ASIAN JOURNAL OF MATHEMATICS AND APPLICATIONS
Volume 2020, Article ID ama0563, 24 pages
ISSN 2307-7743
http://scienceasia.asia

SENSITIVITY ANALYSIS FOR AVIAN INFLUENZA (BIRD FLU) EPIDEMIC MODEL WITH EXPOSED CLASS

C. E. AKPAN*, M. O. IBRAHIM

Department of Mathematics, University of Ilorin, Ilorin, Nigeria

*Correspondence: rexcitadel2016@yahoo.com

Abstract. This paper established that the mathematical modeling of Avian Influenza disease proposed has solution and unique when compare with other models in literature. Three equilibrium points were obtained, which stability of the system is investigated around. From analysis, the model stability depend on; first, $\beta_4 X$ must be less than $(k+n)$, secondly, the characteristic equation has all it coefficients of the λ positive provided

$e\,r + e\,d + r\,d > \beta_2\,S\,\gamma\,\theta$ and $S\gamma\,\theta(\beta_2\,e + \beta_3\,\alpha)$

Also, the model is stable at Avian Influenza Persistent equilibrium as the system satisfies $J = F(0)G'(0) - F'(0)G(0) > 0$ (Bellman and Cooke's [2]) condition. Hence, the model is locally asymptotically stable at both Disease Free and Persistent Equilibrium.

Finally, the sensitivity analysis also, revealed that the factor responsible for the outbreak of Avian Influenza are majorly, four parameters and they are $\gamma,\ \beta_2,\ \beta_3,\ \theta$. These parameters are capable of preventing spread of Avian Influenza while restricting the movement of infected individuals during treatment session will go a long way manage the spread of Avian Influenza.

1. INTRODUCTION

Bird flu, also called avian influenza, is a viral infection that primarily infect birds, but humans and other animals are also susceptible, (World Health Organization-WHO [19]; MedlinePlus [13]; Bree and Sullivan [3]). It is rare for people to get infected with bird flu viruses, but it can happen. Two types, H5N1 and H7N9, have infected some people during outbreaks in Asia, Africa, the Pacific, the Middle East, and parts of Europe. There have also been some rare cases of other types of bird flu affecting people in the United States. Most of

Keywords: Sensitivity Analysis, Avian Influenza, Epidemic Model.

the people who get bird flu have had close contact with infected birds or with surfaces that have been contaminated by the birds' saliva, mucous, or droppings. It is also possible to get it by breathing in droplets or dust that contain the virus. Rarely, the virus has spread from one person to another. It may also be possible to catch bird flu by eating poultry birds or eggs that are not well cooked, MedlinePlus [13].

Bird flu illness in people can range from mild to severe. Often, the symptoms are similar to the seasonal flu, such as

Fever Cough Sore throat Runny or stuffy nose Muscle or body aches Fatigue Headaches Eye redness (or conjunctivitis) Difficulty breathing

In some cases, bird flu can cause serious complications and death. As with seasonal flu, some people are at higher risk for serious illness. They include pregnant women, people with weakened immune systems, and adults 65 and older.

Treatment with antiviral medicines may make the illness less severe. They may also help prevent the flu in people who were exposed to it. There is currently no vaccine available to the public. The government does have a supply of a vaccine for one type of H5N1 bird flu virus and could distribute it if there was an outbreak that spread easily from person to person.

2. LITERATURE REVIEW

Mishra and Sinha [14] formulated mathematical model of Avian Influenza for both human and bird population. they computed basic reproduction number R_0^H and R_0^B for both human and bird population respectively and locally and globally asymptotically stability were obtained for disease-free equilibrium and unique endemic equilibrium point was globally asymptotically state in bird population when $R_0^B > 1$. In an extensive numerical simulations and sensitivity analysis for various parameters of the model were also carried out. the con concluded that critical analysis for the effect of Vaccination and Quarantined class with Recovered class.

Putri, Watanabe and Nova [16] studied bird flu infection within a poultry farm with effect of spatial diffusion. Effect of threshold number that gives measure of infectious intensity was investigated, which depends on three factors; removal of infected bird, vaccination, and capacity of the farm. their analysis showed that transmission of bird flu within a poultry is expressible in terms of travelling wave solutions. It also shows a spatial significance of

diffusivity. These mean, infection can be fully controlled with appropriate vaccination and proper removal of infected individuals.

Liu, Pang, Ruan and Zhang [10] discussed on global dynamics of Avian Infuenza epidemic Models with psychological effect. The main consideration in the formulation was the psychological effect to Avian influenza in human population in which the Avian population exhibits saturated effect. The results demonstrated that the saturation effect within avian population and the psychological effect in human population cannot change the stability of equilibrium but can affect the number of infected human if the disease is prevalent. Also, the numerical simulation support the theoretical results and sensitivity analyses of the basic reproduction number gave effective control measures for avian influenze.

Zhang, Li, Jin, and Zhu [21] presented an avian influenza A (H7N9) transmission model with vaccination and seasonality among human, birds, and poultry. the basic reproduction number for the prevalence of avian influenza is obtained. The global stability of the disease-free equilibrium and the existence of positive periodic solution were proven by the comparison theorem and the asymptotic autonomous system theorem. The study was finalized with numerical simulations to demonstrate the theoretical results. The simulation results revealed that the risk of H7N9 infection is higher in colder environment,and vaccinating poultry can significantly reduce human infection.

Lee, Ko, and Jung [6] developed a spatial-temporal compartmental model that incorporates the culling rate as a function of the reported farms and farm density in each town of the Republic of Korean. The epidemiological and geographical data of two species; chickens and ducks from the farms in the sixteen towns in Eumseong-gun and Jincheon-gun were used to find the best-fitted parameters of the metapopulation model. The local reproductive number R_0 using the next generation method is calculated as an indicator of virus transmission in a given area. Simulation results indicate that R_0 has strong influence not only by epidemiological factors but also by geographical and demographical factors (density) and connectivity (or distance) farms. Based on the result obtained from simulation, they suggested the best culling radius of Preemptive (PE) culling should be adjusted by considering the local reproductive number in a targeted area.

3. MODEL ANALYSES

In view of the surveyed literature (Mishra and Sinha [14]; Putri *et al.*, [16]; Liu *et al.*, [7]; Zhan *et al.*, [21]; Lee *et al.*, [6]), it became imperative to investigate some properties of the

proposed models, specifically, equilibria states, threshold number for infection transmission and sensitivity analysis to know the most sensitive parameter possibly to cause bird flu epidemics. Thus, subsequent subsections were devoted for the studies. The model is formulated based on the model proposed by Jabbari [7] and Liu [11] . The human population is classified into five subclasses; Susceptible individuals (S), Exposed individuals (E) Infected individual with avian strain (I_a), Infected individuals with mutant strain I_m and Recovered individuals (R) while the avian or bird population is classified into two subclasses; susceptible and infective denoted by X and Y respectively.

The work of Jabbari [7] was extended by adding the exposed class and incorporating the progression rate of human from exposed to infections which is the parameter θ. Also, splitting infected human I_h used in Liu [11] into infected individuals with avian strain (I_a) and infected individuals with mutant strain (I_m).

3.1. **Basic Assumption of Model.**

(1) Individuals are only recruited into susceptible sub population.
(2) The number of susceptibles for the bird population is increased by new recruitment but reduced through natural death and infection.
(3) The avian influenza virus is not contagious from an infective human to a subceptible human. It is only contagious from an infective avian to a susceptible human.
(4) An infected avian keeps in the state of disease cannot recover, but an infected human can recover and the recovered human has permanent immunity.

3.2. **Model Equation.**

$$
\begin{aligned}
\frac{dS}{dt} &= b - [\beta_1 Y + \beta_2 I_a + \beta_3 I_m]S - \mu S \\
\frac{dE}{dt} &= [\beta_1 Y + \beta_2 I_a + \beta_3 I_m]S - (\theta + \mu)E \\
\frac{dI_a}{dt} &= \gamma\theta E - (\alpha + \mu + d_a + \varphi_1)I_a \\
\frac{dI_m}{dt} &= (1-\gamma)\theta E + \alpha I_a - (\varphi_2 + \mu + d_m)I_m \\
\frac{dR}{dt} &= \varphi_1 I_a + \varphi_2 I_m - \mu R \\
\frac{dX}{dt} &= c - \beta_4 XY - kX \\
\frac{dY}{dt} &= \beta_4 XY - (k+n)Y
\end{aligned} \tag{1}
$$

TABLE 1. PARAMETER DESCRIPTION

PARAMETER	DESCRIPTION	VALUE	REFERENCE
b	Recruitment rate for human	30	Jabbari *et al* [7]
c	Bird inflow	1000	Assumed
β_1	Rate at which bird to human avian influenza is contacted	0.2	Jabbari *et al* [7]
β_2	Rate at which human-human avian influenza is contracted	0.4	Jabbari *et al* [7]
β_3	Rate at which human-human mutant influenza is contracted	0.0015	Jabbari*et al* [7]
β_4	Rate at which bird contract avian influenza	0.001	Jabbari *et al* [7]
μ	Natural death rate for human	3.91×10^{-5}	Liu *et al* [11]
α	Mutation rate	0.01	Gumel [5]
d_a	Deduced death rate due to avian strain in human	1	Iwami et al. [6]
d_m	Induced death rate due to mutant strain in human	0.06	Jabbari et al. [8]
k	Natural death rate for birds	3.4246×10^{-4}	Liu etal. [11]
n	Induced death rate due to avian strain in birds	5	Iwami et al. [6]
φ_1	Recovery rate of human with avian strain	0.2669	Lucche et al. [12]
φ_2	Recovery rate of human with mutant strain	0.05	Gumel [5]
θ	Disease progression rate of human from exposed to infectious	0.8	Assumed
γ	Proportion of human that are infected with avian strain	0.3	Assumed

3.3. **Existence and Uniqueness of Model. Theorem 3.3.1**

(Derrick and Grossman, cited in Abdulrazak et al., [1]) Let D' denote the region $|t - t_0| \leq a, ||x - x_0|| \leq b, : x = (x_{10}, x_{20}, ..., x_{n0})$ and suppose that $f(t, x)$ satisfies the Lipschitz condition $||f(t, x_1) - f(t, x_2)|| \leq k||x_1 - x_2||$ whenever the pairs (t, x_1) and (t, x_2) belong to R', where k is a positive constant, then, there is a constant $\delta > 0$ such that a unique continuous vector solution $x(t)$ of the system in the interval $t - t_0 \leq \delta$.

It is important to note that the condition is satisfied by requirement that $\dfrac{\partial f_i}{\partial x_i}, i = 1, 2, ...$ be continuous and bounded in D'.

We are interested in the region

$$0 \leq \alpha \leq \mathbb{R}$$

A bounded solution is sought "D'" in the region whose partial derivatives satisfy $\delta \leq \alpha \leq 0$, where α and δ are positive constants.

Proof:

Let (1) be renamed as $f_1 - f_7$

$$(f_1) \qquad \frac{dS}{dt} = b - [\beta_1 Y + \beta_2 I_a + \beta_3 I_m]S - \mu S$$

$$(f_2) \qquad \frac{dE}{dt} = [\beta_1 Y + \beta_2 I_a + \beta_3 I_m]S - (\theta + \mu)E$$

$$(f_3) \qquad \frac{dI_a}{dt} = \gamma\theta E - (\alpha + \mu + d_a + \varphi_1)I_a$$

$$(f_4) \qquad \frac{dI_m}{dt} = (1 - \gamma)\theta E + \alpha I_a - (\varphi_2 + \mu + d_m)I_m$$

$$(f_5) \qquad \frac{dR}{dt} = \varphi_1 I_a + \varphi_2 I_m - \mu R$$

$$(f_6) \qquad \frac{dX}{dt} = c - \beta_4 XY - kX$$

$$(f_7) \qquad \frac{dY}{dt} = \beta_4 XY - (k + n)Y$$

$$\left|\frac{\partial f_1}{\partial S}\right| = |-(\beta_1 Y + \beta_2 I_a + \beta_3 I_m + \mu)| < \infty; \left|\frac{\partial f_1}{\partial E}\right| = 0 < \infty; \left|\frac{\partial f_1}{\partial I_a}\right| = |-\beta_2 S| < \infty;$$
$$\left|\frac{\partial f_1}{\partial I_m}\right| = |-\beta_3 S| < \infty; \left|\frac{\partial f_1}{\partial R}\right| = 0 < \infty; \left|\frac{\partial f_1}{\partial X}\right| = 0 < \infty; \left|\frac{\partial f_1}{\partial Y}\right| = |-\beta_1 S| < \infty;$$

$$|\frac{\partial f_2}{\partial S}| = |\beta_1 Y + \beta_2 I_a + \beta_3 I_m| < \infty; |\frac{\partial f_2}{\partial E}| = |-(\theta + \mu)| < \infty; |\frac{\partial f_2}{\partial I_a}| = |\beta_2 S| < \infty;$$
$$|\frac{\partial f_2}{\partial I_m}| = |\beta_3 S| < \infty; |\frac{\partial f_2}{\partial R}| = 0 < \infty |\frac{\partial f_2}{\partial X}| = 0 < \infty; |\frac{\partial f_2}{\partial Y}| = |\beta_1 S| < \infty;$$

(2) $\vdots$

$$|\frac{\partial f_6}{\partial S}| = 0 < \infty; |\frac{\partial f_6}{\partial E}| = 0 < \infty; |\frac{\partial f_6}{\partial I_a}| = 0 < \infty; |\frac{\partial f_6}{\partial I_m}| = 0 < \infty |\frac{\partial f_6}{\partial R}| = 0 < \infty;$$
$$|\frac{\partial f_6}{\partial X}| = |-k| < \infty; |\frac{\partial f_6}{\partial Y}| = |-\beta_4 X| < \infty;$$

$$|\frac{\partial f_7}{\partial S}| = 0 < \infty; |\frac{\partial f_7}{\partial E}| = 0 < \infty; |\frac{\partial f_7}{\partial I_a}| = 0 < \infty; |\frac{\partial f_7}{\partial I_m}| = 0 < \infty; |\frac{\partial f_7}{\partial R}| = 0 < \infty;$$
$$|\frac{\partial f_7}{\partial X}| = |\beta_4 Y| < \infty; |\frac{\partial f_7}{\partial Y}| = |\beta_4 X - (k + n)| < \infty;$$

3.4. **Region of Feasibility of Model Solution. Theorem 3.4.1 (For Host)**
The feasible region L defined by
$L = \left\{(S(t), E(t), I_a(t), I_m(t), R(t) \in \mathbb{R}^5_+ : 0 \leq N \leq \frac{b}{\mu}\right\}$ is positively invariant and attracting with respect to the ODE for all $t > 0$ (Gilberto *et al.*, [4]).

Proof:
To obtain the region in which the solution is bounded, the total population will be ;

$$\begin{aligned}\frac{dN}{dt} &= \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI_a}{dt} + \frac{dI_m}{dt} + \frac{dR}{dt} \\ &= b - \beta_1 Y S - \beta_2 I_a S - \beta_3 I_m S - \mu S + \beta_1 Y S + \beta_2 I_a S + \beta_3 I_m S - \theta E - \mu E + \\ &\quad \gamma\theta E - \alpha I_a - \mu I_a - d_a I_a - \varphi_1 I_a + \theta E - \gamma\theta E + \alpha I_a - \varphi_2 I_m - \mu I_m - d_m I_m + \\ &\quad \varphi_1 I_a + \varphi_2 I_m - \mu R \end{aligned}$$

(3) $$= b - \mu(S + E + I_a + I_m + R) - d_a I_a - d_m I_m$$

In the absence of induced death in the population (3) reduces to

$$\begin{aligned} \frac{dN}{dt} &\leq b - \mu(S + E + I_a + I_m + R) \\ &\leq b - \mu N \\ &\text{Hence} \\ 0 < N &\leq \frac{b}{\mu} \in \mathbb{R}^5_+ \end{aligned} \tag{4}$$

Theorem 3.4.2 (For Vector)

The feasible region G defined by
$G = \left\{(X(t), Y(t) \in \mathbb{R}^2_+ : 0 \leq N \leq \frac{b}{\mu}\right\}$ is positively invariant and attracting with respect to the ODE for all $t > 0$ (Gilberto *et al.*, [4]).

Proof:

To obtain the region in which the solution is bounded, the total population will be ;

$$\begin{aligned} \frac{dN}{dt} &= \frac{dX}{dt} + \frac{dY}{dt} \\ &= c - \beta_4 XY - kX + \beta_4 XY - kY - nY \end{aligned}$$

In the absence of induced death in the vector population (3.4) reduces to

$$\begin{aligned} \frac{dN}{dt} &\leq c - k(X + Y) \\ &\leq c - kN \\ &\text{Hence} \\ 0 < N &\leq \frac{c}{k} \in \mathbb{R}^2_+ \end{aligned} \tag{5}$$

3.5. **Positivity of Solution. Theorem 3.5.1**

Given that the initial condition of the differential equations are $S_0 > 0$, $E_0 > 0$, $I_{a0} > 0$, $I_{m0} > 0$, $R_0 > 0$, $X_0 > 0$,$Y_0 > 0$ the solutions of $S(t)$, $E(t)$, $I_a(t)$, $I_m(t)$, $R(t)$, $X(t)$,$Y(t)$ are non-negative for all t>0 (Gilberto *et.al.*, [4]).

Proof:

Assume that

$$t = sup\left\{t > 0 : S > 0,\, E \geq 0,\, I_a \geq 0,\, I_m \geq 0,\, R \geq 0,\, X \geq 0,\, Y \geq 0\right\} \in [0, t]$$

Hence $t > 0$;

From the first equation in (1),

$$\frac{dS}{dt} \geq -(\beta_1 Y + \beta_2 I_a + \beta_3 I_m)S \Rightarrow \frac{dS}{S} \geq -(\beta_1 Y + \beta_2 I_a + \beta_3 I_m)dt \tag{6}$$

Integrating both sides of (6) gives

$$\int \frac{dS}{S} \geq -\int (\beta_1 Y + \beta_2 I_a + \beta_3 I_m)dt \Rightarrow \ln S(t) \geq -(\beta_1 Y + \beta_2 I_a + \beta_3 I_m)t + K \tag{7}$$

when $t = 0$ in (7) implies

$$\ln S(0) = K \tag{8}$$

Substituting back (8) into (7) yields

$$\begin{aligned} \ln S(t) &\geq -(\beta_1 Y + \beta_2 I_a + \beta_3 I_m)t + \ln S(0) \\ &\Rightarrow \ln S(t) - \ln S(0) \geq -(\beta_1 Y + \beta_2 I_a + \beta_3 I_m)t \end{aligned}$$

Hence;

$$\ln \frac{S(t)}{S(0)} \geq -(\beta_1 Y + \beta_2 I_a + \beta_3 I_m)t \tag{9}$$

Taking the exponent of both sides of (9) yields

$$\begin{aligned} \frac{S(t)}{S(0)} &\geq e^{-(\beta_1 Y + \beta_2 I_a + \beta_3 I_m)t} \\ &\Rightarrow S(t) \geq S(0)e^{-(\beta_1 Y + \beta_2 I_a + \beta_3 I_m)t} \end{aligned}$$

But $S(0) = S_0$, hence

$$S(t) \geq S_0 e^{-(\beta_1 Y + \beta_2 I_a + \beta_3 I_m)t} > 0 \tag{10}$$

Also from the second equation in (1),

$$\frac{dE}{dt} \geq -(\theta + \mu)E \Rightarrow \frac{dE}{E} \geq -(\theta + \mu)dt \tag{11}$$

Integrating both sides of (11) gives

$$\int \frac{dE}{E} \geq -\int (\theta+\mu)dt \Rightarrow \ln E(t) \geq -(\theta+\mu)t + c \tag{12}$$

when $t = 0$ in (12) implies

$$\ln E(0) = c \tag{13}$$

Substituting back (13) into (12) yields

$$\begin{aligned} \ln E(t) &\geq -(\theta+\mu)t + \ln E(0) \\ &\Rightarrow \ln E(t) - \ln E(0) \geq -(\theta+\mu)t \end{aligned}$$

Hence;

$$\ln \frac{E(t)}{E(0)} \geq -(\theta+\mu)t \tag{14}$$

Taking the exponent of both sides of (14) yields

$$\begin{aligned} \frac{E(t)}{E(0)} &\geq e^{-(\theta+\mu)t} \\ &\Rightarrow E(t) \geq E(0)e^{-(\theta+\mu)t} \end{aligned}$$

But $E(0) = E_0$,hence

$$E(t) \geq E_0 e^{-(\theta+\mu)t} > 0 \tag{15}$$

Considering the sixth equation in (1),

$$\frac{dX}{dt} \geq -(\beta_4 Y + k)X \Rightarrow \frac{dX}{X} \geq -(\beta_4 Y + k)dt \tag{16}$$

Integrating both sides of (16) gives

$$\int \frac{dX}{X} \geq -\int (\beta_4 Y + k)dt \Rightarrow \ln X(t) \geq -(\beta_4 Y + k)t + d \tag{17}$$

when $t = 0$ in (17) implies

$$\ln X(0) = d \tag{18}$$

Substituting back (18) into (17) yields

$$\ln X(t) \geq -(\beta_4 Y + k)t + \ln X(0)$$
$$\Rightarrow \ln E(t) - \ln X(0) \geq -(\beta_4 Y + k)t$$

Hence;

$$\ln \frac{X(t)}{X(0)} \geq -(\beta_4 Y + k)t \tag{19}$$

Taking the exponent of both sides of (19) yields

$$\frac{X(t)}{X(0)} \geq e^{-(\beta_4 Y + k)t}$$
$$\Rightarrow X(t) \geq X(0)e^{-(\beta_4 Y + k)t}$$

But $X(0) = X_0$,hence

$$X(t) \geq X_0 e^{-(\beta_4 Y + k)t} > 0 \tag{20}$$

Similarly, it can be shown that $I_a(t) \geq I_{a0}e^{-(\alpha+\mu+d_a+\varphi_1)t} > 0$, $I_m(t) \geq I_{m0}e^{-(\varphi_2+\mu+d_m)t} > 0$, $R(t) \geq R_0 e^{-(\mu)t} > 0$, $Y(t) \geq Y_0 e^{-(\beta_4 X - k - n)t} > 0$ for all $t > 0$
Hence, this completes the proof.

3.6. **Equilibria States.**

3.6.1. *CASE I.* In the absence of Bird flu and no infection i.e $Y = 0$, $I_a = 0$ and $I_m = 0$. The aim here is to get

$$\epsilon^* = (S^*, E^*, I_a^*, I_m^*, R^*, X^*, Y^*) \tag{21}$$

To achieve (21), each compartments of equation (1) will be equated to zero in the absence of disease, that is;

$$
\begin{aligned}
&b - [\beta_1 Y(t) + \beta_2 I_a(t) + \beta_3 I_m(t)]S(t) - \mu S(t) = 0 \\
&[\beta_1 Y(t) + \beta_2 I_a(t) + \beta_3 I_m(t)]S(t) - (\mu + \theta)E(t) = 0 \\
&\gamma\theta E(t) - (\alpha + \mu + d_a + \varphi_1)I_a(t) = 0 \\
&(1-\gamma)\theta E(t) + \alpha I_a(t) - (\mu + d_m + \varphi_2)I_m(t) = 0 \\
&\varphi_1 I_a(t) + \varphi_2 I_m(t) - \mu R(t) = 0 \\
&c - \beta_4 X(t)Y(t) - kX(t) = 0 \\
&\beta_4 X(t)Y(t) - (k+n)Y(t) = 0
\end{aligned} \tag{22}
$$

Now each variables in (22) will be solved as follows;

From $6th$ equation

$$X = \frac{c}{k} \tag{23}$$

and Human population dynamics is
$E = 0$ from 2^{nd} equation and $R = 0$ from 5^{th} equation
Hence, from $1st$ equation we have;

$$b - \mu S = 0 \tag{24}$$

This implies

$$S = \frac{b}{\mu} \tag{25}$$

Hence

$$\left\{(S, E, I_a, I_m, R) = \left(\frac{b}{\mu}, 0, 0, 0, 0\right)\right\} \tag{26}$$

and

$$\left\{(X, Y) = \left(\frac{c}{k}, 0\right)\right\} \tag{27}$$

3.6.2. *CASE II.* Consider the situation whereby $Y \neq 0, I_m = 0, I_a = 0$

we use

$$X = \frac{k+n}{\beta_4} \tag{28}$$

and from 6^{th} equation

$$X = \frac{c}{k+\beta_4 Y} \tag{29}$$

Equating (28) and (29) we obtain

$$Y = \frac{\beta_4 C - k(k+n)}{\beta_4(k+n)} \tag{30}$$

Since $I_a = 0$ and $I_m = 0$, 1^{st} equation reduces to

$$B - (\mu + \beta_1 Y)S = 0 \tag{31}$$

This implies

$$S = \frac{B}{\mu + \beta_1 Y} \tag{32}$$

Substituting the values of Y into (32) yields

$$S^{**} = \frac{B\beta_4(k+n)}{(k+n)(\mu\beta_4 - \beta_1 k) + \beta_1\beta_4 C} \tag{33}$$

and from 2^{nd} equation

$$E = \frac{\beta_1 Y S}{\mu + \theta} \tag{34}$$

After substituting Y, (34) yields

$$E^{**} = \left(\frac{B\beta_1(C\beta_4 - k(k+n))}{(k+n)(\mu\beta_4 - \beta_1 k) + \beta_1\beta_4 C}\right) \times \frac{1}{\mu + \theta} \tag{35}$$

Thus,

$$E_0^{**} = \{S^{**}, E^{**}, 0, 0, 0\} \tag{36}$$

$$E_0^{**} = \left\{\frac{k+n}{\beta_4}, \frac{C\beta_4 - k(k+n)}{\beta_4(k+n)}\right\} \tag{37}$$

Remark:- It is obtainable to have bird flu in the vector population while human being only get exposed and this is inline with WHO, [20] report that it is hard to transmit the disease to human but when successful, mortality chance is 60%. Thus, we check for this possibility.

3.6.3. *CASE III.* **Persistence Equilibrium**

Suppose $Y \neq 0, I_a \neq 0$ and $I_m \neq 0$

Adding 1^{st} and 2^{nd} equations yield

$$B - \mu S - (\mu + \theta)E = 0 \tag{38}$$

Substituting (33) into (38) to get E yields

$$E = \frac{-B\beta_1 k(k+n) + \beta_1\beta_4 CB}{(\mu+\theta)((k+n)(\mu\beta_4 + k\beta_1) + \beta_1\beta_4 C)} \tag{39}$$

Expose leading to outbreak of Bird flu epidemic

From 3^{rd} equation,

$$I_a = \frac{\gamma\theta}{\alpha + \varphi_1 + \mu + d_a} \times \frac{-B\beta_1 k(k+n) + \beta_1\beta_4 CB}{(\mu+\theta)((k+n)(\mu\beta_4 + k\beta_1) + \beta_1\beta_4 C)} \tag{40}$$

$$\therefore\ I_a = \frac{\gamma\theta(\beta_1\beta_4 CB - B\beta_1 k(k+n))}{(\mu+\theta)(\alpha+\varphi_1+\mu+d_a)((k+n)(\mu\beta_4 + k\beta_1) + \beta_1\beta_4 C)} \tag{41}$$

Similarly from 4^{th} equation

$$\frac{(1-\gamma)\theta g_1}{k_2} - \frac{\alpha\gamma\theta(\beta_1\beta_4 C - \beta_1 k(k+n))B}{k_1} - \frac{(\mu + d_m + \varphi_2)I_m}{1} = 0 \tag{42}$$

$$\implies I_m = \frac{(1-\gamma)\theta g_1(\alpha+\varphi_1+\mu+d_a) - \alpha\gamma\theta(\beta_1\beta_4 C - \beta_1 k(k+n))B}{k_1(\mu + d_m + \varphi_2)} \tag{43}$$

Obviously, from 5^{th} equation,

$$R = \frac{1}{\mu}(\varphi_2 I_m^* + \varphi_1 I_a^*) \tag{44}$$

where

$$\begin{aligned} k_1 &= (\mu+\theta)(\alpha+\varphi_1+\mu+d_a)((k+n)(\mu\beta_4 + k\beta_1) + \beta_1\beta_4 C) \\ k_2 &= (\mu+\theta)((k+n)(\mu\beta_4 + k\beta_1) + \beta_1\beta_4 C) \\ g_1 &= (-B\beta_1 k(k+n) + \beta_1\beta_4 C) \end{aligned} \tag{45}$$

3.7. **Control parameter (R_0) for Model.** The control parameter that governs the spread of disease is here obtained. The method of next generation matrix (Somma *et al.,* [17]) is used.

The model equation (1) is rewritten starting with the new diseases in the system of equation.

$$
\begin{aligned}
\frac{dE}{dt} &= [\beta_1 Y(t) + \beta_2 I_a(t) + \beta_3 I_m(t)]S(t) - (\mu+\theta)E(t) \\
\frac{dI_a}{dt} &= \gamma\theta E(t) - (\alpha + \mu + d_a + \varphi_1)I_\alpha(t) \\
\frac{dI_m}{dt} &= (1-\gamma)\theta E(t) + \alpha I_a(t) - (\mu + d_m + \varphi_2)I_m(t) \\
\frac{dY}{dt} &= \beta_4 X(t)Y(t) - (k+n)Y(t)
\end{aligned} \tag{46}
$$

From (46), f and v are deduced, where f refers to the new infections while v indicates other interactions in the infected compartments. This yields

$$
f = \begin{pmatrix} [\beta_1 Y + \beta_2 I_a + \beta_3 I_m]S \\ \beta_4 XY \\ 0 \\ 0 \end{pmatrix} \text{ and } v = \begin{pmatrix} (\mu+\theta)E \\ -\gamma\theta E + (\alpha + \mu + d_a + \varphi_1)I_a \\ -(1-\gamma)\theta E - \alpha I_a + (\mu + d_m + \varphi_2)I_m \\ (k+n)Y \end{pmatrix}
$$

Now differentiating f and v partially to yield F and V respectively, that is;

$$
F = \begin{pmatrix} \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I_a} & \frac{\partial f_1}{\partial I_m} & \frac{\partial f_1}{\partial Y} \\ \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I_a} & \frac{\partial f_2}{\partial I_m} & \frac{\partial f_2}{\partial Y} \\ \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I_a} & \frac{\partial f_3}{\partial I_m} & \frac{\partial f_3}{\partial Y} \\ \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I_a} & \frac{\partial f_4}{\partial I_m} & \frac{\partial f_4}{\partial Y} \end{pmatrix} \text{ and } V = \begin{pmatrix} \frac{\partial v_1}{\partial E} & \frac{\partial v_1}{\partial I_a} & \frac{\partial v_1}{\partial I_m} & \frac{\partial v_1}{\partial Y} \\ \frac{\partial v_2}{\partial E} & \frac{\partial v_2}{\partial I_a} & \frac{\partial v_2}{\partial I_m} & \frac{\partial f_2}{\partial Y} \\ \frac{\partial v_3}{\partial E} & \frac{\partial v_3}{\partial I_a} & \frac{\partial v_3}{\partial I_m} & \frac{\partial v_3}{\partial Y} \\ \frac{\partial v_4}{\partial E} & \frac{\partial v_4}{\partial I_a} & \frac{\partial v_4}{\partial I_m} & \frac{\partial v_4}{\partial Y} \end{pmatrix}
$$

$$
\Rightarrow F = \begin{pmatrix} 0 & \beta_2 S & \beta_3 S & \beta_1 S \\ 0 & 0 & 0 & \beta_4 S \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}
$$

$$
\text{and } V = \begin{pmatrix} \mu+\theta & 0 & 0 & 0 \\ -\gamma\,\theta & \mu + d_a + \alpha + \varphi_1 & 0 & 0 \\ -(1-\gamma)\,\theta & -\alpha & \varphi_2 + \mu + d_m & 0 \\ 0 & 0 & 0 & k+n \end{pmatrix}
$$

Then

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu+\theta} & 0 & 0 & 0 \\ \frac{\gamma\,\theta}{(\mu+\theta)a} & \frac{1}{a} & 0 & 0 \\ -\frac{\theta\,b}{a(\mu+\theta)d} & \frac{\alpha}{a\,d} & \frac{1}{d} & 0 \\ 0 & 0 & 0 & \frac{1}{k+n} \end{pmatrix} \tag{47}$$

where

$a = \mu + d_a + \alpha + \varphi_1$

$b = \gamma\,\mu + \gamma\,d_a + \gamma\,\varphi_1 - \alpha - \mu - d_a - \varphi_1$

and

$d = \varphi_2 + \mu + d_m$

So the eigen value evaluated by $|FV^{-1} - \lambda I| = 0$ yields;

$$\lambda_{1,2,3,4} = 0 \text{ and } \lambda_5 = \frac{S\,\theta\,g}{j}$$

where

$g = \gamma\,\mu\,\beta_2 - \gamma\,\mu\,\beta_3 + \gamma\,\beta_2 d_m + \gamma\,\beta_2\varphi_2 - \gamma\,\beta_3 d_a - \gamma\,\beta_3\varphi_1 + \alpha\,\beta_3 + \mu\,\beta_3 + \beta_3 d_a + \beta_3\varphi_1$

and

$j = \alpha\,\mu^2 + \alpha\,\mu\,\theta + \alpha\,\mu\,d_m + \alpha\,\mu\,\varphi_2 + \alpha\,\theta\,d_m + \alpha\,\theta\,\varphi_2 + \mu^3 + \mu^2\theta + \mu^2 d_a + \mu^2 d_m + \mu^2\varphi_1 + \mu^2\varphi_2 + \mu\,\theta\,d_a + \mu\,\theta\,d_m + \mu\,\theta\,\varphi_1 + \mu\,\theta\,\varphi_2 + \mu\,d_a d_m + \mu\,d_a\varphi_2 + \mu\,d_m\varphi_1 + \mu\,\varphi_1\varphi_2 + \theta\,d_a d_m + \theta\,d_a\varphi_2 + \theta\,d_m\varphi_1 + \theta\,\varphi_1\varphi_2$

where $R_0 = \rho$(eigenvalues)

Therefore,

$$R_0 = \frac{S\,\theta\,g}{j} \tag{48}$$

The population will experience disease free if and only if $R_0 < 1$

This implies

$$S\,\theta\,g < j$$

which implies that the force of infection must be less than the removal rate.

3.8. **Stability Analysis for Model.** In order to establish chances of controlling the disease, the concept of Jacobian matrix will be used on the system. The Jacobian matrix of the model equation is obtained as;

$$J=\begin{pmatrix} \frac{\partial F_1}{\partial S} & \frac{\partial F_1}{\partial E} & \frac{\partial F_1}{\partial I_a} & \frac{\partial F_1}{\partial I_m} & \frac{\partial F_1}{\partial R} & \frac{\partial F_1}{\partial X} & \frac{\partial F_1}{\partial Y} \\ \frac{\partial F_2}{\partial S} & \frac{\partial F_2}{\partial E} & \frac{\partial F_2}{\partial I_a} & \frac{\partial F_2}{\partial I_m} & \frac{\partial F_2}{\partial R} & \frac{\partial F_2}{\partial X} & \frac{\partial F_2}{\partial Y} \\ \frac{\partial F_3}{\partial S} & \frac{\partial F_3}{\partial E} & \frac{\partial F_3}{\partial I_a} & \frac{\partial F_3}{\partial I_m} & \frac{\partial F_3}{\partial R} & \frac{\partial F_3}{\partial X} & \frac{\partial F_3}{\partial Y} \\ \frac{\partial F_4}{\partial S} & \frac{\partial F_4}{\partial E} & \frac{\partial F_4}{\partial I_a} & \frac{\partial F_4}{\partial I_m} & \frac{\partial F_4}{\partial R} & \frac{\partial F_4}{\partial X} & \frac{\partial F_4}{\partial Y} \\ \frac{\partial F_5}{\partial S} & \frac{\partial F_5}{\partial E} & \frac{\partial F_5}{\partial I_a} & \frac{\partial F_5}{\partial I_m} & \frac{\partial F_5}{\partial R} & \frac{\partial F_5}{\partial X} & \frac{\partial F_5}{\partial Y} \\ \frac{\partial F_6}{\partial S} & \frac{\partial F_6}{\partial E} & \frac{\partial F_6}{\partial I_a} & \frac{\partial F_6}{\partial I_m} & \frac{\partial F_6}{\partial R} & \frac{\partial F_6}{\partial X} & \frac{\partial F_6}{\partial Y} \\ \frac{\partial F_7}{\partial S} & \frac{\partial F_7}{\partial E} & \frac{\partial F_7}{\partial I_a} & \frac{\partial F_7}{\partial I_m} & \frac{\partial F_7}{\partial R} & \frac{\partial F_7}{\partial X} & \frac{\partial F_7}{\partial Y} \end{pmatrix}$$

This yields

$$J=\begin{pmatrix} -(m+\mu) & 0 & -\beta_2 S & -\beta_3 S & 0 & 0 & -\beta_1 S \\ m & -(\theta+\mu) & \beta_2 S & \beta_3 S & 0 & 0 & \beta_1 S \\ 0 & \gamma\theta & -d & 0 & 0 & 0 & 0 \\ 0 & (1-\gamma)\theta & \alpha & -e & 0 & 0 & 0 \\ 0 & 0 & \varphi_1 & \varphi_2 & -\mu & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\beta_4 Y - k & -\beta_4 X \\ 0 & 0 & 0 & 0 & 0 & \beta_4 Y & \beta_4 X - (k+n) \end{pmatrix} \tag{49}$$

where

$$m = \beta_1 Y + \beta_2 I_a + \beta_3 I_m$$

$$d = \alpha + \varphi_1 + \mu + d_a, \qquad e = \varphi_2 + \mu + d_m$$

3.9. **Stability of Disease Free Equilibrium for Model.** At disease free, the Jacobian matrix (49) is considered in the absence of disease and hence reduce to

$$J=\begin{pmatrix} -\mu & 0 & -\beta_2 S & -\beta_3 S & 0 & 0 & -\beta_1 S \\ m & -(\theta+\mu) & \beta_2 S & \beta_3 S & 0 & 0 & \beta_1 S \\ 0 & \gamma\theta & -d & 0 & 0 & 0 & 0 \\ 0 & (1-\gamma)\theta & \alpha & -e & 0 & 0 & 0 \\ 0 & 0 & \varphi_1 & \varphi_2 & -\mu & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -k & -\beta_4 X \\ 0 & 0 & 0 & 0 & 0 & 0 & \beta_4 X - (k+n) \end{pmatrix} \tag{50}$$

Equation (50) is further reduced and the eigenvalue $(J - \lambda I = 0)$ evaluation is carried out to obtain

$$J = \begin{vmatrix} -(r+\lambda) & \beta_2 S & \beta_3 S & \beta_1 S \\ \gamma\theta & -(d+\lambda) & 0 & 0 \\ (1-\gamma)\theta & \alpha & -(e+\lambda) & 0 \\ 0 & 0 & 0 & \beta_4 X - (k+n) - \lambda \end{vmatrix} = 0 \tag{51}$$

Determining Stability at DFE of Model require evaluation of determinant of equation (51) such that the eigenvalues are negative or / and where necessary, characteristics equations results from the determinant satisfies Routh-Hurwitz Necessary Criteria, which stated that:

(1) all the polynomial coefficients must have the same sign
(2) all the polynomial coefficients must be non-zero

In doing this, let $q = \beta_4 X - (k+n) - \lambda$, and $r = \theta + \mu$, then equation (51) yields

$$(-(r+\lambda))(-(d+\lambda))(-(e+\lambda))\,q - (\beta_2\, S\,\gamma\,\theta(-(e))\,q) + \beta_2\, S\,\gamma\,\theta\,\alpha\,q = 0$$

Factorizing q gives

$$q[-(r+\lambda)(d+\lambda)(e+\lambda) + \beta_2\, S\,\gamma\,\theta\, e + \beta_2\, S\,\gamma\,\theta\,\alpha] = 0$$

This implies
either

$$q = 0 \tag{52}$$

or

$$-(r+\lambda)(d+\lambda)(e+\lambda) + \beta_2\, S\,\gamma\,\theta\, e + \beta_2\, S\,\gamma\,\theta\,\alpha = 0 \tag{53}$$

Solving equation (52) yields

$$\lambda = \beta_4\, X - (k+n) \tag{54}$$

and solving (53) yields

$$-e\,r\,d - e\,r\,\lambda - e\,d\,\lambda - e\,\lambda^2 - r\,d\,\lambda - r\,\lambda^2 - d\,\lambda^2 - \lambda^3 + \beta_2\, S\,\gamma\,\theta\, e + \beta_2\, S\,\gamma\,\theta\,\lambda + \beta_3\, S\,\gamma\,\theta\,\alpha = 0$$

This leads to

$$\lambda^3 + \lambda^2(e+r+d) + \lambda(e\,r + e\,d + r\,d - \beta_2\, S\,\gamma\,\theta) + e\,r\,d - \beta_2\, S\,\gamma\,\theta\, e - \beta_3\, S\,\gamma\,\theta\,\alpha = 0 \tag{55}$$

From the solving, the system stability is concluded depending on; first, the equation (54), $\beta_4 X$ must be less than $(k+n)$, and for the characteristic equation (55), which required examining the conditions of the roots. Thus, all the coefficients of the polynomial were found positive provided

$e\,r + e\,d + r\,d > \beta_2\,S\,\gamma\,\theta$ and $S\gamma\,\theta(\beta_2\,e + \beta_3\,\alpha)$

Hence, the model is locally asymptotically stable at Disease Free Equilibrium. Next is the case of persistent of bird flu disease for the proposed model.

3.10. **Stability Analysis for Endemic Equilibrium for Model.** In order to compute the stability analysis of the endemic equilibrium, equation (49) is considered in it full, but at simplification, it is reduced and the eigenvalue is evaluated as follows

(56)
$$J = \begin{pmatrix} -(m+\sigma+\mu)-\lambda & 0 & -\beta_2\,S & -\beta_3\,S & 0 & -\beta_1\,S \\ m & -(\theta+\mu)-\lambda & \beta_2\,S & \beta_3\,S & 0 & \beta_1\,S \\ 0 & \gamma\,\theta & -n-\lambda & 0 & 0 & 0 \\ 0 & (1-\gamma)\,\theta & \alpha & -p-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & -\beta_4 Y - k - \lambda & -\beta_4 X \\ 0 & 0 & 0 & 0 & \beta_4 Y & \beta_4 X - (k+n) - \lambda \end{pmatrix}$$

(57)
$$\begin{aligned} &= -\,(m+\sigma+\lambda)(-(c+\lambda))(-(d+\lambda))(-(e+\lambda))\,(p) + \\ &\quad (\beta_2\,S\,m\,\gamma\,\theta(-(e+\lambda))\,(p) - (-\beta_3\,S\,m\,\gamma\,\theta\,\alpha(p)) \end{aligned}$$

Factorising (p)

(58)
$$p[(b+\lambda)(c+\lambda)(d+\lambda)(e+\lambda) + \beta_2\,S\,m\,\gamma\,\theta(e+\lambda) + \beta_3\,S\,m\,\gamma\,\theta\,\alpha]$$

This implies

(59)
$$p = 0$$

0r

(60)
$$(b+\lambda)(c+\lambda)(d+\lambda)(e+\lambda) + \beta_2\,S\,m\,\gamma\,\theta(e+\lambda) + \beta_3\,S\,m\,\gamma\,\theta\,\alpha = 0$$

where

$$
\begin{aligned}
m &= \beta_1 Y + \beta_2 I_a + \beta_3 I_m \\
p &= -(\beta_4 Y + k + \lambda)(\beta_4 X - (k+n) - \lambda) + \beta_4^2 XY \\
c &= \mu + \theta \\
b &= m + \mu
\end{aligned} \tag{61}
$$

Now evaluating (59) yields

$$
\begin{aligned}
\lambda_1 = {} & 1/2\,\beta_4 X - 1/2\,\beta_4 Y - k - n/2 \\
& + 1/2\,\sqrt{X^2{\beta_4}^2 - 2\,XY{\beta_4}^2 + Y^2{\beta_4}^2 - 2\,Xn\beta_4 - 2\,Yn\beta_4 + n^2}
\end{aligned} \tag{62}
$$

and

$$
\begin{aligned}
\lambda_2 = {} & 1/2\,\beta_4 X - 1/2\,\beta_4 Y - k - n/2 \\
& - 1/2\,\sqrt{X^2{\beta_4}^2 - 2\,XY{\beta_4}^2 + Y^2{\beta_4}^2 - 2\,Xn\beta_4 - 2\,Yn\beta_4 + n^2}
\end{aligned} \tag{63}
$$

Also expanding (60) yields the characteristic polynomial

$$
\begin{aligned}
&\lambda^4 + (b+c+d+e)\lambda^3 + (bc + bd + be + cd + ce + de)\lambda^2 + (bcd + bce + bde + cde \\
&+ \beta_2\, S\, m\, \gamma\, \theta)\lambda + bcde + \beta_2\, S\, m\, e + \beta_3\, S\, m\, \gamma\, \theta\, \alpha = 0
\end{aligned} \tag{64}
$$

Using the concept of Bellman and Cooke's Theorem [2] on equation (64) for establishment of the system stability.

Theorem 3.10.1

Consider the polynomial (64), which it is locally asymptotically stable around equilibria state if on the complex plane satisfies $J = F(0)G'(0) - F'(0)G(0) > 0$.

proof:

First, expressing the polynomial (64) in a complex plane by re-writing the polynomial containing λ in terms if iy and Separate into real and imaginary as $J(iy) = F(y) + iG(y)$ i.e.

$$
\begin{aligned}
J(iy) = {} & (iy)^4 + (b+c+d+e)(iy)^3 + (bc + bd + be + cd + ce + de)(iy)^2 + (bcd + bce + bde \\
& + cde + \beta_2\, S\, m\, \gamma\, \theta)(iy) + bcde + \beta_2\, S\, m\, e + \beta_3\, S\, m\, \gamma\, \theta\, \alpha = 0
\end{aligned} \tag{65}
$$

$$
\begin{aligned}
J(iy) =& y^4 - (b\,c + b\,d + b\,e + c\,d + c\,e + d\,e)y^2 + b\,c\,d\,e + \beta_2\,S\,m\,e + \beta_3\,S\,m\,\gamma\,\theta\,\alpha \\
&- i\Big((b + c + d + e)y^3 + (b\,c\,d + b\,c\,e + b\,d\,e + c\,d\,e + \beta_2\,S\,m\,\gamma\,\theta)y\Big) = 0
\end{aligned} \tag{66}
$$

Therefore, $H(iy) = F(y) + iG(y)$,

where, $F(y) = y^4 - (b\,c + b\,d + b\,e + c\,d + c\,e + d\,e)y^2 + b\,c\,d\,e + \beta_2\,S\,m\,e + \beta_3\,S\,m\,\gamma\,\theta\,\alpha$ and $G(y) = \Big((b + c + d + e)y^3 + (b\,c\,d + b\,c\,e + b\,d\,e + c\,d\,e + \beta_2\,S\,m\,\gamma\,\theta)y\Big)$

differentiating $F(y)$ and $G(y)$, then, substitute in the condition of the theorem, to have $J = F(0)G'(0) > 0$. Hence, the proof. Thus, the conclusion is that the system is locally asymptotically stable.

4. Sensitivity Analysis for Model

Sensitivity analysis is carried out on each parameters, this is used in checking and identifying parameters that are liable to impacting the reproductive number.
In order to obtain this technique,the concept adopted by (Tilahun, Makinde & Malonza, [18]) is here applied. The formula used in this concept for all basic parameters is defined as

$$
\Delta_x^{R_0} = \left(\frac{\partial R_0}{\partial x}\right) \times \frac{x}{R_0}; \tag{67}
$$

where x represents all the basic parameters.
For instance, sensitivity index of R_0 with respect to γ is $\Delta_\gamma^{R_0} = \left(\dfrac{\partial R_0}{\partial \gamma}\right) \times \dfrac{\gamma}{R_0} = 0.8683565952$, all others are obtained in the same manner as $\Delta_\theta^{R_0}$ $\Delta_{\varphi_1}^{R_0}$, $\Delta_{\varphi_2}^{R_0}$, $\Delta_{\beta_2}^{R_0}$, $\Delta_{\beta_3}^{R_0}$, $\Delta_\mu^{R_0}$, $\Delta_\alpha^{R_0}$, $\Delta_{d_m}^{R_0}$, $\Delta_{d_a}^{R_0}$ and are evaluated at γ =0.3 $\theta = 0.8$, $\varphi_1 = 0.2669$, $\varphi_2 = 0.05$, $\beta_2 = 0.4$, $\beta_3 = 0.0015$, $\mu = 0.0000391$, $\alpha = 0.01$, $\theta = 0.8$, $d_m = 0.06$, $d_a = 1$ The sensitivity indices is represented in table 2.

TABLE 2. Sensitivity indices table

Parameter symbol	Sensitivity indices
γ	0.8683565952
φ_1	-0.1897545958
φ_2	-0.04201218574
β_2	0.9075403382
β_3	0.09245966216
μ	-0.0001095245844
α	-0.006800297646e
θ	0.00004887261139
d_m	-0.05041462289
d_a	-0.7109576457

4.1. **Interpretation of Sensitivity Indices.** In table 2, the parameters with positive indices i.e (γ, β_2, β_3, and θ) are those parameters that have great impact on the expansion of the disease in the community if their values are increasing. This is because the basic reproduction number increases as their value increases, that is, the average number of secondary cases of infection increases in the community.

Also from table 2, all the parameters in which their sensitivity indices are negative i.e (φ_1, φ_2, μ, α, d_m and d_a) have capability of minimizing the disease in the community as their values increase while the others are left constant. As their values increase, the basic reproduction number decreases, which leads to minimizing the endemicity of the disease in the community.

5. CONCLUSION

The sensitivity analysis revealed that factor responsible for the outbreak of Avian Influenza is majorly, four denoted parameters and they are γ, β_2, β_3, θ. These parameters are capable of preventing spread of Avian Influenza while restricting the movement of infected individuals during treatment session will go a long way manage the spread of Avian Influenza.

REFERENCES

[1] Abdulrazak A.J., Ibrahim M.O., & Usman I.O. (2012). A SEIV Vaccination Model with General Non-Linear Incidence Rate and Waning Preventive Vaccine. *IOSR Journal of Mathematics* 4(2),44-51.

[2] Bellman, R and Cooke, K.L. (1963). *Differential Difference Equations*. Academics Press, New York & London .

[3] Bree N. and Sullivan (2017). Bird Flu. Healthline. Retrieved from https://www.healthline.com/health/avian-influenza

[4] Gilberto G., Benito C., & Hristo V. (2018). Mathematical modeling of crime as a social epidemic. *Journal of Interdisciplinary mathematics*, 21(3), 623-643.

[5] Gumel, A.B. (2009). Global dynamics of a two-strain avian influenza model. *Int. J. Comput. Maths*, 86, 85-1008.

[6] Iwami,S. Takenchi, Y., & Liu,X. (2007). Avian human influenza epidemic model, *Mathematical Bioscience*. 207, 1-25

[7] Jabbari, A., Kheiri, H., & Akbarfam, A.J. (2016). Dynamical analysis of the avian human influenza epidemic model using multistage analytical method. *International Journal of Biomathematics, World Scientific Publishing Company* 9(6), 1650090(19 pages) DOI: 10.1142/S179352451650090X

[8] Jabbari A., Kheiri A., Bekir A. (2017). Dynamical analysis of the avian-human influenza epidemic model using the semi analytical method.Open eng; 5:148-156.

[9] Lee J., Ko Y., Jung E. (2019). Effective Control Measures Considering Spatial Heterogeneity to Mitigate the 2016?017 Avian Influenza Epidemic in the Republic of Korea. *PLoSONE* 14(6):e0218202. https://doi.org/10.1371/journal.pone.0218202

[10] Liu, S., Pang, L., Ruan, S., & Zhang, X. (2015). Global Dynamics of Avian Influenza Epidemic Models with Psychological Effect. *Computational and Mathematical Methods in Medicine, Hindawi Publishing Corporation.* http://dx.doi.org/10.1155/2015/13726

[11] Liu, S. Ruan, S., & Zhang, X., (2017). Nonlinear dynamics of avian influenza epidemic models. *Mathematical Booseremes*, 283, 118-135.

[12] Luchetti, J., Roy, M., & Martchera, M. (2009). An avian influenza model and its fit to Human avian influenza cases, In: JM Techuneche, Z. Mukandane (Eds). Advances in Disease Epidemiology. *Nova serance Publishers*, New York, 1-30.

[13] MedlinePlus (2019). *Bird Flu Also: Avian Flu, Avian influenza, H5N1, H7N9.* U.S. National Library of Medicine, Medline Plus Trusted Health Information for you. Retrieved from https://medlineplus.gov/birdflu.html

[14] Mishra, B.K. & Sinha, N.D. (2016). Mathematical Model on Avian Influenza with Quarantine and Vaccination. *Journal of Immunol Tech. Infect Diseases* Doi:10.4172/2329

[15] Muhammad K., Amidi, and Arief A. (2019). Mathematical Model of Avian Influenza Epidemics with Vaccinating Poultry and Giving Treatment to Quarantined Infected Human?in *UNNES International Conference on Research Innovation and Commercialization 2018, KnE Social Sciences*, 397?07. DOI 10.18502/kss.v3i18.4731

[16] Putri A.R., Watanabe M., and Nova T.D. (2017). Study on Bird Flu Infection Process within a Poultry Farm with effects of Spatial Diffusion. *Universal Journal of Agricultural Research.* 5(3)190-196. DOI: 10.13189/ujar.2017.050302

[17] Somma, S. A., N. I Akinwande, M. Jiya & S. Abdulrahaman (2017). Stability Analysis of a Disease Free Equilibrium (DFE) State of a Mathematical Model of Yellow Fever Incorporating Secondary Host. *Pacific Journal of Science and Technology*, 18(2), 110-119.

[18] Tilahun G.T., Makinde O.D., & Malonza D. (2017). Modelling and Optimal Control of Typhoid Fever Disease with Cost-Effective Strategies. *Computational and Mathematical Methods in Medicine, Hindawi*, https://doi.org/10.1155/2017/2324518

[19] World Health Organization-WHO (2018). Influenza (Avian and Other Zoonotic). Retrieved from https://www.who.int/en/news-room/fact-sheets/detail/influenza-(avian-and-other-zoonotic)

[20] World Health Organization-WHO (2019). FAQs:H5N1 Influenza. Retrieved from https://www.who.int/influenza/human_animal_interface/avian_influenza/h5n1_research/faqs/en/

[21] Zhang J. Li Y., Jin Z., & Zhu H. (2019). Dynamics Analysis of an Avian Influenza A (H7N9) Epidemic Model with Vaccination and Seasonality. *Hindawi Complexity* https://doi.org/10.1155/2019/4161287