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A Model formulation for the Transmission Dynamics of Avian Influenza

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Abstract:*A deterministic model for the transmission dynamics of avian influenza is formulated. The model is extended from the model proposed by Okosun and Yusuf (2007) by incorporating the culling of infected birds and isolation of infected humans with avian influenza. This study allows for recovery of infected humans. The model showed that the biological feasible region is positively – invariant and attracting. The behaviour of the solutions is illustrated by simulation with different parameter values.*
Key words:*Avian Influenza, Mathematical Model, Positive-Invariant Region, Proportions, Simulation*

I. Introduction

Avian influenza or bird- flu is a contagious disease of animals caused by influenza A virus that normally infects mostly birds and less commonly, pigs (Arora and Arora, 2008; Alexander, 2000). In recent times the term bird flu has been used to describe the H5N1 avian influenza virus that occurs mainly in birds. It is highly contagious among birds and can be deadly to them. Infected birds shed influenza virus in their saliva, nasal secretions and faeces. Susceptible birds become infected when they have contact with contaminated secretions or excretions or with surfaces that are contaminated with secretions or excretions from infected birds (DeJong and Hien, 2006).

Infection with avian influenza A viruses in birds (wild and domestic) causes two main forms of diseases that are distinguished by low and high extremes of virulence, namely low pathogenic avian influenza (LPAI) and highly pathogenic avian influenza (HPAI). The 'low pathogenic' form may go undetected and usually causes only mild symptoms such as ruffled feathers and a drop in egg production in domestic poultry. However the high pathogenic form spreads more rapidly through flocks of poultry. The mortality can approach 100%, often within 48 hours (DeJong and Hien, 2006).

Most cases of avian influenza infection in humans have resulted from direct or close contact with infected poultry such as domesticated chickens, ducks and turkeys or surfaces contaminated with secretions and excretions from infected birds (De Jong and Hien, 2006). Avian influenza remains a very rare disease in humans. Of the human cases associated with the ongoing H5N1 outbreaks in poultry and wild birds in Asia and part of Europe, the Near East and Africa, more than half of those reported infected with the virus have died (WHO, 2008).

If a person is infected, it is very difficult for the virus to spread to another person (Claaset al, 1998; The Writing Committee of the World Health Org. (WHO) Consultation on Human Influenza A/H5, 2006; Longiniet al, 2005).

The objective of any control programme for avian influenza is to safeguard human health, livelihoods of families, and the commercial poultry production sector from the threat of high pathogenic avian influenza by controlling outbreaks quickly. Eliminating the source of infection, that is, infected birds, remains the most effective infection control measure. Mass culling of avian hosts has been the long – standing practice for influenza control within the avian reservoir. Other control measures are primarily based on the use of non – pharmaceutical intervention such as increased hygiene, use of protective devices, isolation in hospital wards, and quarantine of suspected cases, and the use of pharmaceutical interventions such as the use of antivirals like Tamiflu and Relenza and vaccines (Kalupnieks, 2004; WHO, 2004; Butler, 2006).

A number of mathematical modelling studies have been carried out to quantify the potential burden of influenza pandemic and to assess various control strategies (Ferguson et al 2005;Longiniet al, 2005). In the case of avian influenza, deterministic models were used for comparing interventions aimed at preventing and controlling (Derouich and Boutyeb 2008;Srinivasa, 2008) and stochastic models were proposed to model and predict the world wide spread of pandemic influenza (Collizzaet al, 2007 Le Menachet al, 2005). Although many of these studies tend to emphasize the use of pharmaceutical interventions, it is generally believed that such interventions (antivirals and vaccines) would not be readily and widely available at the onset of the pandemic.

The motivation for this study lies in the model by Okosun and Yusuf (2007). They proposed mathematical models that describe the transmission dynamics in birds and human population subject to the

highly pathogenic avian influenza without any emphasis on control strategy. The current study is an extension of the studies by Okosun and Yusuf (2007) by incorporating the dynamics of both wild and domestic birds (only domestic birds (chicken) were considered in Okosun and Yusuf (2007)), the culling of infected birds, and the isolation of infected individuals with avian influenza strain. We model wild and domestic birds using linear population model to generalize the situation, instead of allowing for a constant population of migratory birds. This gives room for a long term study of the dynamics of the infection. Furthermore, the extended model allows for recovery of individuals infected with avian strain.

II. Model Formulation

In describing the new model we subdivide the total avian (birds) population at time t, denoted by $N_B(t)$ into susceptible wild birds, $S_W(t)$, susceptible domestic birds, $S_D(t)$, infected wild birds, $I_W(t)$, and infected domestic birds, $I_D(t)$, so that

$$N_B(t) = S_W(t) + S_D(t) + I_W(t) + I_D(t).$$

In the human population, we assume that humans infected with avian influenza cannot infect susceptible humans. Thus the total human population at time t, denoted by $N_H(t)$ is sub-divided into susceptible humans, $S_H(t)$, infected humans, $I_H(t)$, isolated infected humans, $Q_H(t)$, and recovered humans, $R_H(t)$, so that

$$N_H(t) = S_H(t) + I_H(t) + Q_H(t) + R_H(t)$$

The variables and parameters used in the model are defined below:

Table 1: Variables and Parameters used in the model and their description

Variable/Parameter	Description
$N_W(t)$	Total number of wild birds at time t
$N_D(t)$	Total number of domestic birds at time t
$N_H(t)$	Total number of humans at time t
$S_W(t)$	Total number of Susceptible wild birds at time t
$I_W(t)$	Total number of Infected wild birds at time t
$S_D(t)$	Total number of Susceptible domestic birds at time t
$I_D(t)$	Total number of Infected domestic birds at time t
$S_H(t)$	Total number of Susceptible humans at time t
$I_D(t)$	Total number of Infected humans at time t
$Q_H(t)$	Total number of Isolated humans with avian strain at time t
$R_H(t)$	Total number of Recovered humans at time t
β_W	Average birth rate in wild birds
β_D	Average birth rate in domestic birds
$\alpha_W, \alpha_D, \alpha_A$	Infection transmission rates for birds
η	Destruction (culling) rate for infected birds
δ_B	Natural death rate in birds
d_W	Flu induced death rate in wild birds
d_D	Flu induced death rate in domestic birds
β_H	Average birth rate in humans
δ_H	Natural death rate in humans
d_H	Flu induced death rate in humans
ε_H	Isolation rate for humans with avian stain
ϑ_H	Flu induced death rate in Isolated humans ($\vartheta_H < d_H$)
ν	Recovery rate without immunity
γ	Recovery rate with substantial immunity
σ	Loose of immunity rate in recovered humans

A schematic flow diagram of the extended model for the birds' population and human population is shown in Figure 1 below.

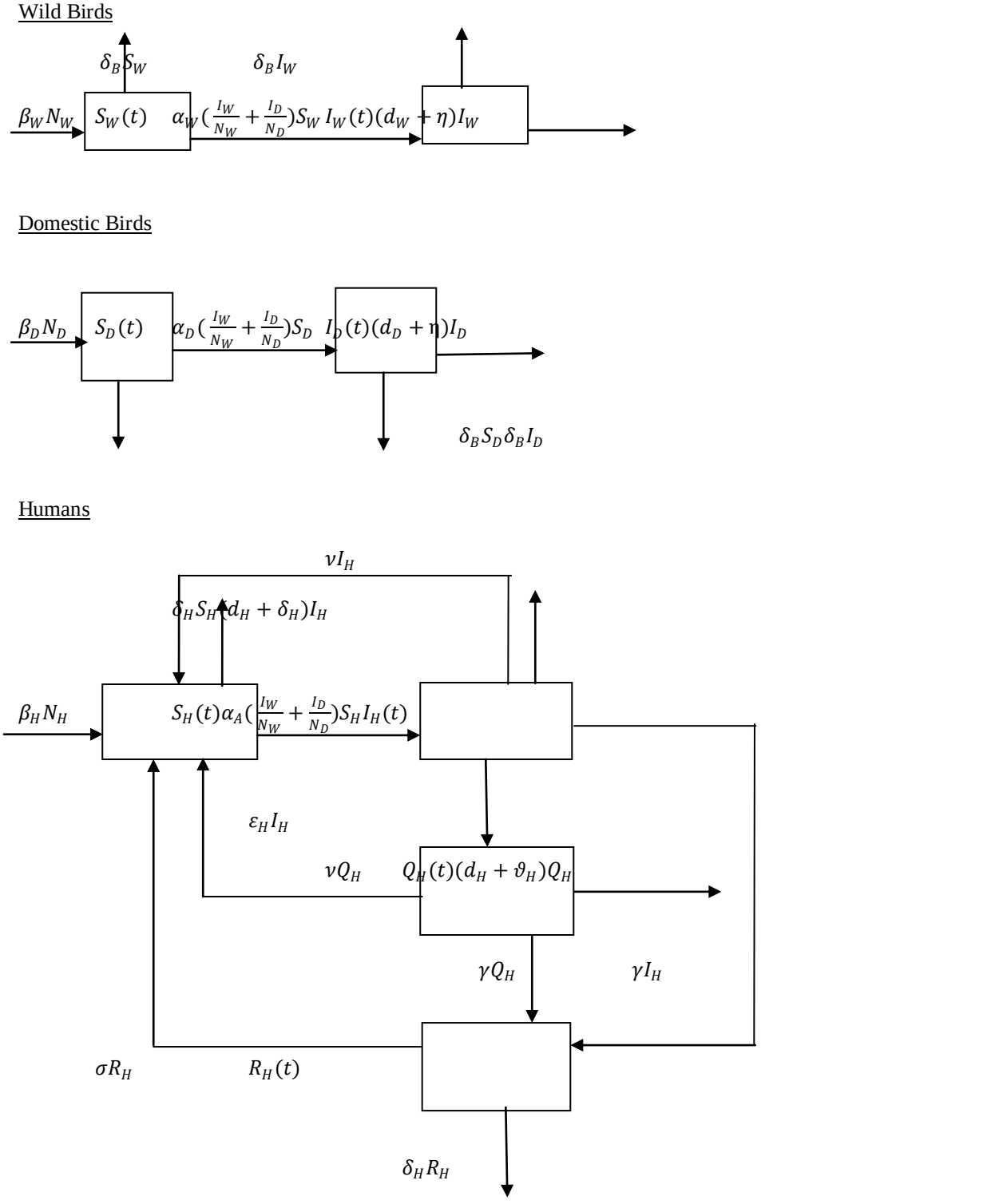

Figure 1: Schematic diagram of the transmission dynamics of avian influenza (H5N1) in birds and human population.

2.1 Susceptible and Infected Wild Birds

The population of susceptible wild birds is generated by birth of wild birds (at the rate β_W). It is reduced by infection, following contact with infected wild birds and infected domestic birds (at the rate α_W), where α_W is the infection transmission rate for wild birds and further reduced by natural death (at the rate δ_B). Hence

$$\frac{dS_W}{dt} = \beta_W N_W - \alpha_W \frac{I_W}{N_W} S_W - \alpha_W \frac{I_D}{N_D} S_W - \delta_B S_W,$$
$$= \beta_W N_W - \alpha_W \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_W - \delta_B S_W,$$

The population of infected wild birds is increased through the infection of susceptible wild birds following contact with infected wild birds and infected domestic birds. It decreased either by natural death (at the rate δ_B) and avian induced mortality (at the rate d_W) and by culling of infected wild birds (at the rate η). So that

$$\frac{dI_W}{dt} = \alpha_W \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_W - (d_W + \delta_B + \eta) I_W,$$

2.2 Susceptible and Infected Domestic Birds

The population of susceptible domestic birds is generated by birth of domestic birds (at the rate β_D). It is reduced by infection, following contact with infected wild birds and infected domestic birds (at the rate α_D), where α_D is the infection transmission rate for domestic birds and further reduced by natural death (at the rate δ_B). Thus

$$\frac{dS_D}{dt} = \beta_D N_D - \alpha_D \frac{I_W}{N_W} S_D - \alpha_D \frac{I_D}{N_D} S_D - \delta_B S_D,$$
$$= \beta_D N_D - \alpha_D \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_D - \delta_B S_D,$$

The population of infected domestic birds is increased through the infection of susceptible domestic birds following contact with infected wild birds and infected domestic birds. It decreased either by natural death (at the rate δ_B) and avian induced mortality (at the rate d_D) and by culling of infected wild birds (at the rate η). This yield

$$\frac{dI_D}{dt} = \alpha_D \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_D - (d_D + \delta_D + \eta) I_D,$$

2.3 Susceptible, Infected, Isolated and Recovered Humans

The population of susceptible humans are increased by birth (at the rate β_H), recovered humans who lost immunity to return to susceptible humans (at the rate σ), recovered infected and isolated humans without immunity (at the rate ν). It decreased by infection of susceptible humans following contact with infected wild birds and infected domestic birds (at the rate α_B), where α_B is the infection transmission rate for humans and further reduced by natural death (at the rate δ_H). This gives

$$\frac{dS_H}{dt} = \beta_H N_H - \alpha_B \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_H - \delta_H S_H + \nu I_H + \nu Q_H + \sigma R_H,$$

Infected humans are generated through infection of susceptible humans following contact with infected wild birds and infected domestic birds (at the rate α_B) and reduced by natural death (at the rate δ_H) and avian induced mortality (at the rate d_D). It is further reduced by isolation of infected humans (at the rate ε_H) recovered infected humans without immunity and with substantial immunity (at the rate ν and γ respectively). Thus,

$$\frac{dI_H}{dt} = \alpha_B \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_H - (\varepsilon_H + d_H + \delta_H + \nu + \gamma) I_H,$$

Isolated humans are generated by isolation of infected humans (at the rate ε_H) and reduced by natural death (at the rate δ_H) and avian induced mortality (at the rate ϑ_H where, $\vartheta_H < d_H$; it is assumed that isolated individuals are given some treatment such as Tamiflu). It is further reduced by recovered isolated humans without immunity and those with substantial immunity (at the rate ν and γ respectively). Hence,

$$\frac{dQ_H}{dt} = \varepsilon_H I_H - (\nu + \vartheta_H + \gamma + \delta_H) Q_H,$$

The recovered humans are generated by the recovery of infected humans and isolated humans (at the rate γ). Decreased by natural death (at the rate δ_H) and losing immunity (at the rate σ). So that

$$\frac{dR_H}{dt} = \gamma I_H + \gamma Q_H - (\sigma + \delta_H) R_H,$$

The above assumptions and derivations leads to the following system of ordinary differential equations

$$\frac{dS_W}{dt} = \beta_W N_W - \alpha_W \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_W - \delta_B S_W, \quad (1)$$
$$\frac{dI_W}{dt} = \alpha_W \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_W - (d_W + \delta_B + \eta) I_W, \quad (2)$$
$$\frac{dS_D}{dt} = \beta_D N_D - \alpha_D \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_D - \delta_B S_D, \quad (3)$$
$$\frac{dI_D}{dt} = \alpha_D \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_D - (d_D + \delta_D + \eta) I_D, \quad (4)$$
$$\frac{dS_H}{dt} = \beta_H N_H - \alpha_B \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_H - \delta_H S_H + \nu I_H + \nu Q_H + \sigma R_H, \quad (5)$$
$$\frac{dI_H}{dt} = \alpha_B \left(\frac{I_W}{N_W} + \frac{I_D}{N_D}\right) S_H - (\varepsilon_H + d_H + \delta_H + \nu + \gamma) I_H, \quad (6)$$
$$\frac{dQ_H}{dt} = \varepsilon_H I_H - (\nu + \vartheta_H + \gamma + \delta_H) Q_H, \quad (7)$$
$$\frac{dR_H}{dt} = \gamma I_H + \gamma Q_H - (\sigma + \delta_H) R_H, \quad (8)$$

III. The Model Equations In Proportions

For prevalence of the disease, it is necessary to consider the model in proportions of susceptible, infectious, isolated and recovered compartments.

Adding equations (1) - (2) and equations (3) – (4) gives

$$\frac{dN_W}{dt} = \beta_W N_W - \delta_B N_W - (d_w + \eta) I_W \quad (9)$$

and

$$\frac{dN_D}{dt} = \beta_D N_D - \delta_B N_D - (d_D + \eta) I_D \quad (10)$$

Similarly, adding equations (5) – (8) gives the rate of change of the total human population:

$$\frac{dN_H}{dt} = \beta_H N_H - \delta_H N_H - d_H I_H - \vartheta_H Q_H \quad (11)$$

We now define the proportion for each class as follows:

$$s_w = \frac{S_W}{N_W}, i_W = \frac{I_W}{N_W}, s_D = \frac{S_D}{N_D}, i_D = \frac{I_D}{N_D}, s_H = \frac{S_H}{N_H}, i_H = \frac{I_H}{N_H}, q_H = \frac{Q_H}{N_H}, r_H = \frac{R_H}{N_H},$$

So that

$$s_W + i_W = 1 \Rightarrow s_W = 1 - i_W, s_D + i_D = 1 \Rightarrow s_D = 1 - i_D$$

and

$$s_H + i_H + q_H + r_H = 1 \Rightarrow s_H = 1 - i_H - q_H - r_H$$

Thus, the system (1) – (8) expressed in proportion is given below:

$$\frac{ds_W}{dt} = \beta_W - \alpha_W (i_W + i_D) s_W - \beta_W s_W + (d_w + \eta) i_W s_W \quad (12)$$
$$\frac{di_W}{dt} = \alpha_W (i_W + i_D) s_w - (d_W + \beta_W + \eta) i_W + (d_w + \eta) i_W^2 \quad (13)$$
$$\frac{ds_D}{dt} = \beta_D - \alpha_D (i_W + i_D) s_D - \beta_D s_D + (d_D + \eta) i_D s_D \quad (13)$$
$$\frac{di_D}{dt} = \alpha_D (i_W + i_D) s_D - (d_D + \beta_D + \eta) i_D + (d_D + \eta) i_D^2 \quad (14)$$
$$\frac{ds_H}{dt} = \beta_H - \alpha_B (i_W + i_D) s_H + \nu (i_H + q_H) + \sigma r_H - \beta_H s_H + d_H s_H i_H + \vartheta_H s_H q_H \quad (15)$$
$$\frac{di_H}{dt} = \alpha_B (i_W + i_D) s_H - (\varepsilon + d_H + \beta_H + \nu + \gamma) i_H + \vartheta_H i_H q_H + d_H i_H^2 \quad (16)$$
$$\frac{dq_H}{dt} = \varepsilon_H i_H - (\nu + \vartheta_H + \gamma + \beta_H) q_H + d_H i_H q_H + \vartheta_H q_H^2 \quad (17)$$
$$\frac{dr_H}{dt} = \gamma i_H + \gamma q_H - (\sigma + \beta_H) r_H + d_H i_H r_H + \vartheta_H q_H r_H \quad (18)`$$

IV. Invariant Region

The avian influenza model (12) – (18) in proportionswill be analyzed to establish the biological feasible region as follows. The system (12) – (18) is split into two parts, namely the avian population where $n_B(t) = s_w(t) + i_W(t) + s_D(t) + i_D(t)$and the human population where$n_H(t) = S_H(t) + i_H(t) + q_H(t) + r_H(t)$. Consider the feasible region

$\Omega = \Omega_B \cup \Omega_H \subset \mathbb{R}_+^4 \times \mathbb{R}_+^4$

with

$\Omega_B = \{(s_w, i_W, s_D, i_D) \in \mathbb{R}_+^4 : s_w + i_W + s_D + i_D \leq 1\}$

and
$\Omega_H = \{(s_H, i_H, q_H, r_H) \in \mathbb{R}_+^4 : s_H + i_H + q_H + r_H \leq 1\}$

The following steps are followed to establish the positive invariance of Ω(i.e., the solution in Ω remain inΩ for all $t > 0$). The rate of change of the avian and human population (by adding the first four equations and the last four equations of the model (12) – (18)) is given

$\frac{dn_B}{dt} = \beta_B - \beta_B n_B + (d_w + \eta)i_W s_W - (d_W + \eta)i_W + (d_w + \eta)i_W^2 + (d_D +)i_D s_D - (d_D + \eta)i_D + (d_D +$
$\eta iD2$(19)
and
$\frac{dn_H}{dt} = \beta_H - \beta_H n_H + d_H s_H i_H + \vartheta_H s_H q_H - \beta_H i_H + \vartheta_H i_H q_H + d_H i_H^2 - \beta_H q_H + d_H i_H q_H + \vartheta_H q_H^2 - \beta_H r_H +$
$d_H i_H r_H + \vartheta_H q_H r_H$ (20)

It follows from (19) and (20) that
$\frac{dn_B}{dt} \leq \beta_B - \beta_B N_B$ (21)
and
$\frac{dn_H}{dt} \leq \beta_H - \beta_H n_H$ (22)
Integrating (21) with respect to t where the integrating factor, $IF = e^{\int \beta_B dt} = e^{\beta_B t}$ we have
$e^{\beta_B t} n_B \leq \int \beta_B e^{\beta_B t} dt + C$
$\Rightarrow e^{\beta_B t} n_B \leq e^{\beta_B t} + C$
$\therefore n_B(t) \leq 1 + Ce^{-\beta_B t}$
At $t = 0$,
$C = n_B(0) - 1$
$\therefore n_B(t) \leq 1 + (n_B(0) - 1)e^{-\beta_B t}$
$n_B(t) \leq n_B(0)e^{-\beta_B t} + 1 - e^{-\beta_B t}$ (23)
Alsointegrating (22) with respect to t where the integrating factor, $IF = e^{\int \beta_H dt} = e^{\beta_H t}$ we have
$e^{\beta_H t} n_H \leq \int \beta_H e^{\beta_H t} dt + C$
$\Rightarrow e^{\beta_H t} n_H \leq e^{\beta_H t} + C$
$n_H(t) \leq 1 + Ce^{-\beta_H t}$

At $t = 0$,
$C = n_H(0) - 1$
$\therefore n_H(t) \leq 1 + (n_H(0) - 1)e^{-\beta_H t}$
$n_H(t) \leq n_H(0)e^{-\beta_H t} + 1 - e^{-\beta_H t}$ (24)
Applying the theorem of differential inequality (Birkhof and Rota, 1982) on equations (23) and (24) we obtain
$0 \leq n_B(t) \leq 1 \text{and} 0 \leq n_H(t) \leq 1$ as $t \to \infty$

Thus, the region Ω is positively invariant. Hence it is sufficient to consider the dynamics of the flow generated by (12) – (18) in Ω.In this region, the model can be considered as being epidemiologically and mathematically well posed. Thus every solution of the model (12) – (18) with initial conditions in Ω remains in Ω for all $t >$ 0.This result is summarized below.

Lemma 4.1: The region$\Omega = \Omega_B \cup \Omega_H \subset \mathbb{R}_+^4 \times \mathbb{R}_+^4$ is positively invariant for the basic model (12) – (18) with non-negative initial conditions in$\mathbb{R}_+^8$.

V. Numerical Simulations

In this section, we demonstrated numerically the long-term behavior of the solutions of the model in proportions. To achieve this, the model formulated in proportion was solved using Runge-Kutta 4th order method coded in MATLAB (ode45). The results from the simulations were as shown in Fig 2 – Fig 8.

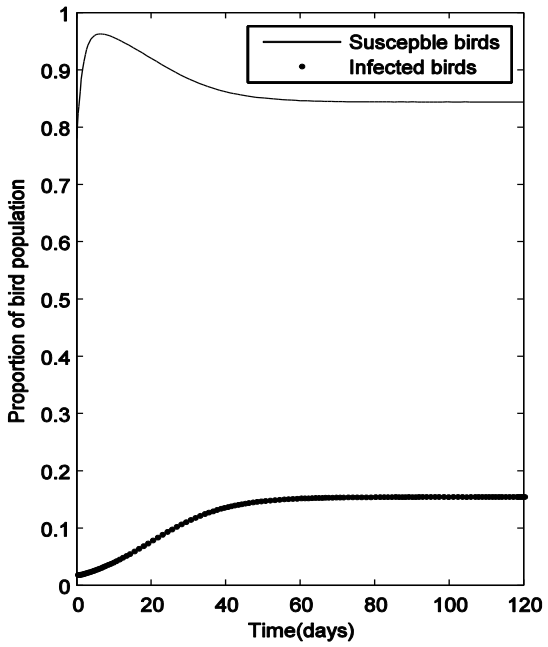

Fig 2: Proportion of susceptible and infected birds as birds as a function of time without control measure ($\alpha_B = 0.45$)

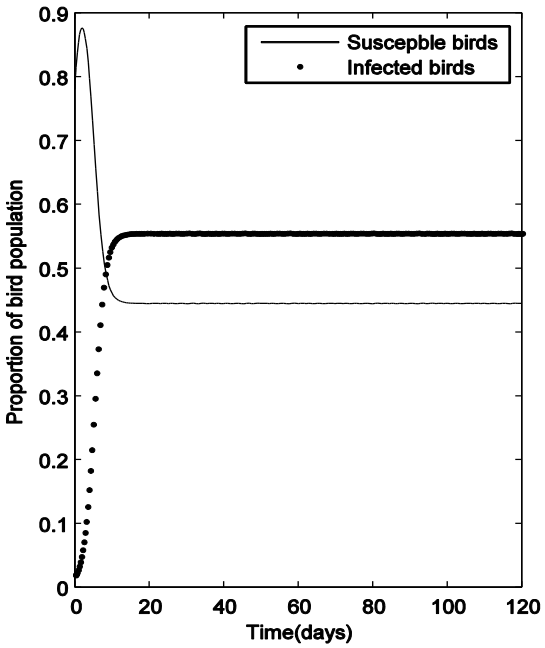

Fig 3: Proportion of susceptible and infected a function of time without control measure ($\alpha_B = 0.75$)

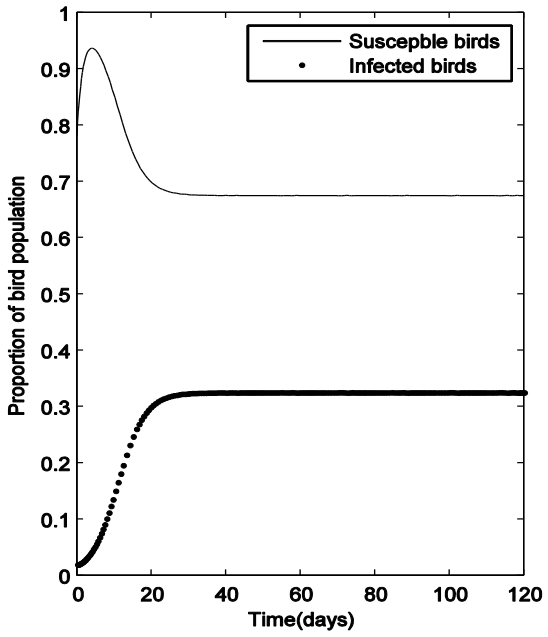

Fig 4: Proportion of susceptible and infected birds as birds as a function of time with culling ($\eta = 0.45$)

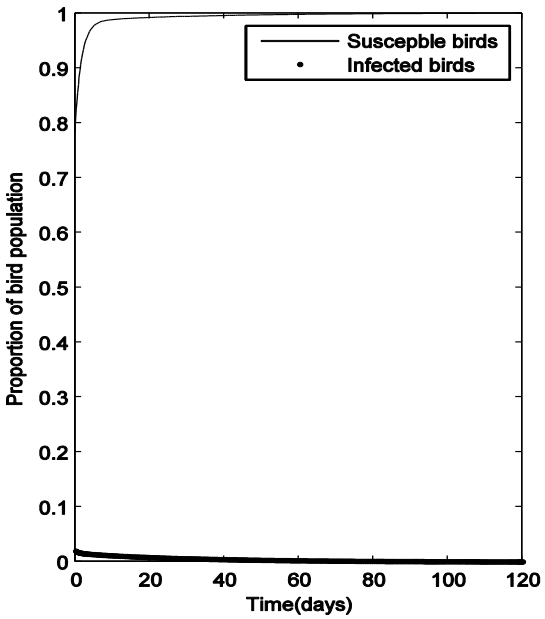

Fig 5: Proportion of susceptible and infected a function of time with culling ($\eta = 0.75$)

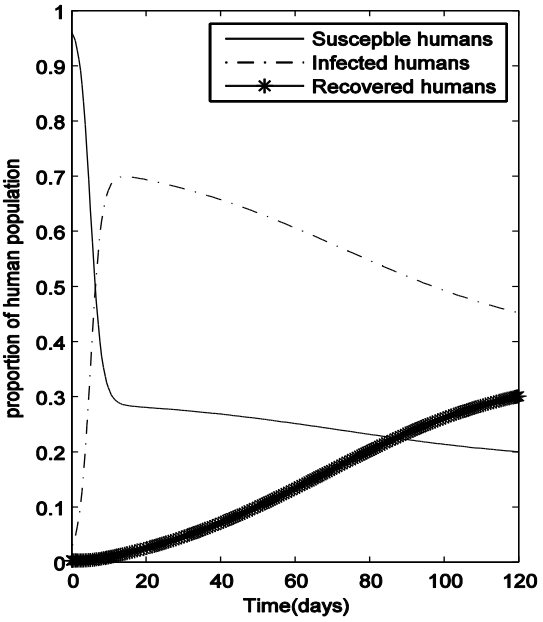

Fig 6: Proportion of human population as as a function of time without control measure ($\alpha_B = 0.75$)

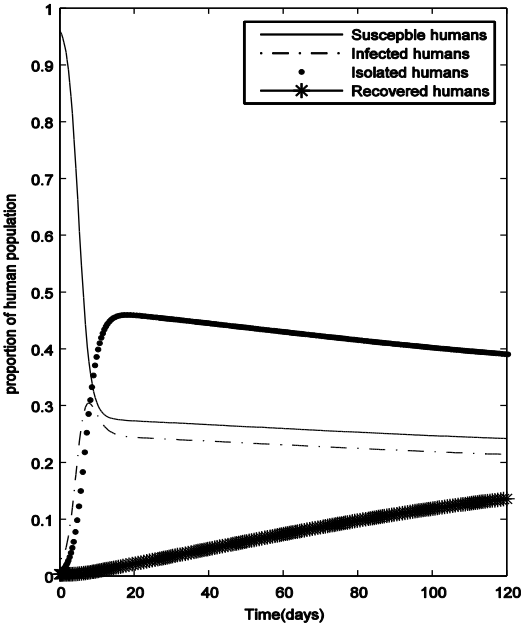

Fig 7: Proportion of susceptible and infected humans a function of time with isolation ($\varepsilon_H = 0.4$)

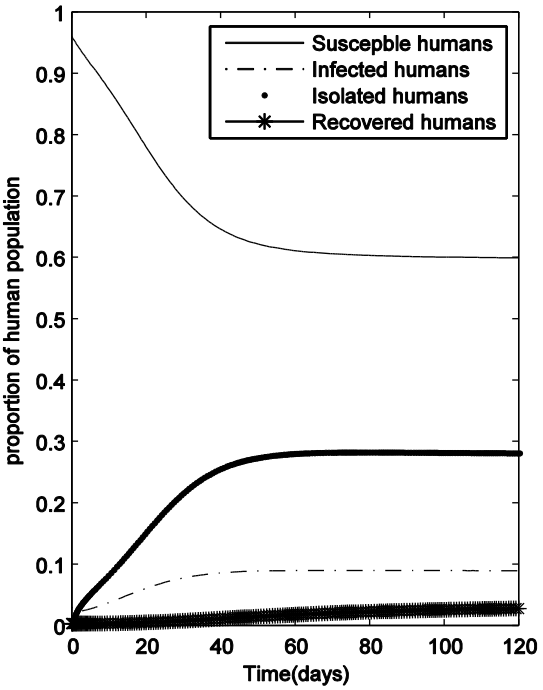

Fig 8: Proportion of human population as a function of time with combined control strategy ($\eta = 0.65\ \varepsilon_H = 0.7$)

As time increases from zero, Fig 2 shows the proportion of susceptible birds gradually drop and remained stable while the proportion of infected birds increases with low infection transmission rate ($\alpha_B = 0.45$), while Fig 3 capture the same flow but there is a sharp decrease in the proportion of susceptible birds and

a sharp increase in the proportion of infected birds with high infection transmission rate ($\alpha_B = 0.75$. In Fig 2 and Fig 3, the proportion of susceptible peaks and drops again capturing the typical behavior of infection during an epidemic.

Fig 4 shows a decrease in the proportion of susceptible birds and increase in the proportion of infected birds in the presence of low culling of infected birds, while Fig 5 shows an increase in the proportion of susceptible birds and a decrease in the proportion of infected over time with high culling of infected birds. The figure further revealed that with effective culling of infected birds, the disease can be eliminated from the birds' population.

As time increases from zero, Fig 6 the proportion of susceptible humans decreases while the proportion of infected and recovered compartments increases. The figure also shows that the proportion of infected humans peaks and drops gradually as the proportion of recovered humans increases.

Fig 7 shows a sharp decrease in the proportion of susceptible humans and increase in the proportion of infected, isolated and recovered humans in the presence of low isolation rate for the proportion of infected birds.

Fig 8 shows that the proportion of susceptible humans gradually decreases and remains stable as time increases while the proportion of infected, isolated and recovered humans gradually increases and remains stable over time in the presence of culling of infected birds and isolation of infected humans. It implies that the combined control strategy could have a great impact in infection reduction.

VI. Conclusion

In this paper, a deterministic model for the transmission dynamics of avian influenza was formulated. The study incorporate culling of infected birds and isolation of infected humans as intervention strategy shows the following:

The model in proportion showed that the existing biological feasible region is positively – invariant and attracting.

The numerical simulation showed that any control strategy aimed at reducing the infection transmission will go a long way in eradication avian influenza infection.

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