delta P_3u_1-\gamma\delta P_3u_1+k\alpha\delta P_1\left(1-u_2\right)+k\gamma\delta P_1\left(1-u_2\right)-k\alpha\delta P_3\left(1-u_2\right)-k\gamma\delta P_3\left(1-u_2\right)+k\alpha P_1P_3\left(1-u_2\right)+k\gamma P_1P_3\left(1-u_2\right)+k\delta P_1P_3\left(1-u_2\right)+k\delta P_2P_4\left(1-u_2\right)+kP_2P_3P_4\left(1-u_2\right)-k\alpha\delta u_1\left(1-u_2\right)-k\gamma\delta u_1\left(1-u_2\right)-k\alpha P_3u_1\left(1-u_2\right)-k\gamma P_3u_1\left(1-u_2\right)-k\delta P_3u_1\left(1-u_2\right)-\alpha\delta P_3\psi_1-\gamma\delta P_3\psi_1-k\alpha\delta\left(1-u_2\right)\psi_1-k\gamma\delta\left(1-u_2\right)\psi_1-k\alpha P_3\left(1-u_2\right)\psi_1-k\gamma P_3\left(1-u_2\right)\psi_1-k\delta P_3\left(1-u_2\right)\psi_1+kP_2P_5\left(1-u_2\right)\psi_1+\delta P_1P_3\psi_2-\delta P_3u_1\psi_2+k\delta P_1\left(1-u_2\right)\psi_2-k\delta P_3\left(1-u_2\right)\psi_2+kP_1P_3\left(1-u_2\right)\psi_2-k\delta u_1\left(1-u_2\right)\psi_2-kP_3u_1\left(1-u_2\right)\psi_2-\delta P_3\psi_1\psi_2-k\delta\left(1-u_2\right)\psi_1\psi_2-kP_3\left(1-u_2\right)\psi_1\psi_2+k\delta P_4\left(1-u_2\right)\left(\gamma+\psi_2\right)-k\beta\delta\sigma P_5\left(1-u_2\right)H^*+k\beta\sigma P_1P_5\left(1-u_2\right)H^*-k\beta\sigma P_5u_1\left(1-u_2\right)H^*-k\beta\sigma P_5\left(1-u_2\right)\psi_1H^*-k\delta P_1\left(1-u_2\right)\left(P_6+\beta\sigma H^*\right)+k\delta P_3\left(1-u_2\right)\left(P_6+\beta\sigma H^*\right)-kP_1P_3\left(1-u_2\right)\left(P_6+\beta\sigma H^*\right)+k\delta u_1\left(1-u_2\right)\left(P_6+\beta\sigma H^*\right)+kP_3u_1\left(1-u_2\right)\left(P_6+\beta\sigma H^*\right)+k\delta\left(1-u_2\right)\psi_1\left(P_6+\beta\sigma H^*\right)+kP_3\left(1-u_2\right)\psi_1\left(P_6+\beta\sigma H^*\right)$

$a_5=-P_1\mu^4+u_1\mu^4+\psi_1\mu^4-\alpha P_1\mu^3-\gamma P_1\mu^3-\delta P_1\mu^3-P_1P_3\mu^3+\alpha u_1\mu^3+\gamma u_1\mu^3+\delta u_1\mu^3+P_3u_1\mu^3-kP_1\left(1-u_2\right)\mu^3+ku_1\left(1-u_2\right)\mu^3+\alpha\psi_1\mu^3+\gamma\psi_1\mu^3+\delta\psi_1\mu^3+P_3\psi_1\mu^3+k\left(1-u_2\right)\psi_1\mu^3-\alpha\delta P_1\mu^2-\gamma\delta P_1\mu^2-\alpha P_1P_3\mu^2-\gamma P_1P_3\mu^2-\delta P_1P_3\mu^2+\alpha\delta u_1\mu^2+\gamma\delta u_1\mu^2+\alpha P_3u_1\mu^2+\gamma P_3u_1\mu^2+\delta P_3u_1\mu^2-k\alpha P_1\left(1-u_2\right)\mu^2-k\gamma P_1\left(1-u_2\right)\mu^2-k\delta P_1\left(1-u_2\right)\mu^2-kP_1P_3\left(1-u_2\right)\mu^2-kP_2P_4\left(1-u_2\right)\mu^2+k\alpha u_1\left(1-u_2\right)\mu^2+k\gamma u_1\left(1-u_2\right)\mu^2+k\delta u_1\left(1-u_2\right)\mu^2+kP_3u_1\left(1-u_2\right)\mu^2+\alpha\delta\psi_1\mu^2+\gamma\delta\psi_1\mu^2+\alpha P_3\psi_1\mu^2+\gamma P_3\psi_1\mu^2+\delta P_3\psi_1\mu^2+k\alpha\left(1-u_2\right)\psi_1\mu^2+k\gamma\left(1-u_2\right)\psi_1\mu^2+k\delta\left(1-u_2\right)\psi_1\mu^2+kP_3\left(1-u_2\right)\psi_1\mu^2-\alpha\delta P_1P_3\mu-\gamma\delta P_1P_3\mu+\alpha\delta P_3u_1\mu+\gamma\delta P_3u_1\mu-k\alpha\delta P_1\left(1-u_2\right)\mu-k\gamma\delta P_1\left(1-u_2\right)\mu-k\alpha P_1P_3\left(1-u_2\right)\mu-k\gamma P_1P_3\left(1-u_2\right)\mu-k\delta P_1P_3\left(1-u_2\right)\mu-k\delta P_2P_4\left(1-u_2\right)\mu-kP_2P_3P_4\left(1-u_2\right)\mu+k\alpha\delta u_1\left(1-u_2\right)\mu+k\gamma\delta u_1\left(1-u_2\right)\mu+k\alpha P_3u_1\left(1-u_2\right)\mu+k\gamma P_3u_1\left(1-u_2\right)\mu+k\delta P_3u_1\left(1-u_2\right)\mu+\alpha\delta P_3\psi_1\mu+\gamma\delta P_3\psi_1\mu+k\alpha\delta\left(1-u_2\right)\psi_1\mu+k\gamma\delta\left(1-u_2\right)\psi_1\mu+k\alpha P_3\left(1-u_2\right)\psi_1\mu+k\gamma P_3\left(1-u_2\right)\psi_1\mu+k\delta P_3\left(1-u_2\right)\psi_1\mu-kP_2P_5\left(1-u_2\right)\psi_1\mu-k\alpha\delta P_1P_3\left(1-u_2\right)-k\gamma\delta P_1P_3\left(1-u_2\right)-k\delta P_2P_3P_4\left(1-u_2\right)+k\alpha\delta P_3u_1\left(1-u_2\right)+k\gamma\delta P_3u_1\left(1-u_2\right)+k\alpha\delta P_3\left(1-u_2\right)\psi_1+k\gamma\delta P_3\left(1-u_2\right)\psi_1-k\delta P_2P_5\left(1-u_2\right)\psi_1$

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