Mathematical modeling and nonstandard finite difference scheme analysis for the environmental and spillover transmissions of Avian Influenza A model

A. Feukouo Fossi[1] J. Lubuma[2] C. Tadmon[1,3] B. Tsanou[1,2,4,*]

[1]Department of Mathematics and Computer Science, University of Dschang, P. O. Box 67, Dschang, Cameroon
[2]Department of Mathematics and Applied Mathematics, University of Pretoria, Pretoria 0002, South Africa
[3]The Abdus Salam International Centre for Theoretical Physics, Trieste, Italy
[4]IRD UMI 209 UMMISCO, University of Yaounde I, P.O. Box 337 Yaounde, Cameroon and LIRIMA-EPITAG Team Project, University of Yaounde I, P.O. Box 812 Yaounde, Cameroon

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ABSTRACT
This work is devoted to the modeling, analysis and assessment of the impacts of environmental and spillover transmissions on Avian Influenza Virus (AIV) type A infection. We formulate a nonlinear ordinary differential system for an Avian Influenza Virus transmission that takes into account five spreading pathways: poultry-to-poultry; environment-to-poultry; poultry-to-human (spillover event); environment-to-human and poultry-to-environment. An in-depth theoretical and numerical analysis of the model is done. For the sub-model without recruitment of infected poultry, the basic reproduction number is computed and serves to prove the global stability of the disease-free equilibrium whenever it is less or equal to unity. Moreover, whenever it is greater than one, the existence of the unique endemic equilibrium is shown and its global stability is established. These global results are shown thanks to the construction of suitable Lyapunov functions based on the judicious choices of Volterra-Lyapunov stable matrices and the application of Poincaré-Bendixson and Lyapunov-LaSalle techniques. When the infected poultry is brought into the population, the model does not have a disease-free equilibrium and exhibits a unique endemic equilibrium whose global asymptotic stability is established similarly using the techniques mentioned above. Further, the model is shown to exhibit a transcritical bifurcation with the value one of the basic reproduction number being the bifurcation parameter threshold. We further prove that during avian influenza epidemics outbreaks, the recruitment of infected poultry increases the endemic level of the disease. We show that the classical Runge-Kutta numerical method fails to preserve the positivity of the solutions and alternatively design a nonstandard finite difference scheme (NSFD), which preserves the essential properties of the continuous system. Numerical simulations are then implemented to illustrate the theoretical results obtained and assess the role of the environmental and spillover transmissions on the disease.

KEYWORDS
Avian Influenza Virus, Environmental transmission, Bifurcation, Spillover, NSFD method, Global stability.

1. Introduction

The avian influenza virus infection is caused by viruses adapted to birds and it normally affects wild birds and poultry. The wild birds are natural reservoir for all the sub-types of influenza A viruses. Influenza viruses are widespread and due to their high mutation rate many subtypes exist. Further, H5N1, H7N4, H7N7, H7N9, H9N2, and other avian influenza

* Corresponding author. berge.tsanou@up.ac.za, Email: bergetsanou@yahoo.fr

viruses with pathogenicity have great potential threat to human. Poultry farms are an important reservoir for the avian influenza virus (AIV) [1]. AIV transmission to humans is largely facilitated by contact with animals and excretion of contaminated droplets or aerosols [2], and to a lesser extent through transport of (dead) birds or contaminated objects (vehicles, humans, or fomites), water, food, and contact with infected wildfowl or insects [3]. Historically, the avian influenza splits into two classes: the "High Pathogenic Avian Influenza (HPAI)" and the "Low Pathogenic Avian Influenza (LPAI)". The HPAI can cause a series of systemic infections that can lead to high mortality. The LPAI causes mild or no symptoms. In general, the risk of direct transmission of avian influenza to human is very low. However, in 2014 a high mortality rate was recorded with around 38.7% of the patients infected with H7N9 virus dead [4]. H7N9 virus can cause pneumonia, respiratory failure, acute respiratory distress syndrome and multi-organ failure.

Some subtypes of AIV, namely H7N9 virus is low pathogenic in poultry although it is high pathogenic in humans. As the infection with H7N9 virus does not cause any symptoms in poultry, it is not easy to monitor the transmission of the virus in poultry farm and from poultry to human. Because of this, there is an urgent need of continued surveillance in the poultry farms. The incubation period for a human infected with H7N9 influenza virus is about seven days and at present there are some medicines to fight against this virus. Although, these antiviral drugs are clinically effective against the H7N9 avian influenza, the mortality due to H7N9 avian influenza is still very high. Normally, H7N9 virus is not thought to have a strong capacity for an efficient human-to-human spread, however there have been two cases of the familial aggregation. Under such circumstances, it is important to study what can be the best available policies for prevention and control of the transmission of H7N9 avian influenza.

Mathematical models have been used to understand the transmission dynamics of avian influenza for some time now. The medical experts have analyzed the sources of infection, the route of transmission and susceptible population. However, these are clearly not enough and a more focused research on the epidemic dynamic model is expected to help us better understand and control the avian influenza. Iwami et al.[5–7] reported many research results on the mathematical modeling of influenza. In 2008, they developed a mathematical model to study the pattern of spread of the mutant avian influenza and later in 2009, they investigated the relations between the evolution of virulence and effectiveness of the pandemic control measures after an emergence of the mutant avian influenza. A deterministic path-structured model in heterogeneous environment was also studied by them in 2009. Bourouiba et al. [8] presented a model to describe the behavior of both HPAI and LPAI strains in a domestic bird population with the culling effect. Chong et al. [4] estimated the basic reproduction number and the mean number of cases generated by poultry-to-human transmission of influenza A (H7N9) infection. Later, to understand the efficacy of screening and culling of infected poultry on the transmission dynamics of influenza A (H7N9), Liu and Fang [9] formulated a mathematical model by considering both the human and poultry infections and they assumed that the transmission of this disease from poultry to human as well as human to human can be modeled using a simple mass action type incidence. Chen and Wen [10] investigated the dynamic properties of H7N9 avian influenza models. Again, they also considered a simple mass-action type incidence for both the bird to bird and bird to human transmissions. They did not consider the human-to-human route of transmission. A delay differential equation model for the avian influenza was formulated and analyzed by Liu et al. [11]. Recently, Shu-Min Guo et al. [12] analyzed the model of avian A (H7N9) based on low pathogenesis on poultry. They considered the saturation type incidence in the poultry, the import of poultry, so some fraction of the total recruitment in the poultry is assumed infected. They also did not consider human-to-human transmission of avian influenza A.

In the present study, motivated by the biological papers [1–3], we build on the baseline mathematical model in [12], extend it and focus on the important, yet neglected role of aerosol on the transmission of AIV. We achieve this by considering the indirect transmission through

the incorporation of a compartment for the concentration of free-living avian influenza A viruses in the soil of the poultry farms, generated by aerosols. We neglect the human-to-human transmission of the avian influenza A, because it is very rare. However, knowing that poultry-to-human transmission is also rare, but devastating, the incorporation of an additional indirect transmission route allows us to account for the spillover infection of AIV from poultry and environment to humans. Moreover, since the avian influenza A does not cause typical clinical signs in the infected poultry, we assume on the one hand that the poultry remains in incubation period without being identified as sick but being capable of transmitting the virus from poultry to human. On the other hand, the culling effect of infected poultry is not considered as it is not easy to identify the infected poultry. The resulted model is deeply analyzed both theoretically and computationally. From the analytical perspectives, we established the threshold dynamic of the system and transcritical bifurcation using Lyapunov-LaSalle, Poincaré-Bendixson techniques and center manifold approximation, respectively. Since the model is highly nonlinear, as usual it can not be solved explicitly. Worse still, the powerful classical Runge-Kutta method failed to preserve the positivity of solutions [13, 14]. Therefore, we overcome this by designing a non-standard finite difference scheme which is dynamically consistent with the continuous model [14–20] and used it to illustrate the theoretical results and assess the role of the spillover and environmental transmissions of Avian Influenza A.

The outline of the remainder of the paper is as follows. In Section 2 we build an avian influenza model that incorporates spillover and indirect transmissions and give the model's basic properties. Section 3 deals with the theoretical and bifurcation analysis of the continuous model, while Section 4 presents a dynamically consistent discrete NSFD scheme with which the theoretical results are numerically illustrated and the role of environmental and spillover transmissions assessed in Section 5. Finally, we conclude the paper in Section 6 and provide some discussions that highlight few relevant perspectives.

2. Model formulation and basic properties

2.1. Model derivation

2.1.1. Dynamics of the susceptible poultry:

The time evolution of the number of susceptible poultry is described as follows. We assume that a total number A of poultry replenishes the farm due to importation per unit time. From this quantity, a proportion $(1-q)A$ is susceptible poultry, while the remaining proportion qA is infected poultry. Susceptible poultry die at rate dX. Upon the direct transmission among poultry, susceptible poultry moves to asymptomatic class following a saturation type incidence at rate $\beta_v XY/(1+\alpha Y)$, where β_v is the transmission coefficient, such that $\beta_v Y$ measures the infection force of the infective poultry, α being the parameter standing for the inhibitory effort, and $1/(1+\alpha Y)$ describing the saturation due to the protection measures of the poultry farmers or the crowding of infected poultry when the number of infective poultry increases [11]. Upon indirect transmission, $\beta_e XC/(C+\kappa)$ corresponds to the incidence rate between environmental contaminated food particles and susceptible poultry. In the latter saturated incidence function, β_e is the transmission coefficient such that $(\beta_e \gg \beta_v)$; $1/(C+\kappa)$ represents saturation due to the cleaning of the farms when the concentration of excretion becomes larger; κ is the concentration of V. avian viruses attached to aerosol particles in the farm which 50% chance of catching the infection. Thus, the variation of X is monitored by the equation:

$$\frac{dX}{dt} = (1-q)A - \beta_v X\frac{Y}{1+\alpha Y} - \beta_e X\frac{C}{C+\kappa} - dX. \tag{1}$$

2.1.2. Dynamics of the asymptomatic poultry

The proportion qA imported poultry is asymptomatic and enters the infected class Y, where they eventually die at rate dY. New infected in the Y-class due to infection contact between susceptible and infected poultry come from two sources: direct transmission and indirect environmental transmission. All these descriptions yield the equation

$$\frac{dY}{dt} = qA + \beta_v X \frac{Y}{1+\alpha Y} + \beta_e X \frac{C}{C+\kappa} - dY. \tag{2}$$

2.1.3. Dynamics of the concentration of AIV in the poultry farms environment

This dynamics is derived as follows. Since an emission rate for pathogens is defined as an amount released per unit of time, it depends on source type (pigs, poultry, industrial, humans, etc.), source characteristics (e.g., stable construction or animal activity), excretion route (e.g., exhaled air or faeces), pathogen species or strain, particle size, etc. For a full quantitative risk assessment, quantified emission rates are required. Hence, the contribution by humans and poultry in the contamination of the poultry farms is respectively $\phi_1 I$ and $\phi_2 Y$; and the degradation or decontamination rate of virus (inactivation) due to the temperature or humidity is ξ. We can neglect the contribution by humans because of the human protection when entering in poultry farms. The time evolution of C reads as follows:

$$\frac{dC}{dt} = \phi_2 Y - \xi C. \tag{3}$$

2.1.4. Dynamics of the susceptible human individuals

New born or immigrated humans are recruited susceptible at rate B and die naturally at rate δ. Since there are some medicines to fight against this virus, the latent and the infected humans recover respectively at rate a and γ. The transmission of this disease from poultry to human occurs at rate τ_v and τ_e is the transmission coefficient of this disease from the pathogenic or infectious environment to human. Here, the incidence terms corresponding to interaction between poultry and human or environment and human are taken in such a way that makes this model more suitable for the study of avian influenza A in human associated with poultry farms. Thus, the variation of S is expressed by the following ODE:

$$\frac{dS}{dt} = B + aE + \gamma I - \tau_v \frac{S}{N} Y - \tau_e \frac{S}{N} C - \delta S. \tag{4}$$

2.1.5. Dynamics of the latent human individuals

Here, the morbidity of the latent human is ϵ. Thus, the time evolution of E is expressed by the following ODE:

$$\frac{dE}{dt} = \tau_v \frac{S}{N} Y + \tau_e \frac{S}{N} C - (a + \delta + \epsilon) E. \tag{5}$$

2.1.6. Dynamics of the infected human individuals

Here, the disease-related death rate is ρ, with ($\rho \gg \delta$). Then, the dynamics of I is modeled by the following ODE:

$$\frac{dI}{dt} = \epsilon E - (\gamma + \rho + \delta) I. \tag{6}$$

The parameters in the model are summarized and explained in Table 3. They are assumed non-negative. The model flowchart is depicted in Figure 1 from which we derive the system of ordinary differential equations in (7) governing the dynamics of the constructed model.

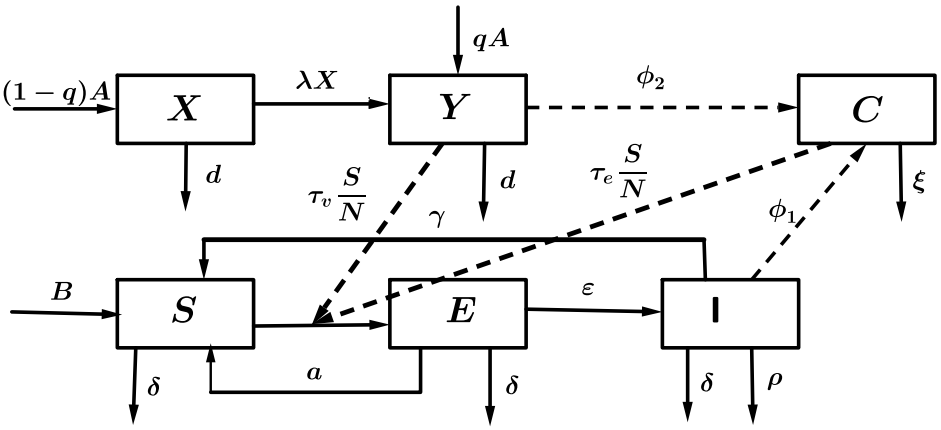

Figure 1. Flow diagraw for the transmission of avian influenza A; $\lambda = \beta_v \frac{Y}{1+\alpha Y} + \beta_e \frac{C}{C+\kappa}$.

Thus, to get better insight into the transmission dynamics of AIV and its spillover event to human, there is a strong need to couple equation (3) (for the production of viruses) with dynamical model in poultry and/or human (for the transmission of AIV in poultry and/or human). Therefore the whole system of ODE governing the AIV transmission becomes

$$\begin{cases} \dfrac{dX}{dt} = (1-q)A - \beta_v X \dfrac{Y}{1+\alpha Y} - \beta_e X \dfrac{C}{C+\kappa} - dX, \\ \dfrac{dY}{dt} = qA + \beta_v X \dfrac{Y}{1+\alpha Y} + \beta_e X \dfrac{C}{C+\kappa} - dY, \\ \dfrac{dS}{dt} = B + aE + \gamma I - \tau_v \dfrac{S}{N} Y - \tau_e \dfrac{S}{N} C - \delta S, \\ \dfrac{dE}{dt} = \tau_v \dfrac{S}{N} Y + \tau_e \dfrac{S}{N} C - (a + \delta + \epsilon)E, \\ \dfrac{dI}{dt} = \epsilon E - (\gamma + \rho + \delta)I, \\ \dfrac{dC}{dt} = \phi_2 Y - \xi C. \end{cases} \tag{7}$$

Here $N(t) = S(t) + E(t) + I(t)$ and $M(t) = X(t) + Y(t)$, are the total population of humans and poultry, respectively.

2.2. *Basic properties*

2.2.1. *Model well-posedness*

Since model (7) describes the evolution of a concentration of AIV in poultry farms, the individuals numbers should remain non-negative and bounded. The Cauchy problem associated

to system (7) is

$$\begin{cases} \dfrac{dw}{dt} = F(w(t)), & t \geq 0, \\ w(0) = w^0, \end{cases} \tag{8}$$

where $w(t) = (X(t), Y(t), S(t), E(t), I(t), C(t))$, $w^0 = (X^0, Y^0, S^0, E^0, I^0, C^0)$ and $F(w(t)) \in \mathbb{R}^6$ is the right-hand vector in equation (7).

Clearly, the function $F(w(t))$ is continuously differentiable in $\mathbb{R}^6$. Thus by Cauchy-Lipschitz theorem, for any $w^0 \in \mathbb{R}^6$ there exists an interval $[-\varsigma, \varsigma]$ and a unique continuously differentiable function $w(t) \in \mathbb{R}^6$ defined for $t \in [-\varsigma, \varsigma]$ and satisfying (8). Thus the existence of a unique maximal solution for (8) is guaranteed.

Theorem 2.1. *Let the initial data be $X^0 > 0, Y^0 > 0, S^0 > 0, E^0 > 0, I^0 > 0$ and $C^0 > 0$. Then, the corresponding solution $(X; Y; S; E; I; C)$ of model (7) is non-negative for all $t > 0$, whenever it exists. Moreover, the solutions are bounded and model (7) is a dynamical system in the set*

$$\Omega = \left\{ (X, Y, S, E, I, C) \in \mathbb{R}^6_+ / X + Y \leq \frac{A}{d} \; ; \; S + E + I \leq \frac{B}{\delta} \; ; \; C \leq \frac{\phi_2 A}{d\xi} \right\}.$$

Proof. Theorem 2.1 is not difficult to prove and the details are skipped. □

Since according to Theorem 2.1, the solutions of system (7) remain non-negative and bounded in $\mathbb{R}^6_+$, We can conclude that the solution exists, globally in time. Moreover, the set Ω is positively invariant with respect to the flow of system (7). Thus, system (7) is mathematically and epidemiologically well-posed and it is sufficient to consider the dynamics of the flow generated by system (7) in Ω, as it is also an attracting and absorbing set.

2.3. *Sensitivity analysis of the basic reproduction number*

2.3.1. *Computation of the basic reproduction number $\mathcal{R}_0$*

In the absence of infection, that is $q = 0$ and $Y = E = I = C = 0$, the model (7) has a disease-free equilibrium (DFE),

$$Z^0 = (X^0, Y^0, S^0, E^0, I^0, C^0) = \left(\frac{A}{d}, 0, \frac{B}{\delta}, 0, 0, 0 \right),$$

which is obtained by setting the right-hand side of the system (7) to zero.

A key quantity in classic epidemiological models is the basic reproduction number, denoted by $\mathcal{R}_0$. It is a useful threshold in the study of a disease for predicting a disease outbreak and for evaluating the control strategies. Usually, $\rho(M)$ will indicate the spectral radius of matrix M. We stress that, (Y, E, I, C) and (X, S) are the infected and uninfected classes, respectively. In order to compute the basic reproduction number $\mathcal{R}_0$, we follow the method in [21]. By so doing we get,

$$\mathcal{R}_0 = \frac{\beta_v A}{d^2} + \frac{\beta_e A \phi_2}{\kappa \xi d^2}. \tag{9}$$

We notice that if the farms environment are free of viruses, that is $\beta_e = 0$, the basic reduces to the sole contribution of direct poultry-to-poultry reproduction number

$$\mathcal{R}_v = \frac{\beta_v A}{d^2}.$$

On the oher hand, if direct transmission (poultry-to-poultry) is absent, that is $\beta_v = 0$ then only the infected poultry farms contribute to build the basic reproduction number which becomes

$$\mathcal{R}_e = \frac{\beta_e A \phi_2}{\kappa \xi d^2}.$$

These two observations suggest that the two modes of avian influenza transmission can independently or jointly trigger an epidemic depending on conditions. Thus, precisely speaking, $\mathcal{R}_0$ measures the number of secondary avian influenza infections generated in a wholly susceptible community when a sufficient concentration of vibrios contaminates the farms environment and/or when an avian influenza-infected individual is introduced into the farm.

In $\mathcal{R}_v$, β_v/d is the average amount of hyper infectious V. avian influenza ingested by a asymptomatic poultry.

In $\mathcal{R}_e$, $1/\xi$ is the lifetime of the vibrios in the farms environment; β_e/κ is the number of new cases generated in terms of vibrios per unit time, measured by the eID_{50} concentration; ϕ_2/d is the average amount of V. avian influenza shed per infected poultry.

2.3.2. *Local sensitivity analysis of* $\mathcal{R}_0$

The local sensitivity analysis is based on the normalized sensitivity index of $\mathcal{R}_0$. The normalized forward sensitivity index of a variable to a parameter is the number of the relative change in the variable to the relative change in the parameter. Since the basic reproduction number is a differentiable function of the parameters, the sensitivity index may alternatively be defined using partial derivatives [22]. To this aim, denoting by Φ the generic parameter of system (7), we evaluate the normalized sensitivity index

$$S_\Phi = \frac{\Phi}{\mathcal{R}_0} \frac{\partial \mathcal{R}_0}{\partial \Phi},$$

which indicates how sensitive $\mathcal{R}_0$ is to a change of parameter Φ. A positive (resp. negative) index indicates that an increase in the parameter value results in an increase (resp. decrease) in the $\mathcal{R}_0$ value.

Consider $\phi_2 = 10^3$ and the other parameter values in Table 3, we tabulate the indexes of the remaining parameters in Table 1. From Table 1, we can observe that the parameters β_v,

Table 1. Sensitivity indexes for $\mathcal{R}_0$.

Parameter	Sensitivity index	Value
β_v	S_{β_v}	+0.9676
β_e	S_{β_e}	+0.03226
A	S_A	+0.99999
ϕ_2	S_{ϕ_2}	+0.03226
ξ	S_ξ	-0.03226
d	S_d	-2.00003

β_e, A and ϕ_2 respectively have a positive influence in the value of $\mathcal{R}_0$. This means that the increase or the decrease of these parameters, will increase or decrease $\mathcal{R}_0$. The indexes for parameters ξ and d which represent the degradation rate of virus and natural death rate of poultry respectively, show that increasing their values, will decrease the value of $\mathcal{R}_0$. From these analyses, it is worth remakable that a higher emission rate of poultry ϕ_2 and the lower degradation rate of virus ξ increases $\mathcal{R}_0$. Using the parameter values in Table 3, the numerical results displayed in Figure 2 illustrate the role of ϕ_2 and ξ on the basic reproduction number $\mathcal{R}_0$, from which we observe that $\mathcal{R}_0$ increases whenever the parameters ϕ_2 and ξ increase and decrease respectively. This suggests that, an optimal control measure could be the combination of the number of emission rate of poultry and degradation rate of virus.

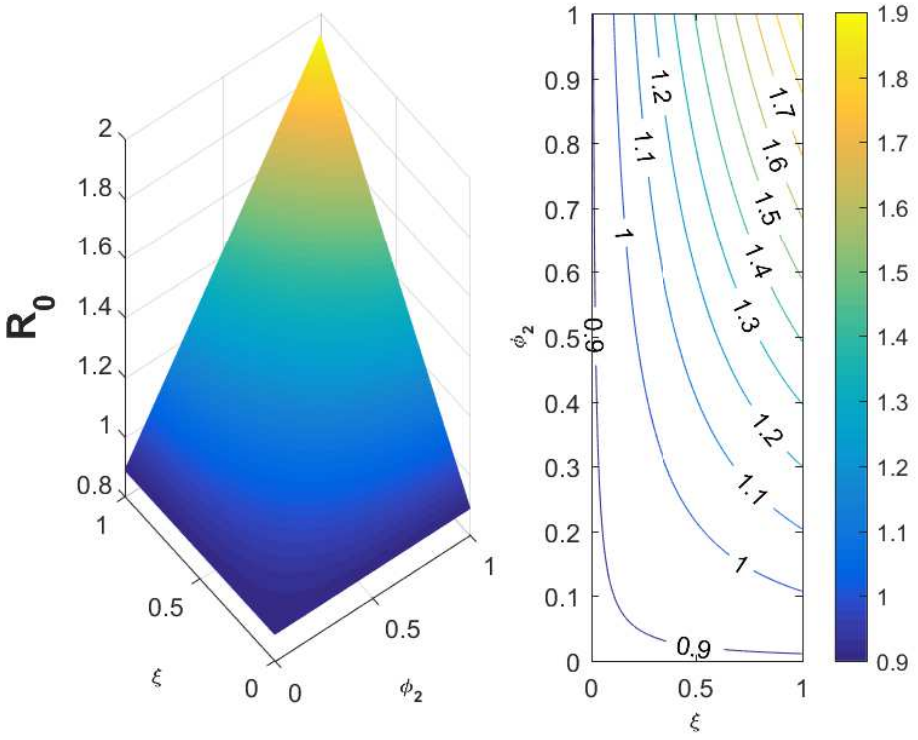

Figure 2. The basic reproduction number $\mathcal{R}_0$ plotted as function of the emission rate of poultry ϕ_2 and inactivation rate ξ.

3. Theoretical and asymptotic analysis of the model

In this section, we study the existence and asymptotic behavior of the steady state of model (7) in two different scenarios. We start with the analysis of the sub-model where there is no immigration of infected poultry, and secondly, we study the full model (7).

3.1. Analysis of the sub-model without imported infected poultry (q=0)

In this case, we get two equilibrium points, namely the disease-free equilibrium and the endemic equilibrium. When $q = 0$, the model (7) reads

$$\begin{cases} \dfrac{dX}{dt} = A - \beta_v X\dfrac{Y}{1+\alpha Y} - \beta_e X\dfrac{C}{C+\kappa} - dX, \\ \dfrac{dY}{dt} = \beta_v X\dfrac{Y}{1+\alpha Y} + \beta_e X\dfrac{C}{C+\kappa} - dY, \\ \dfrac{dS}{dt} = B + aE + \gamma I - \tau_v\dfrac{S}{N}Y - \tau_e\dfrac{S}{N}C - \delta S, \\ \dfrac{dE}{dt} = \tau_v\dfrac{S}{N}Y + \tau_e\dfrac{S}{N}C - (a+\delta+\epsilon)E, \\ \dfrac{dI}{dt} = \epsilon E - (\gamma+\rho+\delta)I, \\ \dfrac{dC}{dt} = \phi_2 Y - \xi C. \end{cases} \tag{10}$$

3.1.1. Existence of equilibrium points

The disease-free equilibrium is given by:

$$Z^0 = (X^0, Y^0, S^0, E^0, I^0, C^0) = \left(\frac{A}{d}, 0, \frac{B}{\delta}, 0, 0, 0\right).$$

Let $Z^* = (X^*, Y^*, S^*, E^*, I^*, C^*)$ be an endemic steady state of model (10). Then, as $N(t) = S(t) + E(t) + I(t)$ and $M(t) = X(t) + Y(t)$, it is easily seen that $dN/dt = B - \rho I - \delta N$ and for the poultry vector population the corresponding total population size is asymptotically constant: $\lim_{t\to+\infty} M(t) = A/d$. Based on this, we have

$$S^* = N^* - E^* - I^*, \quad I^* = \frac{B}{\rho} - \frac{\delta}{\rho}N^*, \quad E^* = \frac{B\eta_2}{\rho\epsilon} - \frac{\eta_2\delta}{\rho\epsilon}N^*, \quad X^* = \frac{A}{d} - Y^*; \quad C^* = \frac{\phi_2}{\xi}Y^*, \tag{11a}$$

$$Y^* = \frac{\alpha_1 N^*\left(N^* - \frac{B}{\delta}\right)}{\alpha_2 - \alpha_3 N^*}, \tag{11b}$$

where,

$$\eta_1 = a + \delta + \epsilon\ ,\ \eta_2 = \gamma + \delta + \rho,$$

$$\alpha_1 = \frac{\eta_1\eta_2\delta}{\rho\epsilon},\ \alpha_2 = \frac{B}{\rho}\left(\frac{\eta_2}{\epsilon} + 1\right)\left(\tau_v + \tau_e\frac{\phi_2}{\xi}\right),\ \alpha_3 = \left(\frac{\eta_2\delta}{\rho\epsilon} + \frac{\delta}{\rho} + 1\right)\left(\tau_v + \tau_e\frac{\phi_2}{\xi}\right). \tag{12}$$

Straightforward substitutions show that Y^* must satisfy the following equation:

$$P(Y^*) = \alpha_4 {Y^*}^2 + \alpha_5 Y^* + \alpha_6 = 0, \tag{13}$$

with

$$\alpha_4 = -\frac{\beta_v\phi_2}{\xi} - \frac{\beta_e\alpha\phi_2}{\xi} - \frac{d\alpha\phi_2}{\xi}, \tag{14a}$$

$$\alpha_5 = -\kappa\beta_v - \frac{\beta_e\phi_2}{\xi} - \left(d\alpha\kappa + \frac{d\phi_2}{\xi}\right)(1 - \mathcal{R}_0) - \frac{\alpha\kappa\beta_v A}{d} - \frac{\beta_e A\phi_2^2}{\kappa d\xi^2}, \tag{14b}$$

$$\alpha_6 = \kappa d(\mathcal{R}_0 - 1). \tag{14c}$$

Equation (13) has a unique positive solution if $\mathcal{R}_0 > 1$ and no positive solution whenever $\mathcal{R}_0 \leq 1$.

Substituting this solution by its value in (11b), we obtain

$$h(N^*) = \alpha_1 {N^*}^2 + \left(\alpha_3 Y^* - \alpha_1\frac{B}{\delta}\right)N^* - \alpha_2 Y^* = 0. \tag{15}$$

Notice that it is not difficult to show that $h(0) < 0$ and $h(B/\delta) > 0$. Indeed,

$$h(0) \quad = \quad -\alpha_2 Y^* < 0,$$

$$h\left(\frac{B}{\delta}\right) = \frac{\alpha_1 B^2}{\delta^2} + \frac{\alpha_3 B}{\delta} Y^* - \frac{\alpha_1 B^2}{\delta^2} - \alpha_2 Y^* = \frac{Y^* B}{\delta}\left(\tau_v + \tau_e \frac{\phi_2}{\xi}\right) > 0.$$

By the intermediate value theorem, there exists a positive root for the quadratic equation (15) which lies between 0 and B/δ. Moreover, since α_1 is positive and the constant coefficient of $h(N^*)$ is negative, the second root of equation (15) is negative. Thus, the existence of a unique endemic equilibrium point for model (10). Furthermore, we need to prove that the corresponding unique endemic equilibrium lies in Ω. To that end, let $P^*(Y^*) = \alpha_4 Y^{*2} + \alpha_5 Y^* + \alpha_6$. It is straightforward that $P^*(0) = \alpha_6 > 0$ and

$$\begin{aligned} P^*\left(\frac{A}{d}\right) &= -\frac{\beta_v \phi_2 A^2}{d^2 \xi} - \frac{\beta_e \alpha A^2 \phi_2}{d^2 \xi} - \frac{\alpha \phi_2 A^2}{d\xi} - \frac{\kappa \beta_v A}{d} - \alpha \kappa A + \frac{\beta_v \phi_2 A^2}{d^2 \xi} \\ &\quad + \frac{\beta_e \alpha \phi_2 A^2}{d^2 \xi} - \frac{\beta_e \phi_2 A}{d\xi} - \frac{A\phi_2}{\xi} + \frac{\kappa \beta_v A}{d} - d\kappa + \frac{\beta_e A \phi_2}{d\xi}, \\ &= -\frac{\alpha \phi_2 A^2}{d\xi} - \frac{A\phi_2}{\xi} - \kappa \alpha A - d\kappa < 0. \end{aligned}$$

Therefore, $0 < Y^* < A/d$, and it follows that C^* and X^* are positive. Finally, the fact that the unique endemic equilibrium $(X^*, Y^*, S^*, E^*, I^*, C^*)$ belongs to Ω is a direct consequence of the formulas in (11a) and (11b). These investigations are summarized in the following result.

Lemma 3.1. *The model (10) has:*

(1) a unique endemic equilibrium whenever $\mathcal{R}_0 > 1$;
(2) no endemic equilibrium whenever $\mathcal{R}_0 \leq 1$.

3.1.2. Local stability the disease-free equilibrium and bifurcation analysis at $\mathcal{R}_0 = 1$

Theorem 3.2. *The disease-free equilibrium Z^0 of system (10) is locally asymptotically stable in Ω when $\mathcal{R}_0 < 1$, but unstable when $\mathcal{R}_0 > 1$.*

Proof. The eigenvalues of a Jacobian matrix of the vector field described by (10) at the DFE, are the roots λ_1 and λ_2 of the quadratic equation

$$\lambda^2 + \lambda\left(\xi + d - \frac{\beta_v A}{d}\right) + d\xi - \frac{\beta_v A \xi}{d} - \frac{\beta_e A \phi_2}{\kappa d} = 0. \tag{16}$$

These roots satisfy the relations

$$\begin{aligned} \lambda_1 + \lambda_2 &= -\xi - d + \frac{\beta_v A}{d} = -\xi - \frac{\beta_e A \phi_2}{\kappa d^2 \xi} + d(\mathcal{R}_0 - 1), \\ \lambda_1 \times \lambda_2 &= d\xi - \frac{\beta_v A \xi}{d} - \frac{\beta_e A \phi_2}{\kappa d} = d\xi(1 - \mathcal{R}_0). \end{aligned}$$

Clearly, If $\mathcal{R}_0 < 1$, then $\lambda_1 \times \lambda_2 > 0$ and $\lambda_1 + \lambda_2 < 0$, such the λ_1 and λ_2 have negative real parts. This proves the local asymptotic stability of Z^0. On the other hand, if $\mathcal{R}_0 > 1$, at least one of the eigeinvalues has a positive real part, which implies that Z^0 is unstable. □

Biologically, Theorem 3.2 shows that the avian influenza can be eliminated (when $\mathcal{R}_0 < 1$) if the initial population lies in the basin of attraction of DFE Z^0.

Lemma 3.1 and Theorem 3.2 establish that $\mathcal{R}_0 = 1$ is a bifurcation parameter. In fact, across $\mathcal{R}_0 = 1$ the disease-free equilibrium, Z^0 changes its stability property from local stability to unstable (see Theorem 3.5). In the next result, the Center Manifold Theory [23] as described by Theorem 4.1 in [23] is used to investigate the appearance of the trans-critical bifurcation at $\mathcal{R}_0 = 1$ where the stable disease-free equilibrium Z^0 becomes unstable when $\mathcal{R}_0$ crosses

1 from below and gives rise to a stable endemic equilibrium Z^*. We have the following theorem.

Theorem 3.3. *The system (10) presents a trans-critical forward bifurcation at* $\mathcal{R}_0 = 1$.

Proof. To apply this theory, we first rename the state variables. Let $x_1 = X, x_2 = Y, x_3 = S, x_4 = E, x_5 = I$ and $x_6 = C$ so that $N = x_3 + x_4 + x_5$. Further, by using the vector notation $x = (x_1, x_2, x_3, x_4, x_5, x_6)^T$, the avian influenza model (10) can be written in the form $dx/dt = f(x)$, with $f = (f_1, f_2, f_3, f_4, f_5, f_6)^T$ as follows:

$$\begin{cases} \frac{dx_1}{dt} = f_1 = A - \beta_v x_1 \frac{x_2}{1+\alpha x_2} - \beta_e x_1 \frac{x_6}{x_6+\kappa} - dx_1, \\ \frac{dx_2}{dt} = f_2 = \beta_v x_1 \frac{x_2}{1+\alpha x_2} + \beta_e x_1 \frac{x_6}{x_6+\kappa} - dx_2, \\ \frac{dx_3}{dt} = f_3 = B + ax_4 + \gamma x_5 - (\tau_v x_2 + \tau_e x_6)\frac{x_3}{x_3+x_4+x_5} - \delta x_3, \\ \frac{dx_4}{dt} = f_4 = (\tau_v x_2 + \tau_e x_6)\frac{x_3}{x_3+x_4+x_5} - (a+\delta+\epsilon)x_4, \\ \frac{dx_5}{dt} = f_5 = \epsilon x_4 - (\gamma+\rho+\delta)x_5, \\ \frac{dx_6}{dt} = f_6 = \phi_2 x_2 - \xi x_6. \end{cases} \tag{17}$$

The Jacobian of system (17) at the DFE $Z^0 = (A/d, 0, B/\delta, 0, 0, 0)$, is the same as for the one in proof of Theorem 3.2. The basic reproduction number of the transformed (linearized) system (17) is the same as that of the original model (10).
Let σ_e be the non-negative real numbers such that $\beta_e = \sigma_e \beta_v$, then the basic reproduction number $\mathcal{R}_0$ becomes

$$\mathcal{R}_0 = \frac{\beta_e \kappa \xi A + \beta_e \phi_2 \sigma_e A}{\kappa \sigma_e \xi d^2}.$$

Therefore, choosing β_e as the bifurcation parameter, by solving for β_e when $\mathcal{R}_0 = 1$, we obtain:

$$\beta_e = \beta_e^* = \frac{\kappa \sigma_e \xi d^2}{A(\kappa\xi + \phi_2 \sigma_e)}.$$

It follows that the Jacobian ($J|_{Z^0}$) of system (17) at the DFE Z^0, with $\beta_e = \beta_e^*$, denoted by $J|_{\beta_e^*}$ has a simple zero eigenvalue (with all other eigenvalues having negative real parts). Hence, the Center Manifold theory [23] can be used to analyze the dynamics of system (17). In particular, Theorem 4.1 in [23], will be used to show that, when $\mathcal{R}_0 > 1$, there exists a unique endemic equilibrium of system (17) (as shown in Lemma 3.1) which is locally asymptotically stable for $\mathcal{R}_0$ near 1, under certain conditions. The application of Theorem 4.1 in [23] hinges on the following computations (it should be noted that we are using β_e^* as the bifurcation parameter, in place of ϕ in Theorem 4.1 [23]).

Eigenvectors of $J|_{\beta_e^*}$: The right eigenvector corresponding to the zero eigenvalue is:

$$u = (u_1, u_2, u_3, u_4, u_5, u_6)^T.$$

By solving the system

$$\begin{cases} -du_1 - \frac{\beta_v A}{d}u_2 - \frac{\beta_e A}{\kappa d}u_6 = 0; \quad \left(\frac{\beta_v A}{d} - d\right)u_2 + \frac{\beta_e A}{\kappa d}u_6 = 0; \quad \phi_2 u_2 - \xi u_6 = 0; \\ -\tau_v u_2 - \delta u_3 + a u_4 + \gamma u_5 - \tau_e u_6 = 0; \quad \tau_v u_2 - \eta_1 u_4 + \tau_e u_6 = 0; \quad \epsilon u_4 - \eta_2 u_5 = 0, \end{cases}$$

we obtain

$$u_1 = -u_2\ ,\ u_2 = u_2 > 0\ ,\ u_3 = \left(\frac{a}{\delta\eta_1} + \frac{\gamma\epsilon}{\delta\eta_1\eta_2} - \frac{1}{\delta}\right)\left(\tau_v + \frac{\tau_e\phi_2}{\xi}\right)u_2,$$
$$u_4 = \frac{1}{\eta_1}\left(\tau_v + \frac{\tau_e\phi_2}{\xi}\right)u_2\ ,\ u_5 = \frac{\epsilon}{\eta_1\eta_2}\left(\tau_v + \frac{\tau_e\phi_2}{\xi}\right)u_2\ ,\ u_6 = \frac{\phi_2}{\xi}u_2.$$

Similarly, the components of the left eigenvectors (corresponding to the zero eigenvalue) $v = (v_1, v_2, v_3, v_4, v_5, v_6)$ is obtained by solving the system

$$\begin{cases} v_1 = 0,\ v_3 = 0; & -\frac{\beta_v A}{d}v_1 + \left(\frac{\beta_v A}{d} - d\right)v_2 - \tau_v v_3 + \tau_v v_4 + \phi_2 v_6 = 0; \quad av_3 - \eta_1 v_4 + \epsilon v_5 = 0; \\ \gamma v_3 - \eta_2 v_5 = 0; & -\frac{\beta_v A}{d}v_1 + \frac{\beta_e A}{\kappa d}v_2 - \tau_e v_3 + \tau_e v_4 - \xi v_6 = 0. \end{cases}$$

Hence,

$$v_1 = 0\ ,\ v_2 = \frac{\kappa d\xi}{\beta_e A}v_6\ ,\ v_3 = 0\ ,\ v_4 = 0\ ,\ v_5 = 0\ ,\ v_6 = v_6 > 0.$$

Computation of a: For system (17), the corresponding non-zero partial derivatives of f_i $(i = 1; 2; 3; 4; 5; 6)$ calculated at the disease-free equilibrium are given by:

$$\frac{\partial^2 f_2}{\partial x_2^2} = -2\alpha\frac{\beta_v A}{d}\ ;\ \frac{\partial^2 f_2}{\partial x_6^2} = -2\frac{\beta_e A}{\kappa^2 d}\ ;\ \frac{\partial^2 f_2}{\partial x_1 \partial x_2} = \beta_v\ ;\ \frac{\partial^2 f_2}{\partial x_1 \partial x_6} = \frac{\beta_e}{\kappa}.$$

Consequently, we calculate the associated bifurcation coefficient **a**.

$$\begin{aligned} \mathbf{a} &= \sum_{k,i,j=1}^{6} v_k u_i u_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(Z^0) = v_2\left(u_2^2\frac{\partial^2 f_2}{\partial x_2^2} + u_6^2\frac{\partial^2 f_2}{\partial x_6^2} + 2u_1u_2\frac{\partial^2 f_2}{\partial x_1\partial x_2} + 2u_1u_6\frac{\partial^2 f_2}{\partial x_1\partial x_6}\right), \\ &= \frac{\kappa d\xi}{\beta_e A}v_6u_2^2\left[-2\alpha\frac{\beta_v A}{d} - 2\frac{\beta_e\phi_2^2 A}{\kappa^2\xi^2 d} - \frac{\beta_e\phi_2}{\kappa\xi} - 2\beta_v\right] < 0. \end{aligned}$$

Computation of b: For system (17), the corresponding non-zero partial derivatives of f_i $(i = 1; 2; 3; 4; 5; 6)$ calculated at the disease-free equilibrium are given by:

$$\frac{\partial^2 f_1}{\partial x_6 \partial \beta_e^*} = -\frac{A}{\kappa d},\quad \frac{\partial^2 f_2}{\partial x_6 \partial \beta_e^*} = \frac{A}{\kappa d}.$$

We compute the associated bifurcation coefficient **b**.

$$\mathbf{b} = \sum_{k,i=1}^{6} v_k u_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_e^*}(Z^0) = v_2 u_6 \frac{A}{\kappa d} = \frac{A}{\kappa d}\frac{\phi_2}{\xi}\frac{\kappa d\xi}{\beta_e A}u_2v_6 = \frac{\phi_2}{\beta_e}u_2v_6 > 0.$$

Thus, the bifurcation coefficient **a** is always negative. Furthermore, the bifurcation coefficient **b** is always positive. Hence, it follows from Theorem 4.1 in [23], that model (17) does undergo the trans-critical forward bifurcation at $\mathcal{R}_0 = 1$. □

Remark 3.4. *The application of Theorem 4.1 in [23] which proves Theorem 3.3, also establishes the local asymptotic stability of the unique endemic equilibrium Z^*, but this result applies only for small values of $\mathcal{R}_0 > 1$. Nonetheless, for all values of $\mathcal{R}_0 > 1$, Z^* is LAS as well and the proof follows similar procedure as in the demonstration of Theorem (3.14).*

3.1.3. Global stability analysis of the equilibrium points

For a better control of the disease, the global asymptotic stability of the DFE is needed.

Theorem 3.5. *The disease-free equilibrium of system (10) is globally asymptotically stable (GAS) in* Ω *if* $\mathcal{R}_0 \leq 1$.

Proof. We use the Lyapunov function approach. Define

$$L(X, Y, S, E, I, C) = \left(\frac{1}{d} + \frac{\phi_2}{d\xi}\right) Y(t) + \frac{1}{\xi} C(t).$$

Then,

$$\begin{aligned}
\frac{dL}{dt} &= \left(\frac{1}{d} + \frac{\phi_2}{d\xi}\right)\frac{dY}{dt} + \frac{1}{\xi}\frac{dC}{dt}, \\
&= \left(\frac{1}{d} + \frac{\phi_2}{d\xi}\right)\left(\beta_v X \frac{Y}{1+\alpha Y} + \beta_e X \frac{C}{C+\kappa} - dY\right) + \frac{1}{\xi}(\phi_2 Y - \xi C), \\
&= \left(\frac{1}{d} + \frac{\phi_2}{d\xi}\right)\left(\beta_v X \frac{Y}{1+\alpha Y} + \beta_e X \frac{C}{C+\kappa}\right) - Y - C, \\
&= \left(\frac{\mathcal{R}_0}{\beta_v X^0} + \frac{\kappa \mathcal{R}_0}{\beta_e X^0} - \frac{\beta_e \phi_2}{\kappa \xi \beta_v d} - \frac{\kappa \beta_v}{\beta_e d}\right)\left(\beta_v X \frac{Y}{1+\alpha Y} + \beta_e X \frac{C}{C+\kappa}\right) - Y - C.
\end{aligned}$$

Direct calculations lead to

$$\begin{aligned}
\frac{dL}{dt} &\leq \left(\frac{\mathcal{R}_0}{\beta_v X^0} + \frac{\kappa \mathcal{R}_0}{\beta_e X^0} - \frac{\beta_e \phi_2}{\kappa \xi \beta_v d} - \frac{\kappa \beta_v}{\beta_e d}\right)\left(\beta_v X \frac{Y}{1+\alpha Y} + \beta_e X \frac{C}{C+\kappa}\right) + \frac{\beta_e \phi_2}{\kappa \xi d} X^0 Y \\
&\quad + \frac{\kappa \beta_v^2}{d\beta_e} X^0 Y + \frac{\beta_e^2 \phi_2}{\kappa^2 d \xi \beta_v} X^0 C + \frac{\beta_v}{d} X^0 C - \frac{\kappa \beta_v}{\beta_e} \mathcal{R}_0 Y - \frac{\beta_e}{\kappa \beta_v} \mathcal{R}_0 C - Y - C, \\
&\leq \frac{\mathcal{R}_0}{X^0}\frac{XY}{1+\alpha Y} + \frac{\kappa \beta_v \mathcal{R}_0}{\beta_e X^0}\frac{XY}{1+\alpha Y} - \frac{\beta_e \phi_2}{\kappa \xi d}\frac{XY}{1+\alpha Y} - \frac{\kappa \beta_v^2}{d\beta_e}\frac{XY}{1+\alpha Y} + \frac{\beta_e \mathcal{R}_0}{\beta_v X^0}\frac{XC}{C+\kappa} + \frac{\kappa \mathcal{R}_0}{X^0}\frac{XC}{C+\kappa} \\
&\quad - \frac{\beta_e^2 \phi_2}{\kappa d \xi \beta_v}\frac{XC}{C+\kappa} - \frac{\kappa \beta_v}{d}\frac{XC}{C+\kappa} + \frac{\beta_e \phi_2}{\kappa \xi d} X^0 Y + \frac{\kappa \beta_v^2}{d\beta_e} X^0 Y + \frac{\beta_e^2 \phi_2}{\kappa^2 d \xi \beta_v} X^0 C + \frac{\beta_v}{d} X^0 C - \frac{\kappa \beta_v}{\beta_e} \mathcal{R}_0 Y \\
&\quad - \frac{\beta_e}{\kappa \beta_v} \mathcal{R}_0 C - Y - C - \mathcal{R}_0 Y + \mathcal{R}_0 Y - \mathcal{R}_0 C + \mathcal{R}_0 C.
\end{aligned}$$

Some simple rearrangements yield,

$$\begin{aligned}
\frac{dL}{dt} &\leq (\mathcal{R}_0 - 1)(Y + C) - \frac{\mathcal{R}_0 Y}{X^0}\left(X^0 - \frac{X}{1+\alpha Y}\right) - \frac{\kappa \mathcal{R}_0 \beta_v}{\beta_e X^0} Y \left(X^0 - \frac{X}{1+\alpha Y}\right) \\
&\quad + \frac{\beta_e \phi_2}{\kappa \xi d} Y \left(X^0 - \frac{X}{1+\alpha Y}\right) + \frac{\kappa \beta_v^2}{d\beta_e} Y \left(X^0 - \frac{X}{1+\alpha Y}\right) - \frac{\beta_e \mathcal{R}_0}{\beta_v X^0} C \left(\frac{X^0}{\kappa} - \frac{X}{C+\kappa}\right) \\
&\quad - \frac{\kappa \mathcal{R}_0}{X^0} C \left(\frac{X^0}{\kappa} - \frac{X}{C+\kappa}\right) + \frac{\beta_e^2 \phi_2}{\kappa d \xi \beta_v} C \left(\frac{X^0}{\kappa} - \frac{X}{C+\kappa}\right) + \frac{\kappa \beta_v}{d}\left(\frac{X^0}{\kappa} - \frac{X}{C+\kappa}\right).
\end{aligned}$$

Finally,

$$\frac{dL}{dt} \leq (\mathcal{R}_0 - 1)(Y + C) - \frac{\kappa \xi \mathcal{R}_0}{(\beta_v + \beta_e \phi_2) X^0}\left(1 + \frac{\phi_2}{\xi}\right)\left[\beta_v Y \left(X^0 - \frac{X}{1+\alpha Y}\right) + \beta_e C \left(\frac{X^0}{\kappa} - \frac{X}{C+\kappa}\right)\right].$$

Since $X \leq X^0$, we have

$$\frac{dL}{dt} \leq (\mathcal{R}_0 - 1)(Y + C) - \frac{\kappa\xi\mathcal{R}_0}{(\beta_v + \beta_e\phi_2)X^0}\left(1 + \frac{\phi_2}{\xi}\right)\left[\beta_v Y(X^0 - X) + \beta_e C\left(\frac{X^0}{\kappa} - \frac{X}{\kappa}\right)\right] \leq 0,$$

whenever $\mathcal{R}_0 \leq 1$. Moreover, $\frac{dL}{dt} = 0, \Leftrightarrow Y = C = 0$ or $X = X^0$ and $\mathcal{R}_0 = 1$.

Thus, the largest invariant set $\mathcal{H}$ such that $\mathcal{H} \subset \left\{(X, Y, S, E, I, C) \in \mathbb{R}^6_+ / dL/dt = 0\right\}$ is $\{Z^0\}$ because in $\mathcal{H}$ one has $\lim_{t\to+\infty} Y(t) = \lim_{t\to+\infty} C(t) = 0$. In system (10), we obtain $\lim_{t\to+\infty} X(t) = X^0, \lim_{t\to+\infty} S(t) = S^0, \lim_{t\to+\infty} E(t) = \lim_{t\to+\infty} I(t) = 0$. By LaSalle's Invariance Principle [24], $\{Z^0\}$ is globally asymptotically stable in Ω. The proof is complete. □

As for the proof of the GAS of the endemic equilibrium Z^*, one should notice that, since the poultry sub-model is independent of the human population variables (S, E, I), system (10) takes the triangular form

$$\begin{cases} \dfrac{dx}{dt} = f(x), \quad x = (X, Y, C), \\ \dfrac{dy}{dt} = g(x, y), \quad y = (S, E, I). \end{cases} \tag{18}$$

Therefore, in order to deal with the global asymptotic stability of the unique endemic equilibrium stated in Theorem 3.9, the following three results are instrumental.

Theorem 3.6. *(Vidyagasar [25])*
Consider a C^1 class system with an equilibrium point $(x^*; y^*)$.

$$\begin{cases} \dfrac{dx}{dt} = f(x), \\ \dfrac{dy}{dt} = g(x, y), \qquad x \in \mathbb{R}^n, y \in \mathbb{R}^m, \\ f(x^*) = 0, g(x^*, y^*) = 0. \end{cases} \tag{19}$$

If x^* is GAS in $\mathbb{R}^n$ for system $dx/dt = f(x)$, and if y^* is GAS in $\mathbb{R}^m$, for system $dy/dt = g(x^*; y)$, then equilibrium point $(x^*; y^*)$ is (locally) asymptotically stable for system (19). Moreover, if all the trajectories of (19) are positively bounded , then $(x^*; y^*)$ is GAS for (19).

Theorem 3.7. *Let H be a 2×2 matrix [26, 27]. Then*

$$H = \begin{bmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{bmatrix},$$

is Volterra-Lyapunov stable if and only if $a_{11} < 0, a_{22} < 0$, and $a_{11}a_{22} - a_{12}a_{21} > 0$.

Theorem 3.8. *Let H be a non-singular $n \times n$ matrix, where $n \geq 2$, with inverse $H^{-1} = K$ and W a positive diagonal $n \times n$ matrix [28]. Let H^*, K^*, and W^* denote the $(n-1) \times (n-1)$ matrices obtained from H, K, and W, respectively, by deleting the last row and the last column. Then*

(i) if $WH + (WH)^T > 0$, we must have $a_{nn} > 0$, $W^*H^* + (W^*H^*)^T > 0$, and $W^*K^* + (W^*K^*)^T > 0$;
(ii) if $a_{nn} > 0, W^*H^* + (W^*H^*)^T > 0$, and $W^*K^* + (W^*K^*)^T > 0$, it is possible to choose $w_n > 0$ such that $WH + (WH)^T > 0$.

Now, we claim the following result.

Theorem 3.9. *The unique positive endemic equilibrium point $x^* = (X^*, Y^*, C^*)$ of the system (20) is globally asymptotically stable if $\mathcal{R}_0 > 1$.*

Proof. Following, Theorem 3.6, we first study the GAS of the endemic equilibrium x^* of the poultry system:

$$\frac{dx}{dt} = f(x) \equiv \begin{cases} \dfrac{dX}{dt} = A - \beta_v X\dfrac{Y}{1+\alpha Y} - \beta_e X\dfrac{C}{C+\kappa} - dX, \\ \dfrac{dY}{dt} = \beta_v X\dfrac{Y}{1+\alpha Y} + \beta_e X\dfrac{C}{C+\kappa} - dY, \\ \dfrac{dC}{dt} = \phi_2 Y - \xi C. \end{cases} \tag{20}$$

Consider the following domain as a result of a nondimensionalized system (20)

$$\Omega_1 = \left\{(X, Y, C) \in \mathbb{R}^3_+ / 0 < X + Y \leq \frac{A}{d}, C \leq \frac{\phi_2 A}{d\xi}\right\}.$$

Next, construct the Lyapunov function

$$V = \omega_1(X - X^*)^2 + \omega_2(Y - Y^*)^2 + \omega_3(C - C^*)^2, \tag{21}$$

with $\omega_1 > 0, \omega_2 > 0$ and $\omega_3 > 0$. Note that for the endemic equilibrium x^* , we have the following three equations for the nondimensionalized system:

$$A - \beta_v X^*\frac{Y^*}{1+\alpha Y^*} - \beta_e X^*\frac{C^*}{C^*+\kappa} - dX^* = 0, \tag{22a}$$

$$\beta_v X^*\frac{Y^*}{1+\alpha Y^*} + \beta_e X^*\frac{C^*}{C^*+\kappa} - dY^* = 0, \tag{22b}$$

$$\phi_2 Y^* - \xi C^* = 0. \tag{22c}$$

Using (22a)-(22c), we obtain

$$\begin{aligned} \frac{dV}{dt} = {} & 2\omega_1(X - X^*)\left[-\beta_v X\frac{Y}{1+\alpha Y} - \beta_e X\frac{C}{C+\kappa} - dX + \beta_v X^*\frac{Y^*}{1+\alpha Y^*} + \beta_e X^*\frac{C^*}{C^*+\kappa} + dX^*\right] \\ & +2\omega_2(Y - Y^*)\left[\beta_v X\frac{Y}{1+\alpha Y} + \beta_e X\frac{C}{C+\kappa} - dY - \beta_v X^*\frac{Y^*}{1+\alpha Y^*} - \beta_e X^*\frac{C^*}{C^*+\kappa} + dY^*\right] \\ & +2\omega_3(C - C^*)\left[\phi_2 Y - \xi C - \phi_2 Y^* + \xi C^*\right]. \end{aligned}$$

Some substitutions yield

$$\begin{aligned} \frac{dV}{dt} = {} & 2\omega_1(X - X^*)\left[-\left(\beta_v X\frac{Y}{1+\alpha Y} - \beta_v X^*\frac{Y}{1+\alpha Y} + \beta_v X^*\frac{Y}{1+\alpha Y} - \beta_v X^*\frac{Y^*}{1+\alpha Y^*}\right)\right. \\ & \left.-\left(\beta_e X\frac{C}{C+\kappa} - \beta_e X^*\frac{C}{C+\kappa} + \beta_e X^*\frac{C}{C+\kappa} - \beta_e X^*\frac{C^*}{C^*+\kappa}\right)\right] - 2\omega_1 d(X - X^*)^2 \\ & +2\omega_2(Y - Y^*)\left[\left(\beta_v X\frac{Y}{1+\alpha Y} - \beta_v X^*\frac{Y}{1+\alpha Y} + \beta_v X^*\frac{Y}{1+\alpha Y} - \beta_v X^*\frac{Y^*}{1+\alpha Y^*}\right)\right. \\ & \left.+\left(\beta_e X\frac{C}{C+\kappa} - \beta_e X^*\frac{C}{C+\kappa} + \beta_e X^*\frac{C}{C+\kappa} - \beta_e X^*\frac{C^*}{C^*+\kappa}\right)\right] - 2\omega_2 d(Y - Y^*)^2 \\ & +2\omega_3(C - C^*)\left[\phi_2(Y - Y^*) - \xi(C - C^*)\right]. \end{aligned}$$

The gatherings of some terms give

$$\begin{aligned}\frac{dV}{dt} &= -2\omega_1\Big(\beta_v\frac{Y}{1+\alpha Y}+\beta_e\frac{C}{C+\kappa}+d\Big)(X-X^*)^2-2\omega_1\frac{\beta_v X^*}{(1+\alpha Y)(1+\alpha Y^*)}(X-X^*)(Y-Y^*)\\ &\quad -2\omega_1\frac{\kappa\beta_e X^*}{(C+\kappa)(C^*+\kappa)}(X-X^*)(C-C^*)+2\omega_2\Big(\frac{\beta_v X^*}{(1+\alpha Y)(1+\alpha Y^*)}-d\Big)(Y-Y^*)^2\\ &\quad +2\omega_2\Big(\beta_v\frac{Y}{1+\alpha Y}+\beta_e\frac{C}{C+\kappa}\Big)(Y-Y^*)(X-X^*)+2\omega_2\frac{\kappa\beta_e X^*}{(C+\kappa)(C^*+\kappa)}(Y-Y^*)(C-C^*)\\ &\quad +2\omega_3\phi_2(C-C^*)(Y-Y^*)-2\omega_3\xi(C-C^*)^2,\\ &= U(WH+H^TW)U^T,\end{aligned}$$

where $U=[X-X^*, Y-Y^*, C-C^*]$, $W=\mathrm{diag}(\omega_1,\omega_2,\omega_3)$ and

$$H=\begin{bmatrix} -\beta_v\frac{Y}{1+\alpha Y}-\beta_e\frac{C}{C+\kappa}-d & -\frac{\beta_v X^*}{(1+\alpha Y)(1+\alpha Y^*)} & -\frac{\kappa\beta_e X^*}{(C+\kappa)(C^*+\kappa)}\\ \beta_v\frac{Y}{1+\alpha Y}+\beta_e\frac{C}{C+\kappa} & \frac{\beta_v X^*}{(1+\alpha Y)(1+\alpha Y^*)}-d & \frac{\kappa\beta_e X^*}{(C+\kappa)(C^*+\kappa)}\\ 0 & \phi_2 & -\xi\end{bmatrix}. \tag{23}$$

The global asymptotic stability of x^* will be established if we can show that the matrix H defined in (23) is Volterra-Lyapunov stable [28]; that is, a positive diagonal matrix W exists such that $WH+H^TW$ is negative definite.
From (23), one can see that H is non-singular because

$$\begin{aligned}\det H &= \frac{d\kappa\beta_e\phi_2 X^*}{(C+\kappa)(C^*+\kappa)}+\frac{d\beta_v\xi X^*}{(1+\alpha Y)(1+\alpha Y^*)}-d\xi\Big(\beta_v\frac{Y}{1+\alpha Y}+\beta_e\frac{C}{C+\kappa}+d\Big),\\ &= \frac{d\kappa\beta_e\phi_2 X^*}{(C+\kappa)(C^*+\kappa)}+\frac{d\beta_v\xi X^*}{(1+\alpha Y)(1+\alpha Y^*)}\\ &\quad -\Big(\frac{\beta_v\xi X^*}{1+\alpha Y^*}+\frac{\beta_e\phi_2 X^*}{C^*+\kappa}\Big)\Big(\beta_v\frac{Y}{1+\alpha Y}+\beta_e\frac{C}{C+\kappa}+d\Big),\\ &= -\frac{d\beta_e\phi_2 X^*C}{(C^*+\kappa)(C+\kappa)}-\frac{d\beta_v\xi X^*Y}{(1+\alpha Y)(1+\alpha Y^*)}\\ &\quad -\Big(\frac{\beta_v\xi X^*}{1+\alpha Y^*}+\frac{\beta_e\phi_2 X^*}{C^*+\kappa}\Big)\Big(\beta_v\frac{Y}{1+\alpha Y}+\beta_e\frac{C}{C+\kappa}\Big)<0.\end{aligned}$$

Moreover,

$$H^{-1}=\frac{1}{\det H}\begin{bmatrix} h_{11} & h_{12} & -\frac{d\kappa\beta_e X^*}{(C+\kappa)(C^*+\kappa)}\\ h_{21} & h_{22} & \frac{d\kappa\beta_e X^*}{(C+\kappa)(C^*+\kappa)}\\ \frac{\beta_v\phi_2 Y}{1+\alpha Y}-+\frac{\beta_e\phi_2 C}{C+\kappa} & d\phi_2+\frac{\beta_v\phi_2 Y}{1+\alpha Y}+\frac{\beta_e\phi_2 C}{C+\kappa} & h_{33}\end{bmatrix},$$

where,

$$
\begin{aligned}
h_{11} &= d\xi - \frac{\beta_v \xi X^*}{(1+\alpha Y)(1+\alpha Y^*)} - \frac{\kappa \beta_e \phi_2 X^*}{(C+\kappa)(C^*+\kappa)}, \\
h_{12} &= -\frac{\beta_v \xi X^*}{(1+\alpha Y)(1+\alpha Y^*)} - \frac{\kappa \beta_e \phi_2 X^*}{(C+\kappa)(C^*+\kappa)}, \\
h_{21} &= \frac{\beta_v \xi Y}{1+\alpha Y} + \frac{\beta_e \xi C}{C+\kappa}, \\
h_{22} &= d\xi + \frac{\beta_v \xi Y}{1+\alpha Y} + \frac{\beta_e \xi C}{C+\kappa}, \\
h_{33} &= d\left(\frac{\beta_v Y}{1+\alpha Y} + \frac{\beta_e C}{C+\kappa}\right) + d^2 - \frac{d\beta_v X^*}{(1+\alpha Y)(1+\alpha Y^*)}.
\end{aligned}
$$

Using the fact that $\det H < 0$, and the relations that link the endemic equilibrium component, one can readily verify the hypotheses of Theorem 3.7 for the matrix $(H^{-1})^*$ and conclude that it is Volterra-Lyapunov stable. Hence, a 2×2 positive diagonal matrix $W^* = \mathrm{diag}(\omega_1, \omega_2)$ exists such that $W^*(H^{-1})^* + (W^*(H^{-1})^*)^T < 0$. Setting $O = (-H)^{-1}$, we have $W^*O^* + (W^*O^*)^T > 0$. After lenghty but direct calculations, we obtain

$$
(-\det H)[W^*O^* + (W^*O^*)^T] = \begin{bmatrix} a_{11} & a_{12} \\ a_{12} & a_{22} \end{bmatrix},
$$

with

$$
\begin{aligned}
a_{11} &= 2\omega_1\left(d\xi - \frac{\beta_v \xi X^*}{(1+\alpha Y)(1+\alpha Y^*)} - \frac{\kappa \beta_e \phi_2 X^*}{(C+\kappa)(C^*+\kappa)}\right), \\
a_{12} &= \omega_2\left(\beta_v \xi \frac{Y}{1+\alpha Y} + \beta_e \xi \frac{C}{C+\kappa}\right) - \omega_1\left(\frac{\beta_v \xi X^*}{(1+\alpha Y)(1+\alpha Y^*)} + \frac{\kappa \beta_e \phi_2 X^*}{(C+\kappa)(C^*+\kappa)}\right), \\
a_{22} &= 2\omega_2\left(d\xi + \beta_v \xi \frac{Y}{1+\alpha Y} + \beta_e \xi \frac{C}{C+\kappa}\right).
\end{aligned}
$$

On the other hand,

$$
W^*(-H)^* + (W^*(-H)^*)^T = \begin{bmatrix} b_{11} & b_{12} \\ b_{12} & b_{22} \end{bmatrix}, \quad \text{with}
$$

$$
\begin{aligned}
b_{11} &= 2\omega_1\left(d + \beta_v \frac{Y}{1+\alpha Y} + \beta_e \frac{C}{C+\kappa}\right), \\
b_{12} &= \omega_1 \frac{\beta_v X^*}{(1+\alpha Y)(1+\alpha Y^*)} - \omega_2\left(\beta_v \frac{Y}{1+\alpha Y} + \beta_e \frac{C}{C+\kappa}\right), \\
b_{22} &= 2\omega_2\left(d - \frac{\beta_v X^*}{(1+\alpha Y)(1+\alpha Y^*)}\right).
\end{aligned}
$$

Next, we prove that $W^*(-H)^* + (W^*(-H)^*)^T > 0$. Indeed, since $W^*O^* + (W^*O^*)^T$ is positive

definite and $-\det H > 0$, we have $\det\Big\{(-\det H)[W^*O^* + (W^*O^*)^T]\Big\} > 0$ and

$$\begin{aligned}
\det\Big\{W^*(-H)^* + (W^*(-H)^*)^T\Big\} &= 4\omega_1\omega_2\Big[d + \beta_v\frac{Y}{1+\alpha Y} + \beta_e\frac{C}{C+\kappa}\Big]\Big[d - \frac{\beta_v X^*}{(1+\alpha Y)(1+\alpha Y^*)}\Big] \\
&\quad -\omega_1^2\Big[\frac{\beta_v\xi X^*}{(1+\alpha Y)(1+\alpha Y^*)}\Big]^2 - \omega_2^2\Big[\beta_v\frac{Y}{1+\alpha Y} + \beta_e\frac{C}{C+\kappa}\Big]^2 \\
&\quad +2\omega_1\omega_2\frac{\beta_v\xi X^*}{(1+\alpha Y)(1+\alpha Y^*)}\Big[\beta_v\frac{Y}{1+\alpha Y} + \beta_e\frac{C}{C+\kappa}\Big], \\
&= \det\Big\{(-\det H)[W^*O^* + (W^*O^*)^T]\Big\} \\
&\quad +4\omega_1\omega_2\frac{d\xi\kappa\beta_e\phi_2 X^*}{(C+\kappa)(C^*+\kappa)} + \omega_1^2\Big[\frac{\kappa\beta_e\phi_2 X^*}{(C+\kappa)(C^*+\kappa)}\Big]^2 \\
&\quad +2\omega_1\omega_2\frac{\kappa\beta_e\phi_2 X^*}{(C+\kappa)(C^*+\kappa)}\Big[\beta_v\frac{Y}{1+\alpha Y} + \beta_e\frac{C}{C+\kappa}\Big] \\
&\quad +2\omega_1^2\frac{\beta_v\xi X^*}{(1+\alpha Y)(1+\alpha Y^*)}\frac{\kappa\beta_e\phi_2 X^*}{(C+\kappa)(C^*+\kappa)} > 0,
\end{aligned}$$

where,

$$\begin{aligned}
\det\Big\{(-\det H)[W^*O^* + (W^*O^*)^T]\Big\} &= 4\omega_1\omega_2\Big[d\xi + \beta_v\xi\frac{Y}{1+\alpha Y} + \beta_e\xi\frac{C}{C+\kappa}\Big] \\
&\quad \times\Big[d\xi - \frac{\beta_v\xi X^*}{(1+\alpha Y)(1+\alpha Y^*)} - \frac{\kappa\beta_e\phi_2 X^*}{(C+\kappa)(C^*+\kappa)}\Big] \\
&\quad -\omega_1^2\Big[\frac{\beta_v\xi X^*}{(1+\alpha Y)(1+\alpha Y^*)} + \frac{\kappa\beta_e\phi_2 X^*}{(C+\kappa)(C^*+\kappa)}\Big]^2 \\
&\quad -\omega_2^2\Big[\beta_v\xi\frac{Y}{1+\alpha Y} + \beta_e\xi\frac{C}{C+\kappa}\Big]^2 \\
&\quad +2\omega_1\omega_2\Big[\beta_v\xi\frac{Y}{1+\alpha Y} + \beta_e\xi\frac{C}{C+\kappa}\Big] \\
&\quad \times\Big[\frac{\beta_v\xi X^*}{(1+\alpha Y)(1+\alpha Y^*)} + \frac{\kappa\beta_e\phi_2 X^*}{(C+\kappa)(C^*+\kappa)}\Big].
\end{aligned}$$

A judicious exploitation of the equilibrium relations shows that

$$b_{22} = d - \frac{\beta_v X^*}{(1+\alpha Y)(1+\alpha Y^*)} > 0.$$

Hence, the matrix $W^*(-H)^* + (W^*(-H)^*)^T$ is positive definite. Since $b_{22} > 0$, $W^*(-H)^* + (W^*(-H)^*)^T > 0$ and $W^*(-H^{-1})^* + (W^*(-H^{-1})^*)^T > 0$, then thanks to Theorem 3.8 (*ii*), there exists $\omega_3 > 0$ such that $W(-H) + (W(-H))^T > 0$; that is, $WH + H^TW < 0$. Thus H is Volterra-Lyapunov stable. Hence a feasible equilibrium x^* is globally asymptotically stable in Ω_1. □

Next, we investigate the dynamics of the human sub-system:

$$\frac{dy}{dt} = g(x^*; y) \equiv \begin{cases} \dfrac{dS}{dt} = B + aE + \gamma I - \tau_v \dfrac{S}{N} Y^* - \tau_e \dfrac{S}{N} C^* - \delta S, \\ \dfrac{dE}{dt} = \tau_v \dfrac{S}{N} Y^* + \tau_e \dfrac{S}{N} C^* - (a + \delta + \epsilon)E, \\ \dfrac{dI}{dt} = \epsilon E - (\gamma + \rho + \delta)I. \end{cases} \tag{24}$$

Theorem 3.10. *The unique positive endemic equilibrium point $y^* = (S^*, E^*, I^*)$ of the system (24) is globally asymptotically stable if $\mathcal{R}_0 > 1$.*

Proof. We introduce the fractions $x = \delta S/B, y = \delta E/B, z = \delta I/B$ and scale time by introducing a new time $\tau = \delta t$. This gives us the simplified system as:

$$\begin{cases} \dfrac{dx}{d\tau} = 1 + \bar{a}y + \overline{\gamma}z - (\overline{\tau}_v Y^* + \overline{\tau}_e C^*)\dfrac{\delta}{B}\dfrac{x}{\overline{N}} - x, \\ \dfrac{dy}{d\tau} = (\overline{\tau}_v Y^* + \overline{\tau}_e C^*)\dfrac{\delta}{B}\dfrac{x}{\overline{N}} - (1 + \bar{a} + \bar{\epsilon})y, \\ \dfrac{dz}{d\tau} = \bar{\epsilon}y - (1 + \overline{\rho} + \overline{\gamma})z, \end{cases} \tag{25}$$

where

$$\bar{a} = \frac{a}{\delta}, \overline{\tau}_v = \frac{\tau_v}{\delta}, \overline{\tau}_e = \frac{\tau_e}{\delta}, \bar{\epsilon} = \frac{\epsilon}{\delta}, \overline{\rho} = \frac{\rho}{\delta}, \overline{\gamma} = \frac{\gamma}{\delta}, \ \overline{N} = x + y + z.$$

Here, we have used the fact that

$$\frac{d\overline{N}}{d\tau} = 1 - \overline{N} - \overline{\rho}z.$$

It can be shown that the region

$$\Omega_2 = \left\{(x, y, z) \in \mathbb{R}^3_+ / 0 \leq x + y + z \leq 1\right\},$$

is positively invariant. Now, consider the equivalent system:

$$\begin{cases} \dfrac{dy}{d\tau} = (\overline{\tau}_v Y^* + \overline{\tau}_e C^*)\dfrac{\delta}{B}\dfrac{x}{\overline{N}} - (1 + \bar{a} + \bar{\epsilon})y, \\ \dfrac{dz}{d\tau} = \bar{\epsilon}y - (1 + \overline{\rho} + \overline{\gamma})z, \\ \dfrac{d\overline{N}}{d\tau} = 1 - \overline{N} - \overline{\rho}z. \end{cases} \tag{26}$$

Denote

$$\Omega_3 = \left\{(y, z, \overline{N}) \in \Omega_2 / \overline{N} = 1 - \overline{\rho}z\right\} = \left\{(y, z, \overline{N}) \in \Omega_2 / x + y + (1 + \overline{\rho})z = 1\right\}.$$

Then it not difficult to prove that Ω_3 is a positively invariant and attracting subset of Ω_2. Next we use the Poincaré-Bendixson techniques to prove that system (24) has no periodic solution. Let us assume that the system (24) has a periodic solution $\psi(\tau) = \{x(\tau), y(\tau), z(\tau)\}$. Let $\psi(\tau)$ be the trajectory of periodic solution, and Π be the planar region of $\psi(\tau)$. Let

$f_1(x,y,z)$, $f_2(x,y,z)$ and $f_3(x,y,z)$ respectively represent the three expressions of the right-hand side of the system (25). Set $\mathbf{f} = (f_1, f_2, f_3)^T$, $\mathbf{g}(x,y,z) = \mathbf{r} \times \mathbf{f}/(xyz)$, where $\mathbf{r} = (x,y,x)^T$. Then $\mathbf{g} \cdot \mathbf{f} = 0$, let $\mathbf{g} = (g_1, g_2, g_3)$, where

$$g_1 = \frac{f_3(x,z)}{xz} - \frac{f_2(x,y)}{xy}\ ,\ g_2 = \frac{f_1(x,y)}{xy} - \frac{f_3(y,z)}{yz}\ ,\ g_3 = \frac{f_2(y,z)}{yz} - \frac{f_1(x,z)}{xz}.$$

Then

$$Curl\mathbf{g} = \left(\frac{\partial g_3}{\partial y} - \frac{\partial g_2}{\partial z}\ ,\quad \frac{\partial g_1}{\partial z} - \frac{\partial g_3}{\partial x}\ ,\quad \frac{\partial g_2}{\partial x} - \frac{\partial g_1}{\partial y}\right).$$

By simple calculations, we have

$$\begin{aligned}
\frac{f_1(x,y)}{xy} &= \frac{1}{xy} + \frac{\overline{a}}{x} + \frac{\overline{\gamma}(1-x-y)}{(1+\overline{\rho})xy} - (\overline{\tau}_v Y^* + \overline{\tau}_e C^*)\frac{\delta}{By}\frac{1+\overline{\rho}}{1+\overline{\rho}(x+y)} - \frac{1}{y},\\
\frac{f_1(x,z)}{xz} &= \frac{1}{xz} + \frac{\overline{a}[1-x-(1+\overline{\rho})z]}{xz} + \frac{\overline{\gamma}}{x} - (\overline{\tau}_v Y^* + \overline{\tau}_e C^*)\frac{\delta}{Bz}\frac{1}{1-\overline{\rho}z} - \frac{1}{z},\\
\frac{f_2(y,z)}{yz} &= (\overline{\tau}_v Y^* + \overline{\tau}_e C^*)\frac{\delta}{B}\frac{1-y-(1+\overline{\rho})z}{yz(1-\overline{\rho})z} - \frac{1+\overline{a}+\overline{\epsilon}}{z},\\
\frac{f_2(x,y)}{xy} &= (\overline{\tau}_v Y^* + \overline{\tau}_e C^*)\frac{\delta}{By}\frac{1+\overline{\rho}}{1+\overline{\rho}(x+y)} - \frac{1+\overline{a}+\overline{\epsilon}}{x},\\
\frac{f_3(y,z)}{yz} &= \frac{\overline{\epsilon}}{z} - \frac{1+\overline{\gamma}+\overline{\rho}}{y},\\
\frac{f_3(x,z)}{xz} &= \frac{\overline{\epsilon}[1-x-(1+\overline{\rho})z]}{xz} - \frac{1+\overline{\gamma}+\overline{\rho}}{x}.
\end{aligned}$$

Now, since $x + y + (1+\overline{\rho})z = 1$, it is clear that $-[1-(1+\overline{\rho})z] = -(x+y) < 0$, so that

$$\frac{\partial g_3}{\partial y} - \frac{\partial g_2}{\partial z} = -\frac{\overline{\epsilon}}{z^2} - (\overline{\tau}_v Y^* + \overline{\tau}_e C^*)\frac{\delta}{B}\frac{1-(1+\overline{\rho})z}{y^2 z(1-\overline{\rho}z)} < 0.$$

Further, we have

$$\begin{aligned}
\frac{\partial g_1}{\partial z} - \frac{\partial g_3}{\partial x} &= -\frac{\overline{\epsilon}(1-x)}{xz^2} - \frac{1}{x^2 z} - \frac{\overline{a}[1-(1+\overline{\rho})z]}{x^2 z} - \frac{\overline{\gamma}}{x^2},\\
\frac{\partial g_2}{\partial x} - \frac{\partial g_1}{\partial y} &= -\frac{1}{x^2 y} - \frac{\overline{a}}{x^2} - \frac{\overline{\gamma}(1-y)}{(1+\overline{\rho})x^2 y} - (\overline{\tau}_v Y^* + \overline{\tau}_e C^*)\frac{\delta}{By^2}\frac{1+\overline{\rho}}{1+\overline{\rho}(x+y)}.
\end{aligned}$$

Obviously, the right hand sides in the two equations above are negative. Taking the unit normal vector of Ω_3

$$\mathbf{n} = \frac{(1,1,1+\overline{\rho})^T}{\sqrt{\overline{\rho}^2 + 2\overline{\rho} + 3}},$$

we obtain $(Curl\mathbf{g}) \cdot \mathbf{n} < 0$. By the Poincaré-Bendixson theorem, we know that the system (24) has no periodic solution. Thus, the equilibrium y^* is GAS in Ω_2. □

Finally, the combination of Theorem 3.6, Theorem 3.9 and Theorem 3.10 establishes the GAS of Z^* as stated in the corollary below.

Corollary 3.11. *The positive endemic equilibrium Z^* of model (10) is globally asymptotically stable when $\mathcal{R}_0 > 1$.*

3.2. *Analysis of the full model* ($q \neq 0$)

3.2.1. *Existence and uniqueness of the equilibrium point*

When $q \neq 0$, one can easily establish that the components of the positive equilibrium $\overline{Z} = (\overline{X}, \overline{Y}, \overline{S}, \overline{E}, \overline{I}, \overline{C})$ of the system (7) satisfy Eqs.(11a), where $\overline{X}, \overline{E}, \overline{I}, \overline{C}$, and $\overline{S}$ replace X^*, E^*, I^*, C^* and S^* respectively. $\overline{Y}$ is the solution of the equation

$$qA + \beta_v\left(\frac{A}{d} - \overline{Y}\right)\frac{\overline{Y}}{1+\alpha\overline{Y}} + \beta_e\left(\frac{A}{d} - \overline{Y}\right)\frac{\overline{C}}{\overline{C}+\kappa} - d\overline{Y} = 0, \tag{27}$$

which takes the form

$$Q(\overline{Y}, q) = a_4\overline{Y}^3 + a_5\overline{Y}^2 + a_6\overline{Y} + a_7 = 0, \tag{28}$$

where,

$$a_4 = -\frac{\beta_v\phi_2}{\xi} - \frac{\beta_e\alpha\phi_2}{\xi} - \frac{d\alpha\phi_2}{\xi}; \quad a_5 = \frac{qA\alpha\phi_2}{\xi} - \kappa\beta_v - d\alpha\kappa + \frac{\beta_v A\phi_2}{d\xi} + \frac{\beta_e A\alpha\phi_2}{d\xi} - \frac{\beta_e\phi_2}{\xi} - \frac{d\phi_2}{\xi},$$
$$a_6 = \kappa qA\alpha + \frac{qA\phi_2}{\xi} + \frac{\kappa\beta_v A}{d} - d\kappa + \frac{\beta_e A\phi_2}{d\xi}; \quad a_7 = \kappa qA.$$

And $\overline{N}$ is the positive solution of the quadratic equation

$$H(\overline{N}) = \alpha_1\overline{N}^2 + \left(\alpha_3\overline{Y} - \alpha_1\frac{B}{\delta}\right)\overline{N} - \alpha_2\overline{Y} = 0. \tag{29}$$

Notice that an endemic equilibrium (i. e. a positive constant solution) $\overline{Z}$ for system (7) is obtained by solving for $\overline{Y}$ equation (28), then solves for $\overline{N}$ equation (29) and finally recover the remaining components of $\overline{Z}$ by replacing the obtained $\overline{Y}$ and $\overline{N}$ into Eqs.(11*a*).

We claim the following result.

Theorem 3.12. *When $q > 0$, system (7) has a unique positive solution $\overline{Z}$. Moreover, the asymptomatic components of $\overline{Z}$ and Z^* satisfy the inequality $\overline{Y} > Y^*$, where $\overline{Y}$ is a positive solution of (28) and Y^* is the unique positive solution of (13) .*

Proof. It is straightforward that, if $\overline{Y}$ is a positive root for (28), then since $\alpha_1 > 0$ and $\alpha_2 > 0$, equation (29) has a unique positive solution. Therefore, it is enough to solve only equation (28) for positive $\overline{Y}$.

Notice that a_7 is positive and a_4 is negative. Thus, according to the Descartes' rule of signs, the number of positive real roots of the polynomial (28) depends on the signs of a_5 and a_6 as depicted in Table 2 below.

Table 2. Total number of possible real roots of (28)

Case	a_4	a_5	a_6	a_7	Number of sign changes	Number of positive real roots
(i)	-	+	+	+	1	1
	-	-	-	+	1	1
	-	-	+	+	1	1
(ii)	-	+	-	+	3	1 or 3

Now, if the signs of a_5 and a_6 follow case (i) of Table 2, then there exists exactly one positive solution $\overline{Y}$ of (28). If rather case (ii) holds, that is $a_5 > 0$ and $a_6 < 0$, the existence of at least one positive solution $\overline{Y}$ is ensured. The uniqueness of $\overline{Y}$ in this case (ii) will follow later by the proof (in Corollary (3.15)) of the global asymptotic stability of endemic equilibrium

$\overline{Z}$, which normally rules out the possibility of multiple equilibria. It remains to prove that $\overline{Y} > Y^*$.

To achieve that, observe that the polynomial $Q(\overline{Y}, q)$ can be rewritten as the explicit function q in the form:

$$Q(\overline{Y}, q) = \overline{Y}P(\overline{Y}) + q\left\{\frac{A\alpha\phi_2}{\xi}\overline{Y}^2 + \left(kA\alpha + \frac{A\phi_2}{\xi}\right)\overline{Y} + kA\right\}. \tag{30}$$

Next for $q > 0, Y^* > 0$, and because $P(Y^*) = 0$ and $\alpha_4 < 0$, we have:

$$Q(Y^*, q) = q\left\{\frac{A\alpha\phi_2}{\xi}(Y^*)^2 + \left(kA\alpha + \frac{A\phi_2}{\xi}\right)Y^* + kA\right\} > 0, \quad \text{and} \quad \lim_{\overline{Y}\to+\infty} Q(\overline{Y}, q) = -\infty.$$

Therefore, it follows by the intermediate value theorem that the unique positive solution $\overline{Y}$ for equation (28) lies in $(Y^*, +\infty)$. That is $\overline{Y} > Y^*$. □

Remark 3.13. *The last statement of Theorem 3.12 established that, when the avian influenza without immigration of infected poultry ($q = 0$) has a unique endemic equilibrium Z^*, then the importation of infected poultry ($q > 0$) introduces no new endemic equilibrium but serves to shift the existing (unique) endemic equilibrium to a higher disease endemic level. In order works, during avian influenza epidemic outbreaks, the recruitment of infected poultry in the farms increases the endemic level of avian influenza in the poultry population.*

3.2.2. *Local and global stability analysis of the unique endemic equilibrium point*

Theorem 3.14. *If $q \neq 0$, the positive equilibrium point $\overline{Z}$ of system (7) is locally asymptotically stable.*

Proof. The Jacobian matrix of the vector field described by (7) at the EE is

$$J = [J_{ij}]_{1\le i,j\le 6} = \begin{bmatrix} -P-d & -Q & 0 & 0 & 0 & -R \\ P & Q-d & 0 & 0 & 0 & R \\ 0 & -\tau_v\frac{\overline{S}}{N} & -\tau_v\frac{\overline{Y}}{N} - \tau_e\frac{\overline{C}}{N} - \delta & a & \gamma & -\tau_e\frac{\overline{S}}{N} \\ 0 & \tau_v\frac{\overline{S}}{N} & \tau_v\frac{\overline{Y}}{N} + \tau_e\frac{\overline{C}}{N} & -(a+\delta+\epsilon) & 0 & \tau_e\frac{\overline{S}}{N} \\ 0 & 0 & 0 & \epsilon & -(\gamma+\rho+\delta) & 0 \\ 0 & \phi_2 & 0 & 0 & 0 & -\xi \end{bmatrix}, \tag{31}$$

where J_{ij} is the non-vanishing (i, j) entry of J and

$$P = \beta_v\frac{\overline{Y}}{1+\alpha\overline{Y}} + \beta_e\frac{\overline{C}}{\overline{C}+\kappa} \quad , \quad Q = \beta_v\frac{\overline{X}}{(1+\alpha\overline{Y})^2} \quad , \quad R = \kappa\beta_e\frac{\overline{X}}{(\kappa+\overline{C})^2}.$$

Let

$$\begin{aligned} c_1 &= -(J_{33} + J_{44} + J_{55}), \\ c_2 &= (J_{33}J_{55} + J_{33}J_{44} + J_{44}J_{55} - J_{34}J_{43}), \\ c_3 &= (J_{34}J_{43}J_{55} + J_{43}J_{54}J_{35} - J_{33}J_{44}J_{55}), \\ \bar{c}_1 &= -(J_{11} + J_{22} + J_{66}), \\ \bar{c}_2 &= (J_{11}J_{66} + J_{22}J_{66} + J_{11}J_{22} + J_{26}J_{62} - J_{12}J_{21}), \\ \bar{c}_3 &= (-J_{11}J_{26}J_{62} + J_{12}J_{21}J_{66} - J_{11}J_{22}J_{66} - J_{21}J_{16}J_{62}). \end{aligned}$$

Then the characteristic equation of $J|_{EE}$ is given by

$$(\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3)(\lambda^3 + \bar{c}_1\lambda^2 + \bar{c}_2\lambda + \bar{c}_3) = 0. \tag{32}$$

We show that

$$c_1 > 0\ ,\ c_2 > 0\ ,\ c_3 > 0\ ,\ \bar{c}_1 > 0\ ,\ \bar{c}_2 > 0\ ,\ \bar{c}_3 > 0\ ,\ c_1c_2 - c_3 > 0\ \ and\ \ \bar{c}_1\bar{c}_2 - \bar{c}_3 > 0. \tag{33}$$

It is clear that $J_{11} < 0\ ,\ J_{22} < 0\ ,\ J_{33} < 0\ ,\ J_{44} < 0\ ,\ J_{55} < 0\ ,\ J_{66} < 0\ ,\ J_{12} < 0\ ,\ J_{16} < 0\ ,\ J_{21} > 0\ ,\ J_{26} > 0\ ,\ J_{32} < 0\ ,\ J_{34} > 0\ ,\ J_{35} > 0\ ,\ J_{42} > 0\ ,\ J_{43} > 0\ ,\ J_{54} > 0\ ,\ J_{62} > 0.$
It follows that

$$c_1 > 0\ ,\ c_2 > 0,\quad \bar{c}_1 > 0\ ,\ \bar{c}_2 > 0\ ,\ \bar{c}_3 > 0\ ,\ c_3 = J_{55}(J_{34}J_{43} - J_{33}J_{44}) + J_{43}J_{54}J_{35} > 0.$$

Meanwhile, we notice that

$$\begin{aligned} c_1c_2 - c_3 &= -J_{33}^2J_{55} - J_{33}^2J_{44} - 2J_{33}J_{44}J_{55} + J_{33}J_{34}J_{43} - J_{44}^2J_{33} - J_{44}^2J_{55} \\ &\quad + J_{44}J_{34}J_{43} - J_{55}^2J_{33} - J_{55}^2J_{44} - J_{43}J_{54}J_{35}, \\ &= -J_{33}^2J_{55} - J_{44}^2J_{55} - 2J_{33}J_{44}J_{55} - J_{43}J_{54}J_{35} + (J_{33} + J_{44})(J_{34}J_{43} - J_{33}J_{44}) > 0, \\ \bar{c}_1\bar{c}_2 - \bar{c}_3 &= -J_{11}^2J_{66} - J_{11}^2J_{22} - J_{22}^2J_{66} - J_{22}^2J_{11} - J_{66}^2J_{11} - J_{66}^2J_{22} \\ &\quad + J_{11}J_{12}J_{21} - 2J_{11}J_{22}J_{66} - J_{22}J_{26}J_{62} + J_{22}J_{12}J_{21} - J_{26}J_{62}J_{66} + J_{16}J_{21}J_{62} > 0. \end{aligned}$$

According to the Routh-Hurwitz criterion, all the eigenvalues of (32) have negative real parts. Thus the endemic equilibrium $\overline{Z}$ of system (7) is locally asymptotically stable in Ω when $q \neq 0$. □

Following the same procedure as for $q = 0$, we can prove that the equilibrium $\overline{Z}$ is globally asymptotically stable even when $q \neq 0$. This result is stated below.

Corollary 3.15. *When $q \neq 0$, the unique positive equilibrium $\overline{Z}$ of system (7) is unconditionally globally asymptotically stable.*

4. Numerical analysis: construction and analysis of a discrete NSFD scheme

In this section, we design a nonstandard finite difference scheme that satisfies the positivity of the state variables involved in the system.

It is important that a numerical method preserves this property when used to solve differential models arising in population biology because these state variables represent sub-populations which never take negative values. To begin with, the time domain $[0, T]$ is partitioned through the discrete time levels $t_n = nh$, where $h > 0$ is the time step-size. To construct the NSFD scheme, we discretize system (7) based on the approximation of the temporal derivatives by a generalized first order forward difference method as follows.

4.1. Construction of a discrete NSFD scheme

For $X(t) \in \mathbb{C}^1$, the discrete derivative is defined by

$$\frac{dX(t)}{dt} = \frac{X(t+h) - X(t)}{\phi(h)} + O(\phi(h)), \quad as \quad h \to 0, \tag{34}$$

where ϕ is a function of step-size, called "denominator" functions. $X(t) \to X_n$ and ϕ have the property

$$\phi(h) = h + O(h^2). \tag{35}$$

The discrete derivative for other state variables are obtained analogously whereas the non-derivative terms are approximated locally, i.e., at the base time level. Denoting the approximations of $X(nh)$, $Y(nh)$, $S(nh)$, $E(nh)$, $I(nh)$ and $C(nh)$ by X^n, Y^n, S^n, E^n, I^n and C^n, respectively, where $n = 0, 1, 2, ...$, the NSFD scheme reads

$$\begin{cases} X^{n+1} - X^n = \phi(h)\left[(1-q)A - \beta_v X^{n+1} f_1(Y^n) - \beta_e X^{n+1} f_2(C^n) - dX^{n+1}\right], \\ Y^{n+1} - Y^n = \phi(h)\left[qA + \beta_v X^{n+1} f_1(Y^n) + \beta_e X^{n+1} f_2(C^n) - dY^{n+1}\right], \\ S^{n+1} - S^n = \phi(h)\left[B + aE^{n+1} + \gamma I^{n+1} - \tau_v g_1(Y^n) S^{n+1} - \tau_e g_2(C^n) S^{n+1} - \delta S^{n+1}\right], \\ E^{n+1} - E^n = \phi(h)\left[\tau_v g_1(Y^n) S^{n+1} + \tau_e g_2(C^n) S^{n+1} - (a + \delta + \epsilon) E^{n+1}\right], \\ I^{n+1} - I^n = \phi(h)\left[\epsilon E^{n+1} - (\gamma + \rho + \delta) I^{n+1}\right], \\ C^{n+1} - C^n = \phi(h)\left[\phi_2 Y^n - \xi C^{n+1}\right], \end{cases} \tag{36}$$

where the discretizations for $f_1(Y^n)$, $f_2(C^n)$, $g_1(Y^n)$ and $g_2(C^n)$ are given by

$$f_1(Y^n) = \frac{Y^n}{1 + \alpha Y^n}, \quad f_2(C^n) = \frac{C^n}{C^n + \kappa}, \quad g_1(Y^n) = \frac{Y^n}{S^n + E^n + I^n}, \quad g_2(C^n) = \frac{C^n}{S^n + E^n + I^n}. \tag{37}$$

In work we choose once for good the function ϕ as follows:

$$\phi(h) = \frac{1 - e^{-(a + \delta + \epsilon + \gamma + \rho + d)h}}{a + \delta + \epsilon + \gamma + \rho + d}. \tag{38}$$

Let

$$\begin{aligned} D_e &= (1 + \phi\delta)(1 + \eta_1\phi)(1 + \eta_2\phi) \\ &\quad + \phi\{\tau_v g_1(Y^n) + \tau_e g_2(C^n)\}\{(1 + \eta_1\phi)(1 + \eta_2\phi) - \phi a(1 + \eta_2\phi) - \phi^2\gamma\epsilon\}. \end{aligned}$$

Simplifying (36), we obtain

$$\begin{aligned} Y^{n+1} &= \frac{qA\phi + Y^n + \phi\{\beta_v f_1(Y^n) + \beta_e f_2(C^n)\}X^{n+1}}{1 + d\phi}, \\ &= \frac{qA\phi(1 + d\phi) + A\phi^2\{\beta_v f_1(Y^n) + \beta_e f_2(C^n)\}}{(1 + d\phi)[1 + \phi\{\beta_v f_1(Y^n) + \beta_e f_2(C^n) + d\}]} \\ &\quad + \frac{[1 + \phi\{\beta_v f_1(Y^n) + \beta_e f_2(C^n) + d\}]Y^n + \phi\{\beta_v f_1(Y^n) + \beta_e f_2(C^n)\}X^n}{(1 + d\phi)[1 + \phi\{\beta_v f_1(Y^n) + \beta_e f_2(C^n) + d\}]}, \\ X^{n+1} &= \frac{(1-q)A\phi + X^n}{1 + \phi\{\beta_v f_1(Y^n) + \beta_e f_2(C^n) + d\}}, \end{aligned}$$

$$
\begin{aligned}
S^{n+1} &= \frac{S^n + \phi\{B + aE^{n+1} + \gamma I^{n+1}\}}{1 + \phi\{\tau_v g_1(Y^n) + \tau_e g_2(C^n) + \delta\}}, \\
&= \frac{B\phi(1+\eta_1\phi)(1+\eta_2\phi)}{D_e} + \frac{(1+\eta_1\phi)(1+\eta_2\phi)S^n}{D_e} \\
&\quad + \frac{\phi[a(1+\eta_2\phi) + \gamma\epsilon\phi]E^n}{D_e} + \frac{\phi\gamma(1+\eta_1\phi)I^n}{D_e}, \\
E^{n+1} &= \frac{E^n + \phi\{\tau_v g_1(Y^n) + \tau_e g_2(C^n)\}S^{n+1}}{1 + \eta_1\phi}, \\
&= \frac{B\phi^2\{\tau_v g_1(Y^n) + \tau_e g_2(C^n)\}(1+\eta_2\phi)}{D_e} \\
&\quad + \frac{\phi\{\tau_v g_1(Y^n) + \tau_e g_2(C^n)\}(1+\eta_2\phi)S^n}{D_e} \\
&\quad + \frac{\phi^2\{\tau_v g_1(Y^n) + \tau_e g_2(C^n)\}[(1+\eta_2\phi) + \gamma\epsilon\phi]E^n}{(1+\eta_1\phi)D_e} \\
&\quad + \frac{\gamma\phi^2\{\tau_v g_1(Y^n) + \tau_e g_2(C^n)\}I^n}{D_e} + \frac{E^n}{(1+\eta_1\phi)}, \\
I^{n+1} &= \frac{\epsilon\phi E^{n+1} + I^n}{1 + \eta_2\phi}, \\
&= \frac{B\epsilon\phi^3\{\tau_v g_1(Y^n) + \tau_e g_2(C^n)\}}{D_e} \\
&\quad + \frac{\epsilon\phi^2\{\tau_v g_1(Y^n) + \tau_e g_2(C^n)\}S^n}{D_e} \\
&\quad + \frac{\epsilon\phi^3\{\tau_v g_1(Y^n) + \tau_e g_2(C^n)\}[(1+\eta_2\phi) + \gamma\epsilon\phi]E^n}{(1+\eta_2\phi)(1+\eta_1\phi)D_e} \\
&\quad + \frac{\epsilon\phi^3\gamma\{\tau_v g_1(Y^n) + \tau_e g_2(C^n)\}I^n}{(1+\eta_2\phi)D_e} \\
&\quad + \frac{\epsilon\phi E^n}{(1+\eta_2\phi)(1+\eta_1\phi)} + \frac{I^n}{1+\eta_2\phi}, \\
C^{n+1} &= \frac{\phi_2\phi Y^n + C^n}{1 + \xi\phi},
\end{aligned}
$$

where, $\eta_1 = a + \delta + \epsilon$ and $\eta_2 = \gamma + \rho + \delta$.
The positivity of the solutions of the scheme (36) follows readily by its construction.

It remains to prove the positive invariance of Ω. Firstly adding the first and the second equations in (36), one has $M^{n+1}(1 + d\phi) = A\phi + M^n$. Therefore, $M^{n+1} \leq A/d$ whenever

$M^n \leq A/d$. Secondly, adding the third, fourth and fifth equations in (36) we have

$$N^{n+1}(1+\delta\phi) = B\phi + N^n - \rho\phi I^{n+1} \leq B\phi + N^n.$$

Therefore, $N^{n+1} \leq B/\delta$ whenever $N^n \leq B/\delta$.

4.2. *Analysis of the discrete NSFD scheme* (36)

In the following sub-section we examine the stability properties of system (36) by proving that: (i) the continuous and the discrete models have the same equilibrium points, and (ii) both models possess similar qualitative features near these equilibrium points.

4.2.1. *The fixed points of the numerical scheme*

Theorem 4.1. *The discrete scheme (36) preserves the equilibrium points of the continuous model (7). That is, on the one hand, the only fixed points of the scheme (36) are either the endemic equilibrium point of the continuous model (7) when $q > 0$ or its disease-free and endemic equilibria when $q = 0$. On the other hand, the fixed points have the same stability properties are those of the equilibrium points of the continuous model (7).*

Proof. Details of this theorem are omitted, because they lead us to existence of equilibria for the continuous model. □

4.2.2. *Stability analysis of the fixed points*

Theorem 4.2. *The disease-free fixed point of the NSFD scheme (36) for the sub-model without imported infected poultry ($q = 0$) is LAS whenever $\mathcal{R}_0 \leq 1$.*

Proof. Note that the Jury criterion is used to study the local stability of the fixed points. The Jacobian matrix of the NSFD scheme is:

$$J^* = \begin{bmatrix} J^*_{11} & J^*_{12} & 0 & 0 & 0 & J^*_{16} \\ J^*_{21} & J^*_{22} & 0 & 0 & 0 & J^*_{26} \\ 0 & J^*_{32} & J^*_{33} & J^*_{34} & J^*_{35} & J^*_{36} \\ 0 & J^*_{42} & J^*_{43} & J^*_{44} & J^*_{45} & J^*_{46} \\ 0 & J^*_{52} & J^*_{53} & J^*_{54} & J^*_{55} & J^*_{56} \\ 0 & J^*_{62} & 0 & 0 & 0 & J^*_{66} \end{bmatrix},$$

where the non vanishing entry of J are:

$$J^*_{11} = \frac{\partial F_1(X^n, Y^n, C^n)}{\partial X^n} = \frac{1}{1+\phi\left(\beta_v \dfrac{Y^n}{1+\alpha Y^n} + \beta_e \dfrac{C^n}{C^n+\kappa} + d\right)},$$

$$J^*_{12} = \frac{\partial F_1(X^n, Y^n, C^n)}{\partial Y^n} = -\frac{\phi\beta_v[(1-q)A\phi + X^n]}{(1+\alpha Y^n)^2\left(1+\phi\{\beta_v \dfrac{Y^n}{1+\alpha Y^n} + \beta_e \dfrac{C^n}{C^n+\kappa} + d\}\right)^2},$$

$$J^*_{16} = \frac{\partial F_1(X^n, Y^n, C^n)}{\partial C^n} = -\frac{\phi\beta_e\kappa[(1-q)A\phi + X^n]}{(C^n+\kappa)^2\left(1+\phi\{\beta_v \dfrac{Y^n}{1+\alpha Y^n} + \beta_e \dfrac{C^n}{C^n+\kappa} + d\}\right)^2},$$

$$J^*_{21} = \frac{\partial F_2(X^n, Y^n, C^n)}{\partial X^n} = \frac{\phi\left(\beta_v \frac{Y^n}{1+\alpha Y^n} + \beta_e \frac{C^n}{C^n+\kappa}\right)}{(1+d\phi)\left(1+\phi\left(\beta_v \frac{Y^n}{1+\alpha Y^n} + \beta_e \frac{C^n}{C^n+\kappa} + d\right)\right)},$$

$$J^*_{22} = \frac{\partial F_2(X^n, Y^n, C^n)}{\partial Y^n} = \frac{\phi\beta_v[(1-q)A\phi + X^n]}{(1+d\phi)(1+\alpha Y^n)^2\left(1+\phi\{\beta_v \frac{Y^n}{1+\alpha Y^n} + \beta_e \frac{C^n}{C^n+\kappa} + d\}\right)}$$
$$- \frac{\phi^2\beta_v[(1-q)A\phi + X^n]\left(\beta_v \frac{Y^n}{1+\alpha Y^n} + \beta_e \frac{C^n}{C^n+\kappa}\right)}{(1+d\phi)(1+\alpha Y^n)^2\left(1+\phi\{\beta_v \frac{Y^n}{1+\alpha Y^n} + \beta_e \frac{C^n}{C^n+\kappa} + d\}\right)^2} + \frac{1}{1+d\phi},$$

$$J^*_{26} = \frac{\partial F_2(X^n, Y^n, C^n)}{\partial C^n} = \frac{\phi\beta_e\kappa[(1-q)A\phi + X^n]}{(1+d\phi)(\kappa+C^n)^2\left(1+\phi\{\beta_v \frac{Y^n}{1+\alpha Y^n} + \beta_e \frac{C^n}{C^n+\kappa} + d\}\right)}$$
$$- \frac{\phi^2\beta_e\kappa(1-q)A\phi + X^n]\left(\beta_v \frac{Y^n}{1+\alpha Y^n} + \beta_e \frac{C^n}{C^n+\kappa}\right)}{(1+d\phi)(C^n+\kappa)^2\left(1+\phi\{\beta_v \frac{Y^n}{1+\alpha Y^n} + \beta_e \frac{C^n}{C^n+\kappa} + d\}\right)^2},$$

$$J^*_{62} = \frac{\partial F_6(X^n, Y^n, C^n)}{\partial Y^n} = \frac{\phi_2\phi}{1+\xi\phi},$$
$$J^*_{66} = \frac{\partial F_6(X^n, Y^n, C^n)}{\partial C^n} = \frac{1}{1+\xi\phi},$$
$$J^*_{33} = \frac{\partial F_3(X^n, Y^n, C^n)}{\partial S^n} = \frac{O_1}{D_e^2},$$

$$J^*_{34} = \frac{\partial F_3(X^n, Y^n, C^n)}{\partial E^n} = \frac{O_2}{D_e^2},$$
$$J^*_{35} = \frac{\partial F_3(X^n, Y^n, C^n)}{\partial I^n} = \frac{O_3}{D_e^2},$$
$$J^*_{43} = \frac{\partial F_4(X^n, Y^n, C^n)}{\partial S^n} = \frac{O_4}{1+\eta_1\phi},$$
$$J^*_{44} = \frac{\partial F_4(X^n, Y^n, C^n)}{\partial E^n} = \frac{1}{1+\eta_1\phi} + \frac{O_5}{1+\eta_1\phi},$$
$$J^*_{45} = \frac{\partial F_4(X^n, Y^n, C^n)}{\partial I^n} = \frac{O_6}{1+\eta_1\phi},$$
$$J^*_{53} = \frac{\partial F_5(X^n, Y^n, C^n)}{\partial S^n} = \frac{\epsilon\phi \times O_4}{(1+\eta_1\phi)(1+\eta_2\phi)},$$
$$J^*_{54} = \frac{\partial F_5(X^n, Y^n, C^n)}{\partial E^n} = \frac{\epsilon\phi \times O_5}{(1+\eta_1\phi)(1+\eta_2\phi)} + \frac{\epsilon\phi}{(1+\eta_1\phi)(1+\eta_2\phi)},$$
$$J^*_{55} = \frac{\partial F_5(X^n, Y^n, C^n)}{\partial I^n} = \frac{\epsilon\phi \times O_6}{(1+\eta_1\phi)(1+\eta_2\phi)} + \frac{1}{1+\eta_2\phi},$$

$$
\begin{aligned}
O_1 &= (1+\eta_1\phi)(1+\eta_2\phi)D_e \\
&\quad +\Big(\tau_v\frac{Y^n}{(S^n+E^n+I^n)^2}+\tau_e\frac{C^n}{(S^n+E^n+I^n)^2}\Big)\Big((1+\eta_1\phi)(1+\eta_2\phi)-\phi a(1+\eta_2\phi)-\phi^2\gamma\epsilon\Big)N_u,\\
O_2 &= \phi[(1+\eta_2\phi)+\gamma\epsilon\phi]D_e \\
&\quad +\Big(\tau_v\frac{Y^n}{(S^n+E^n+I^n)^2}+\tau_e\frac{C^n}{(S^n+E^n+I^n)^2}\Big)\Big((1+\eta_1\phi)(1+\eta_2\phi)-\phi a(1+\eta_2\phi)-\phi^2\gamma\epsilon\Big)N_u,\\
O_3 &= \phi\gamma(1+\eta_1\phi)D_e \\
&\quad +\Big(\tau_v\frac{Y^n}{(S^n+E^n+I^n)^2}+\tau_e\frac{C^n}{(S^n+E^n+I^n)^2}\Big)\Big((1+\eta_1\phi)(1+\eta_2\phi)-\phi a(1+\eta_2\phi)-\phi^2\gamma\epsilon\Big)N_u,\\
O_4 &= -\phi\Big(\tau_v\frac{Y^n}{(S^n+E^n+I^n)^2}+\tau_e\frac{C^n}{(S^n+E^n+I^n)^2}\Big)\times\frac{N_u}{D_e} \\
&\quad +J_{33}\times\phi\Big(\tau_v\frac{Y^n}{(S^n+E^n+I^n)}+\tau_e\frac{C^n}{(S^n+E^n+I^n)}\Big),\\
O_5 &= -\phi\Big(\tau_v\frac{Y^n}{(S^n+E^n+I^n)^2}+\tau_e\frac{C^n}{(S^n+E^n+I^n)^2}\Big)\times\frac{N_u}{D_e} \\
&\quad +J_{34}\times\phi\Big(\tau_v\frac{Y^n}{(S^n+E^n+I^n)}+\tau_e\frac{C^n}{(S^n+E^n+I^n)}\Big),\\
O_6 &= -\phi\Big(\tau_v\frac{Y^n}{(S^n+E^n+I^n)^2}+\tau_e\frac{C^n}{(S^n+E^n+I^n)^2}\Big)\times\frac{N_u}{D_e} \\
&\quad +J_{35}\times\phi\Big(\tau_v\frac{Y^n}{(S^n+E^n+I^n)}+\tau_e\frac{C^n}{(S^n+E^n+I^n)}\Big),\\
N_u &= B\phi(1+\eta_1\phi)(1+\eta_2\phi)+(1+\eta_1\phi)(1+\eta_2\phi)S^n+\phi[(1+\eta_2\phi)+\gamma\epsilon\phi]E^n+\phi\gamma(1+\eta_1\phi)I^n.
\end{aligned}
$$

Firstly, we show the local stability of disease-free fixed point $\widehat{Z}^0$. Substituting the disease-free fixed point $\widehat{Z}^0$ into the Jacobian matrix, we find that the eigenvalues are

$$\lambda_1=\frac{1}{1+d\phi},\quad \lambda_3=\frac{1}{1+\phi\delta},\quad \lambda_4=\frac{1}{1+\eta_1\phi},\quad \lambda_5=\frac{1}{1+\eta_2\phi},$$

and the others satisfies

$$\lambda^2-b_1\lambda+b_2=0, \tag{39}$$

where

$$b_1=\frac{1}{1+\xi\phi}+\frac{d+\phi\beta_v A}{d(1+d\phi)},\quad b_2=\frac{d+\phi\beta_v A}{d(1+\xi\phi)(1+d\phi)}-\frac{\phi^2\beta_e A\phi_2}{d\kappa(1+\xi\phi)(1+d\phi)}.$$

We have

$$1+b_1+b_2=\frac{d\xi\phi_2(1-\mathcal{R}_0)}{(1+\xi\phi)(1+d\phi)}, \tag{40a}$$

$$1-b_1+b_2=1+\frac{d\kappa(1+d\phi)+2\kappa(d+\phi\beta_v A)+2\kappa\xi\phi^2\beta_v A+d\kappa\xi\phi(1-\phi\mathcal{R}_0)}{d\kappa(1+d\phi)(1+\xi\phi)}, \tag{40b}$$

$$1-b_2=\frac{d^2\kappa\phi(1-\mathcal{R}_0)(1+\xi\phi)+d\kappa\xi\phi(1+\mathcal{R}_0 d\phi)+\dfrac{\beta_e A\phi_2\phi}{\xi}(1+\xi\phi)}{d\kappa(1+d\phi)(1+\xi\phi)}. \tag{40c}$$

Clearly, the expressions in (40a), (40b) and (40c) are positive if $\mathcal{R}_0 \leq 1$. The LAS of the disease-free fixed point follows from Jury criterion for (39) given by:

$$1-b_1+b_2>0, \quad 1+b_1+b_2>0, \quad 1-b_2>0.$$

□

As for the local asymptotic stability of endemic fixed point, the characteristic equation associated with the above matrix J evaluated at the endemic fixed point is

$$(\lambda^3+b_3\lambda^2+b_4\lambda+b_5)(\lambda^3+b_6\lambda^2+b_7\lambda+b_8)=0, \tag{41}$$

where,

$$\begin{aligned}
b_3 &= -(J_{11}+J_{22}+J_{66}) = \frac{J_1}{(1+\xi\phi)(1+d\phi)(\kappa+\widehat{C})^2(1+\alpha\widehat{Y})^2[1+\phi(P+d)]^3},\\
b_4 &= J_{11}J_{22}+J_{11}J_{66}+J_{22}J_{66}-J_{26}J_{62}-J_{12}J_{21},\\
&= \frac{J_2}{(1+\xi\phi)(1+d\phi)(\kappa+\widehat{C})^2(1+\alpha\widehat{Y})^2[1+\phi(P+d)]^3},\\
b_5 &= J_{11}J_{26}J_{62}+J_{12}J_{21}J_{66}-J_{11}J_{22}J_{66}+J_{16}J_{21}J_{62},\\
&= \frac{J_3}{(1+\xi\phi)(1+d\phi)(\kappa+\widehat{C})^2(1+\alpha\widehat{Y})^2[1+\phi(P+d)]^3},\\
b_6 &= -(J_{33}+J_{44}+J_{55}) = \frac{J_4}{D_e^6(1+\eta_1\phi)^2(1+\eta_2\phi)},\\
b_7 &= J_{33}J_{44}+J_{33}J_{55}+J_{44}J_{55}-J_{45}J_{54}-J_{34}J_{43}-J_{35}J_{53} = \frac{J_5}{D_e^6(1+\eta_1\phi)^2(1+\eta_2\phi)},\\
b_8 &= J_{33}J_{45}J_{54}+J_{34}J_{43}J_{55}-J_{33}J_{44}J_{55}+J_{35}J_{43}J_{54}+J_{35}J_{53}J_{44}-J_{33}J_{53}J_{45} = \frac{J_6}{D_e^6(1+\eta_1\phi)^2(1+\eta_2\phi)},
\end{aligned}$$

$$\begin{aligned}
J_1 = {} & -(1+\xi\phi)(1+d\phi)(\kappa+\widehat{C})^2(1+\alpha\widehat{Y})^2[1+\phi(P+d)]^2\\
& -\phi\beta_v[(1-q)A\phi+\widehat{X}](1+\xi\phi)(\kappa+\widehat{C})^2[1+\phi(P+d)]^2\\
& +\phi\beta_v P[(1-q)A\phi+\widehat{X}](1+\xi\phi)(\kappa+\widehat{C})^2[1+\phi(P+d)]\\
& -(1+\xi\phi)(\kappa+\widehat{C})^2(1+\alpha\widehat{Y})^2[1+\phi(P+d)]^3\\
& -(1+d\phi)(\kappa+\widehat{C})^2(1+\alpha\widehat{Y})^2[1+\phi(P+d)]^3,
\end{aligned}$$

$$
\begin{aligned}
J_2 &= \phi\beta_v[(1-q)A\phi+\widehat{X}](1+\xi\phi)(\kappa+\widehat{C})^2[1+\phi(P+d)]\\
&\quad -\phi^2P\beta_v[(1-q)A\phi+\widehat{X}](1+\xi\phi)(\kappa+\widehat{C})^2\\
&\quad +\phi\beta_v[(1-q)A\phi+\widehat{X}](\kappa+\widehat{C})^2[1+\phi(P+d)]^2\\
&\quad -\phi^2P\beta_v[(1-q)A\phi+\widehat{X}](\kappa+\widehat{C})^2[1+\phi(P+d)]\\
&\quad +(\kappa+\widehat{C})^2(1+\alpha\widehat{Y})^2[1+\phi(P+d)]^3\\
&\quad +(1+\xi\phi)(\kappa+\widehat{C})^2(1+\alpha\widehat{Y})^2[1+\phi(P+d)]^2\\
&\quad +(1+d\phi)(\kappa+\widehat{C})^2(1+\alpha\widehat{Y})^2[1+\phi(P+d)]^2\\
&\quad -\phi^2\beta_e\kappa\phi_2[(1-q)A\phi+\widehat{X}](1+\alpha\widehat{Y})^2[1+\phi(P+d)]^2\\
&\quad +\phi^3\beta_e\kappa\phi_2P[(1-q)A\phi+\widehat{X}](1+\alpha\widehat{Y})^2[1+\phi(P+d)]\\
&\quad +\phi^2\beta_vP[(1-q)A\phi+\widehat{X}](1+\xi\phi)(\kappa+\widehat{C})^2,
\end{aligned}
$$

$$
\begin{aligned}
J_3 &= -\phi\beta_v[(1-q)A\phi+\widehat{X}](\kappa+\widehat{C})^2[1+\phi(P+d)]+\phi^2P\beta_v[(1-q)A\phi+\widehat{X}](\kappa+\widehat{C})^2\\
&\quad -(\kappa+\widehat{C})^2(1+\alpha\widehat{Y})^2[1+\phi(P+d)]^2+\phi^2\beta_e\kappa\phi_2[(1-q)A\phi+\widehat{X}](1+\alpha\widehat{Y})^2[1+\phi(P+d)]\\
&\quad -\phi^3\beta_e\kappa\phi_2P[(1-q)A\phi+\widehat{X}](1+\alpha\widehat{Y})^2-\phi^2\beta_vP[(1-q)A\phi+\widehat{X}](\kappa+\widehat{C})^2\\
&\quad -\phi^3\beta_e\kappa\phi_2P[(1-q)A\phi+\widehat{X}](1+\alpha\widehat{Y})^2,
\end{aligned}
$$

$$
\begin{aligned}
J_4 &= -(1+\eta_1\phi)^3(1+\eta_2\phi)^2D_e^5-(1+\eta_1\phi)^2(1+\eta_2\phi)P_1P_2N_uD_e^4\\
&\quad -D_e^6(1+\eta_1\phi)(1+\eta_2\phi)-D_e^6(1+\eta_1\phi)^2+\phi D_e^5(1+\eta_1\phi)(1+\eta_2\phi)P_1N_u\\
&\quad -\phi^2(1+\eta_1\phi)[(1+\eta_2\phi)+\gamma\epsilon\phi](1+\eta_2\phi)P_3D_e^5-\phi(1+\eta_1\phi)(1+\eta_2\phi)P_1P_2P_3D_e^4N_u\\
&\quad +\epsilon\phi^2(1+\eta_1\phi)P_1N_uD_e^5-\phi^2\gamma(1+\eta_1\phi)^2D_e^5P_3-(1+\eta_1\phi)P_1P_2P_3\phi D_e^4N_u,
\end{aligned}
$$

$$
\begin{aligned}
J_5 &= O_1(1+\eta_1\phi)(1+\eta_2\phi)D_e^4-O_1\phi P_1N_u(1+\eta_1\phi)(1+\eta_2\phi)D_e^3\\
&\quad +\epsilon\phi^2O_1O_3P_3D_e^2(1+\eta_1\phi)-\epsilon\phi^2O_1P_1N_uD_e^3(1+\eta_1\phi)+O_1D_e^4(1+\eta_1\phi)^2\\
&\quad +\epsilon\phi^2O_2O_3P_3D_e^2(1+\eta_1\phi)-\epsilon\phi^2O_2P_1D_e^3N_u(1+\eta_1\phi)+O_2D_e^4(1+\eta_1\phi)^2\\
&\quad -\epsilon\phi(\phi P_1N_uD_e)^2D_e^2+\epsilon\phi^3O_3P_1P_3N_uD_e^3+\epsilon\phi^3O_2P_1P_3N_uD_e^3\\
&\quad -\epsilon\phi^3O_2O_3P_3^2D_e^2-\epsilon\phi D_e^6+O_2\phi P_1N_u(1+\eta_1\phi)(1+\eta_2\phi)D_e^3\\
&\quad +\epsilon\phi O_3O_4(1+\eta_1\phi)D_e^4,
\end{aligned}
$$

$$
\begin{aligned}
J_6 &= \epsilon\phi O_2(\phi P_1N_uD_e)^2-2\epsilon\phi^3O_2O_3P_1P_3N_uD_e-\epsilon\phi^3O_1O_2P_1P_3N_uD_e\\
&\quad +2\epsilon\phi^3O_1O_2O_3P_3^2-\phi O_2P_1N_uD_e^3(1+\eta_1\phi)+\phi O_1P_1N_uD_e^3(1+\eta_1\phi)\\
&\quad -O_1D_e^4(1+\eta_1\phi)+\epsilon\phi O_3(\phi P_1N_uD_e)^2-\epsilon\phi^3O_1O_3P_1P_3N_uD_e\\
&\quad +\epsilon\phi^2O_1O_3P_3D_e^2-\epsilon\phi^2O_3P_1N_uD_e^3\\
&\quad +\epsilon\phi O_3O_4D_e^4+\epsilon\phi O_3O_4O_5D_e^4-\epsilon\phi O_1O_5O_6D_e^4.
\end{aligned}
$$

Theorem 4.3. *If $\mathcal{R}_0>1$, the endemic fixed point for the sub-model without imported infected poultry is LAS if and only if the following conditions are met:*

(1) $: 1+b_3+b_4+b_5>0,$
(2) $: 1-b_3+b_4-b_5>0,$
(3) $: 1+b_3b_5-b_4-b_5^2>0,$
(4) $: 3+b_3-b_4-3b_5>0,$
(5) $: 1+b_6+b_7+b_8>0,$
(6) $: 1-b_6+b_7-b_8>0,$
(7) $: 1+b_6b_8-b_7-b_8^2>0,$
(8) $: 3+b_6-b_7-3b_8>0.$

The LAS of the endemic fixed point for the full model can be shown analogously to Theorem 4.3 and the following theorem hold true.

Theorem 4.4. *The endemic fixed point for the full model is LAS if and only if the following conditions hold:*

(a) : $1 + d_3 + d_4 + d_5 > 0$,
(b) : $1 - d_3 + d_4 - d_5 > 0$,
(c) : $1 + d_3 d_5 - d_4 - d_5^2 > 0$,
(d) : $3 + d_3 - d_4 - 3d_5 > 0$,
(e) : $1 + d_6 + d_7 + d_8 > 0$,
(f) : $1 - d_6 + d_7 - d_8 > 0$,
(i) : $1 + d_6 d_8 - d_7 - d_8^2 > 0$,
(j) : $3 + d_6 - d_7 - 3d_8 > 0$,

where the coefficients d_3 to d_8 have respectively the same form as b_3 to b_8. Moreover, they are obtained from the characteristic polynomial of J evaluated at the endemic fixed point of full model which has the same form as equation (41). That is:

$$(\lambda^3 + d_3\lambda^2 + d_4\lambda + d_5)(\lambda^3 + d_6\lambda^2 + d_7\lambda + d_8) = 0. \tag{42}$$

Conditions $(1) - (8)$ and $(a) - (j)$ are very difficult to established analytically, however they can be verifed numerically.

From the results in this section, we can conclude that both models (the continuous one (7) and the discrete one (36) have the same equilibria, and their behaviors are qualitatively similar near these equilibria. Hence, the nonstandard finite difference method (36) is elementary stable.

We end this section by providing some numerical simulations using the non-standard finite difference method (36) and the 4th order Runge Kutta method (RK4) encoded in the MatLab platform. At the start, we compare both RK4 and NSFD schemes for the discretization step size h =0.05. It is observed that both the numerical schemes are respectively convergent and converge numerically to the true steady states (Z^0 and Z^*) of the continuous model as shown in Figures 3-4 . Moreover, for h = 0.05, RK4 and NSFD exhibit positive solutions in the basic feasible region Ω.

Critically, if we compare the schemes for the step size h = 0.1, RK4 exhibits negative solutions and does not converge to both Z^0 and Z^*, respectively, but NSFD preserves positivity and gives convergence of the solutions, as shown in Figures 5-6.

The above discussion shows that the RK4 scheme does not always converge rather conditionally converge and depends on the value of the step size h, and does not preserve the convergence and positivity for a large step size ($h \geq 0.1$), while Figures 3-6 illustrate the power of an unconditionally convergent NSFD scheme to produce the converged and positives solutions of the model for any value of the step size h.

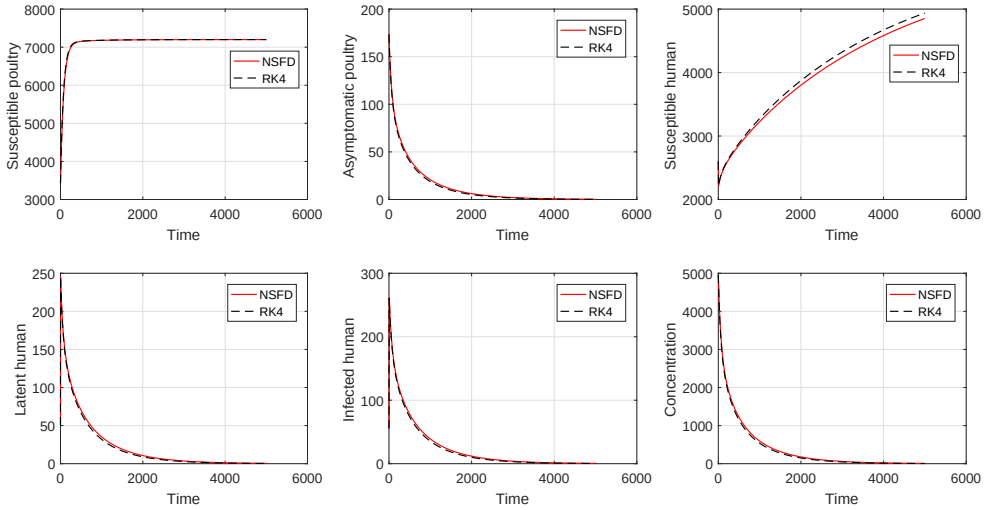

Figure 3. Comparison of solutions obtained by NSFD and RK4 numerical schemes for Z^0 with an initial condition (X(0), Y(0), S(0), E(0), I(0), C(0)) = (3426.56, 173.442, 2603.125, 50, 55.478, 4959.487) and step size h = 0.05. Both the schemes converge to the true steady state of Z^0 with $\mathcal{R}_0 = 0.9183 < 1$.

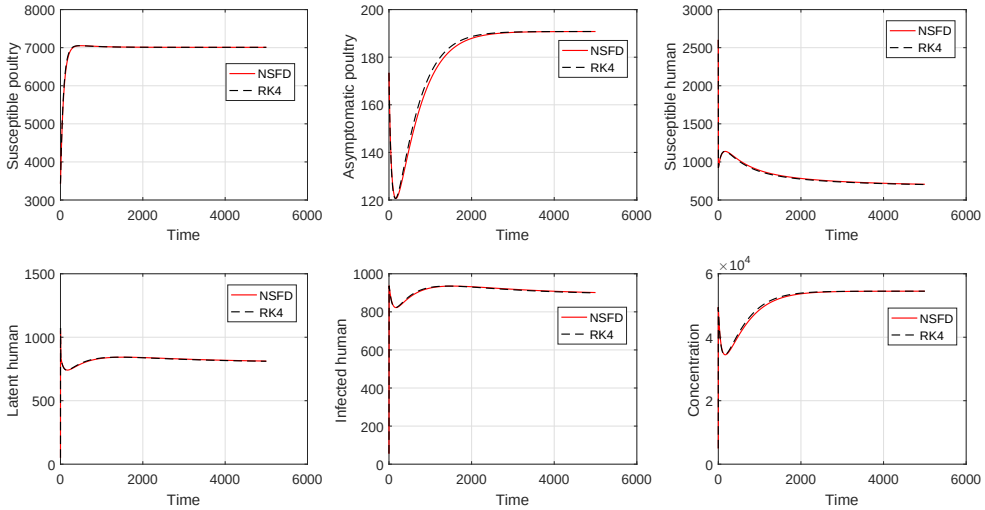

Figure 4. Comparison of solutions obtained by NSFD and RK4 numerical schemes for Z^* with an initial condition (X(0), Y(0), S(0), E(0), I(0), C(0)) = (3426.56, 173.442, 2603.125, 50, 55.478, 4959.487) and step size h = 0.05. Both the schemes converge to the true steady state of Z^* with $\mathcal{R}_0 = 1.1849 > 1$.

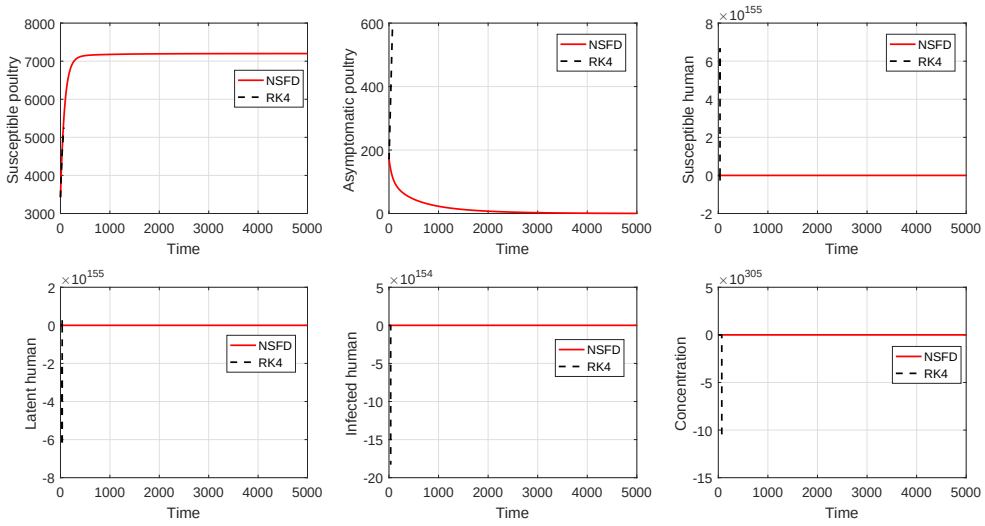

Figure 5. Comparison of solutions obtained by NSFD and RK4 numerical schemes for Z^0 with an initial condition (X(0), Y(0), S(0), E(0), I(0), C(0)) = (3426.56, 173.442, 2603.125, 50, 55.478, 4959.487) and step size h = 0.1, with $\mathcal{R}_0 = 0.9183 < 1$. The NSFD scheme converges to the true steady state of Z^0, whereas the RK4 scheme is seen to be divergent.

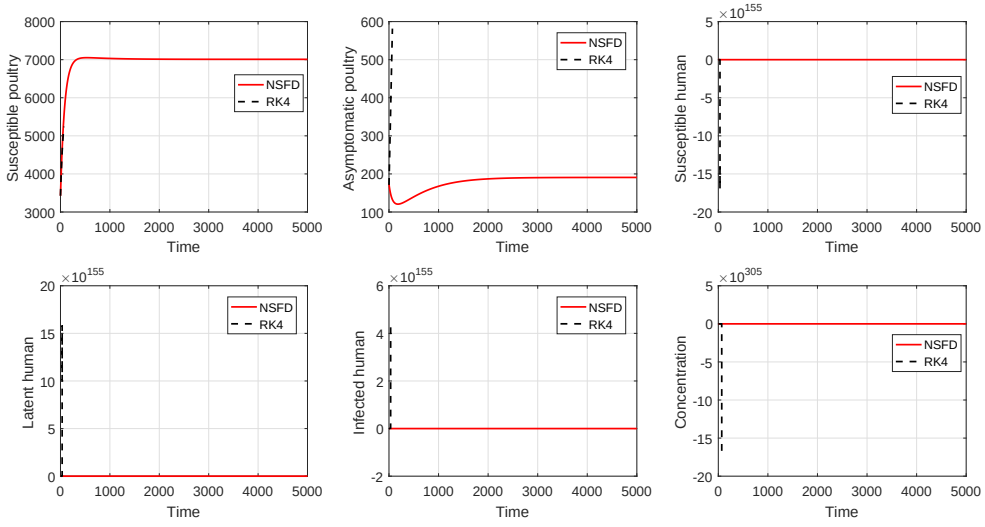

Figure 6. Comparison of solutions obtained by NSFD and RK4 numerical schemes for Z^* with an initial condition (X(0), Y(0), S(0), E(0), I(0), C(0)) = (3426.56, 173.442, 2603.125, 50, 55.478, 4959.487) and step size h = 0.1 with $\mathcal{R}_0 = 1.1849 > 1$. The comparison shows that the RK4 scheme fails to converge. However, the NSFD scheme improves the result obtained by RK4 and it is seen to be convergent dynamically to the correct endemic equilibrium Z^*.

5. Numerical illustrations: assessment of the role of environmental and spillover transmissions

In this section, we use model (7) to further investigate the impact of some control strategies on the spread of Avian influenza infection among poultry and human populations. Based on the sensitivity analysis of the basic reproduction number with respect to its parameters, we seek optimal measures to control the transmission of the disease using parameter values in Table 3 related, which have been found in the literature.

Table 3. Parameters, their definitions and baseline values.

Symbols	Definitions	Estimate for AIV	Source
q	Proportion of asymptomatic imported poultry	0.01	[12]
A	Numbers of imported poultry	100 ind.week^{-1}	[12]
β_v	Direct contact rate in poultry host	1.71 $\times 10^{-6}$/(ind.week)	[31]
β_e	Indirect contact rate in poultry host	0.002 week^{-1}	Assumed
d	Natural death rate of poultry	1/72 week^{-1}	[9]
α	Parameter of the inhibitory effort	0.001 ind^{-1}	[31]
B	Recruitment rate for humans	1.5 ind/week	[12]
a	Recovery rate of the latent humans	1 week^{-1}	[31]
γ	Recovery rate of the infected humans	0.9/ week	[31]
ρ	Disease-related death rate	0.001 week^{-1}	[12]
τ_v	Transmission rate of AIV from poultry to human	0.6 week^{-1}	[12]
ϵ	Morbidity of the latent humans	1 week^{-1}	[12]
δ	Natural death rate of humans	0.00025641 week^{-1}	[9]
κ	Half saturation rate (eID_{50})	$10^6 g.m^3$	[12]
ξ	Degradation rate of virus	35 week^{-1}	Assumed
τ_e	Transmission rate of AIV from environment to human	ind./($g.m^3$.week)	Assumed
ϕ_2	Emission rate of poultry	$g.m^3$ /(ind.week)	Assumed

5.0.1. *Numerical illustrations of stability results.*

To illustrate the stability results contained in this paper, model (7) is simulated using the parameter values in Table 3. It is assumed that the average poultry lifespan is 72 weeks and the average lifespan of human being is 75 years.

Figure 7 presents the time series of model (7) for different initial conditions when $\phi_2 = 10^3$, $\tau_e = 0.1$ and $\mathcal{R}_0 \leq 1$. From these figures, we can see that there are always susceptible individuals all types in the population while all the infected populations and avian influenza viruses disappear. Thus, the trajectories converge to the disease-free equilibrium. This means that the disease disappears in the host populations as proved in Theorem 3.5.

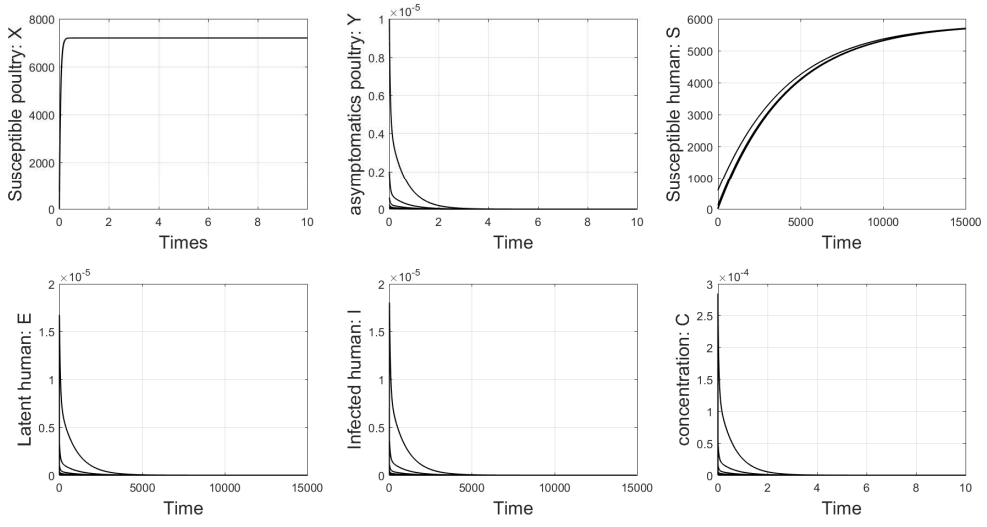

Figure 7. Global stability of disease-free equilibrium. The parameters are: q=0 , A=100, β_v=1.7143, β_e=0.002, d=1/72, α=0.001, B=1.5, a=1, γ=0.9, ρ=0.001, τ_v=0.6, ϵ=1, δ=0.00025641, $\kappa = 10^6$, ξ=35, τ_e=0.1, $\phi_2 = 10^3$ and $\mathcal{R}_0 = 0.9183 \leq 1$.

Figure 8 plots the solutions when $\phi_2 = 10^4$, $\tau_e = 0.1$, $\mathcal{R}_0 > 1$ and for various initial conditions. We can observe that the infected individuals and viruses persist in the population. This means that the trajectories converge to the endemic equilibrium point. Thus, whenever $\mathcal{R}_0 > 1$, the disease persists in the population as established in Corollary 3.11.

Figure 9 depicts the solutions when $\phi_2 = 10^3$, $\tau_e = 0.1$ and $q = 0.1$. From this figure, the infected individuals and viruses remain in the population as the trajectories converge to the endemic equilibrium point. Thus, whenever $q \neq 0$, the disease persists in the population as established in Theorem 3.14.

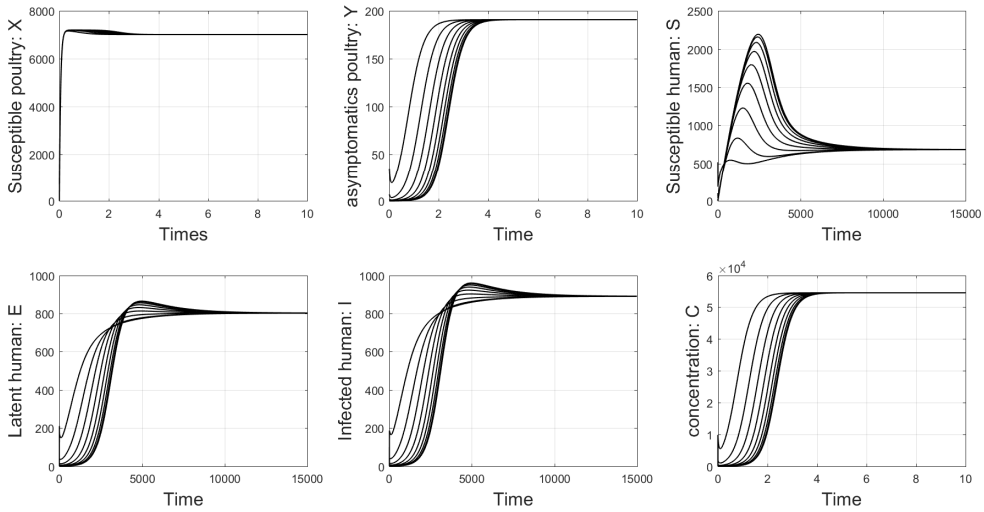

Figure 8. Global stability of endemic equilibrium. The parameters are: q=0 , A=100, β_v=1.7143, β_e=0.002, d=1/72, α=0.001, B=1.5, a=1, γ=0.9, ρ=0.001, τ_v=0.6, ϵ=1, δ=0.00025641, $\kappa = 10^6$, ξ=35, τ_e=0.1, $\phi_2 = 10^4$ and $\mathcal{R}_0 = 1.1849 > 1$.

5.0.2. Impact of transmission coefficient of AIV from environment to human.

Firstly, we fix $\phi_2 = 10^4$, $\tau_v = 0.6$ and vary τ_e to observe the effect of increasing the transmission rate of AIV from environment to asymptomatic poultry and infected humans.

When we vary τ_e from 10% to 15% after fixing $\tau_v = 0.6$, $\phi_2 = 10^4$, we see in Figure 10 that the impact of transmission rate τ_e is a bit stronger. Thus, the low ratio of this transmission rate stands for a control measure against AIV infection in places with high prevalent AIV. Even in this favorable situation, the number of infected individuals remains high. This implies that the control of τ_e alone is not sufficient to effectively eliminate the disease.

5.0.3. Impact of transmission coefficient of AIV from poultry to human.

Secondly, we fix $\phi_2 = 10^4$, $\tau_e = 0.1$ and vary τ_v to observe the effect of increasing the transmission coefficient of AIV from poultry to human on infected humans. We see from Figure 11 that, the increment of τ_v from 60% to 65% has an increase effect on both asymptomatic poultry and infected humans.

5.0.4. Impact of the imported infected poultry proportion q on asymptomatic poultry and infected humans

The effect of the parameter q on both asymptomatic poultry and infected human is shown in Figure 12. From this figure, we conclude that increase in the proportion from 10% to 15% causes the increase in both asymptomatic poultry and infected humans.

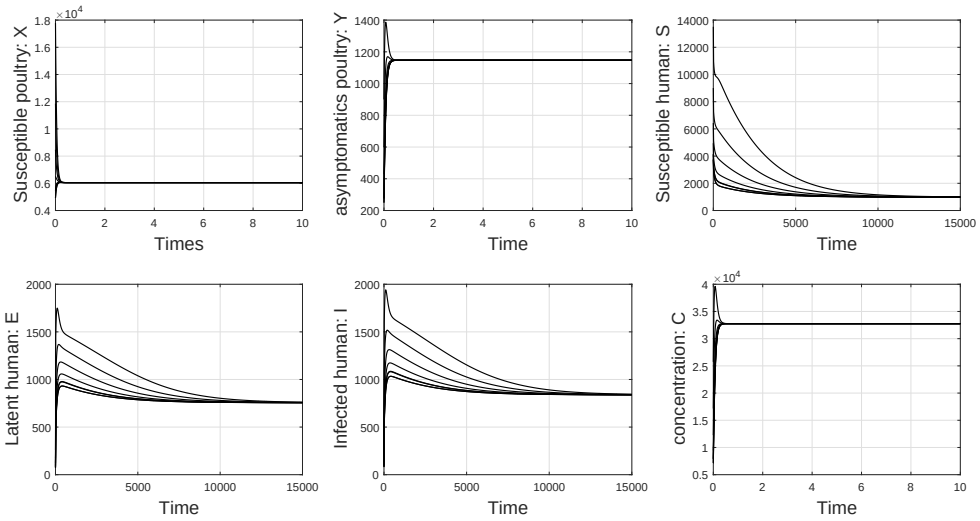

Figure 9. Global stability of the endemic equilibrium whenever $q \neq 0$. The parameters are: q=0.1 , A=100, β_v=1.7143, β_e=0.002, d=1/72, α=0.001, B=1.5, a=1, γ=0.9, ρ=0.001, τ_v=0.6, ϵ=1, δ=0.00025641, $\kappa = 10^6$, ξ=35, τ_e=0.1, $\phi_2 = 10^3$.

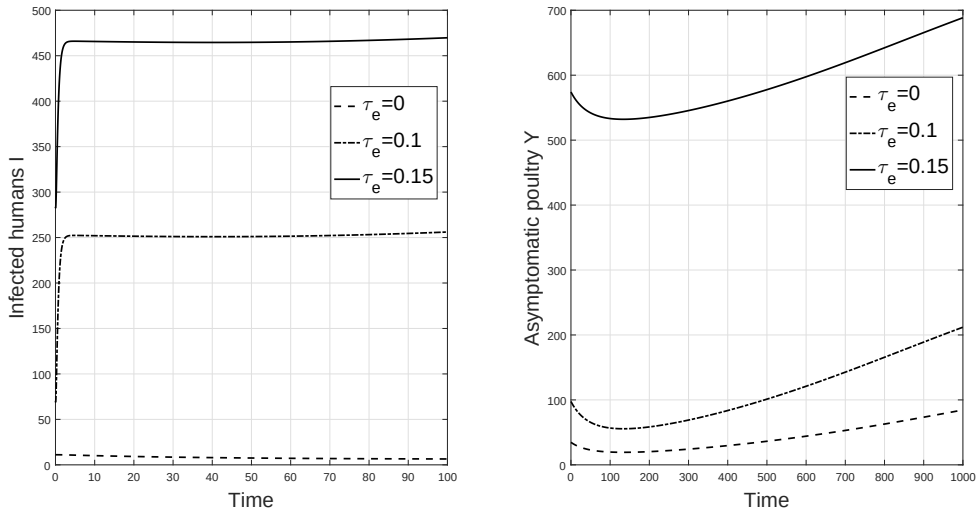

Figure 10. Simulation results showing the impact of transmission rate τ_e. On infected humans (left panel). On asymptomatic poultry (right panel). Both graphs are sketched with the following parameters: q=0, A=100, β_v=1.7143, β_e=0.002, d=1/72, α=0.001, B=1.5, a=1, γ=0.9, ρ=0.001, τ_v=0.6, ϵ=1, δ=0.00025641, $\kappa = 10^6$, ξ=35, $\phi_2 = 10^4$.

These numerical simulations of model (7) suggest that a significant reduction of transmission rate τ_e can be an efficient control strategy for AIV transmission. However, combining the reduction of the emission rate of poultry and the recruitment of infected poultry could be a better control measure for the disease.

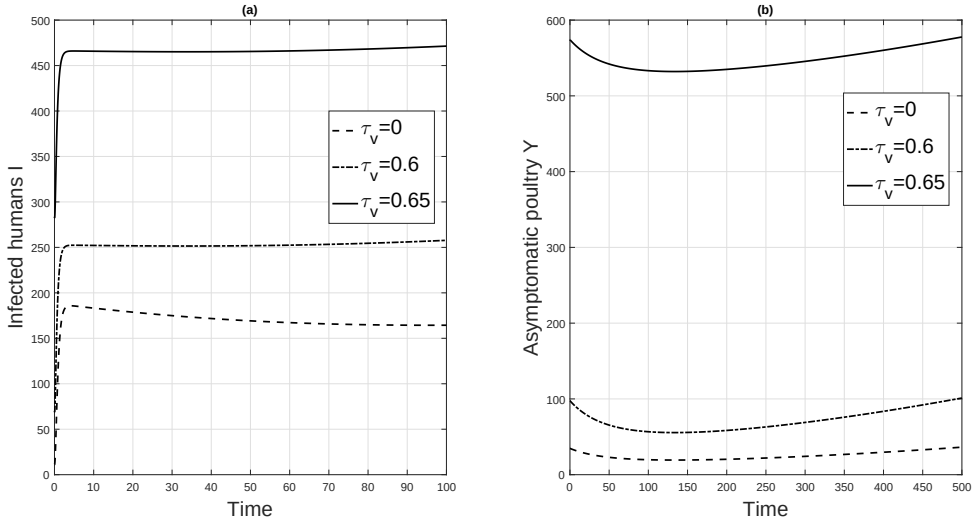

Figure 11. Simulation results showing the impact of transmission rate τ_v: On infected humans (a); On asymptomatic poultry (b). Both graphs are sketched with the following parameters: q=0, A=100, β_v=1.7143, β_e=0.002, d=1/72, α=0.001, B=1.5,a=1, γ=0.9, ρ=0.001, ϵ=1, δ=0.00025641, $\kappa = 10^6$,ξ=35, τ_e=0.1, $\phi_2 = 10^4$.

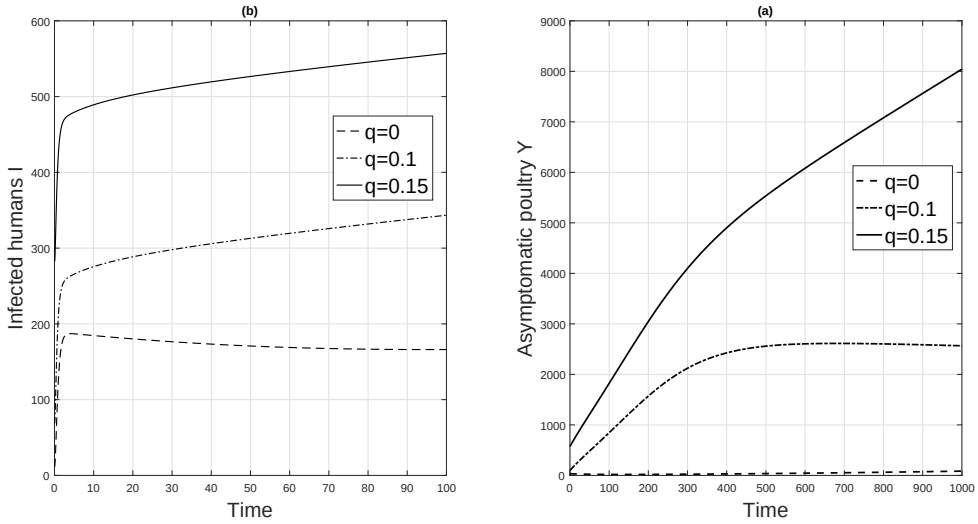

Figure 12. Variation of Y and I with time showing the effect of the parameter q. Both graphs are sketched with the following parameters: A=100, β_v=1.7143, β_e=0.002, d=1/72, α=0.001, B=1.5, a=1, γ=0.9, ρ=0.001, τ_v=0.6, ϵ=1, δ=0.00025641, $\kappa = 10^6$, ξ=35, τ_e=0.1, $\phi_2 = 10^4$.

6. Conclusion and discussion.

This paper has formulated and analyzed a new mathematical model for the dynamical transmission of AIV which specifically incorporates the following usually neglected,yet important features:
(i) The indirect transmission via the environment.
(ii) The recruitment of imported infected poultry and the spillover phenomenon from poultry to human beings. A qualitative, quantitative, as well as sensitivity analysis of the model and variables have been presented using a range of techniques and methods.

From the qualitative point of view, the ingenious construction of suitable Lyapunov functions, the application of LaSalle Invariance Priniciple, the intuitive selection of Lyapunov-stable matrices and the application of Poincaré-Bendixson theorem have been judiciously

combined where necessary to deal with global asymptomatic stability of equilibria. The explicitly computed basic reproduction number $\mathcal{R}_0$ served to establish the threshold dynamic of the model and to prove the transcritical bifurcation of the system. The calculations of elasticity indexes were to study the sensitivity of $\mathcal{R}_0$ with respect to model parameters. In a more precise manner, we have shown:

(1) If the importation of infected poultry is banned ($q = 0$), then the basic reproduction number $\mathcal{R}_0$ determines whether the disease will persist in the population or dies out by proving that whenever $\mathcal{R}_0 < 1$, the disease-free equilibrium Z^0 is globally asymptotically stable, while $\mathcal{R}_0 > 1$, ensures existence of a unique globally asymptotically stable endemic equilibrium Z^*. Furthermore, from elasticity indexes of $\mathcal{R}_0$, it was observed that the most effective control measure is to reduce the number of new born poultry and the number of emission rate of virus by asymptomatic poultry.

(2) If the importation of infected poultry is permitted ($q \neq 0$), the unique endemic equilibrium Z^* is simply shifted to the one $\overline{Z}$ for which the asymptomatic component $\overline{Y}$ is larger to Y^* and is unconditionally globally asymptotically stable. This highlighted the fact that the recruitment of infected poultry worsens the endemic level of AIV during outbreaks.

As far as the quantitative and computational perspectives are concerned, we have constructed a dynamical consistent NSFD scheme, to overcome the undesirable situation for which the classical Rung-Kutta numerical scheme exhibit negative solutions. In addition, we have proved analytically and numericaly that the proposed NSFD scheme preserves the boundedness of solutions, as well as the number, values and local stability of equilibria.

A reasonable extension of this paper on which we are already working, despite the mathematical analysis complications, will be to add the convection/advection and diffusion of viruses into air, as well as the displacement of poultry in the farms.

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References

[1] Peiris JSM, De Jong MD, Guan Y. Avian influenza virus (H5N1) : a threat to human health. Clin. Microbiol; (2007). Rev. 20:2, 243-267.

[2] Killingley B, Nguyen-Van-Tam J. Routes of influenza transmission. Influenza Other Respir. Viruses; (2013). 7, 42–51.

[3] Dent JE, Kao RR, Kiss IZ, Hyder K, Arnold M. Contact structures in the poultry industry in Great Britain: exploring transmission routes for a potential avian influenza virus epidemic. BMC Vet; (2008). Res 4:27, 1746–6148.

[4] Chong KC, Wang X, Liu S, Cai J, Su X, Zee BC, Tam G, Wang MH, Chen E. Interpreting the transmissibility of the avian influenza A(H7N9) infection from 2013 to 2015 in Zhejiang Province, China. Epidemiol.Infect; (2016). 144, 1584–1591.

[5] Iwami S, Takeuchi Y, Korobeinikov A, Liu X. Prevention of avian influenza epidemic: What policy should we choose?. J.Theor.Biol; (2008). 252, 732–741.

[6] Iwami S, Takeuchi Y, Liu X. Avian-human influenza epidemic model. Math.Biosci; (2007). 207, 1–25.

[7] Iwami S, Takeuchi Y, Liu X. Avian flu pandemic: Can we prevent it?. J.Theor.Biol; (2009). 257, 181–190.

[8] Bourouiba L, Teslya A, Wu J. Highly pathogenic avian influenza outbreak mitigated by seasonal low pathogenic strains: Insights from dynamic modeling. J.Theor.Biol; (2011). 271, 181–201.
[9] Liu Z, Fang CT. A modeling study of human infections with avian influenza A H7N9 virus in mainland China. Int.J.Infect.Dis; (2015). 41, 73–78.
[10] Chen Y, Wen Y. Global dynamic analysis of a H7N9 avian-human influenza model in an outbreak region. J.Theor.Biol; (2015). 367, 180–188.
[11] Liu S, Ruan S, Zhang X. On avian influenza epidemic models with time delay. Theory.Biosc; (2015). 134, 75–82.
[12] Guo S-M, Wang J, Ghost M and Xue-Zhi: Analysis of Avian Influenza A (H7N9) model based on the low pathogenicity in poultry. J.Biol.Syst; (2017). 25:2, 1–16.
[13] Anguelov R and Lubuma JM-S. Contributions to the mathematics of the nonstandard finite difference method and applications. Numer. Methods Partial Differ. Equ; (2001). 17, 518–543.
[14] Anguelov R, Dumont Y, Lubuma JM-S, and Shillor M: Comparison of some standard and nonstandard numerical methods for the MSEIR Epidemiological Model, in Proceedings of the International Conference of Numerical Analysis and Applied Mathematics, (2009) September, Crete, Greece, 18–22, Volume 2, American Institute of Physics Conference Proceedings - (AIP 1168), T. Simos, G. Psihoyios, and Ch. Tsitouras, eds, 1209–1212.
[15] Dimitrov DT and Kojouharov HV. Positive and elementary stable nonstandard numerical methods with applications to predator-prey models. J. Comput. Appl. Math; (2006). 189, 98-108.
[16] Gumel AB, Patidar KC and Spiteri RJ. Asymptotically consistent non-standard finite difference methods for solving mathematical models arising in population biology, in Advances in the Applications of Nonstandard Finite Difference Schemes,(2005). Mickens RE, ed. World Scientific Publishing, Hackensack, NJ, 385–421.
[17] Mickens RE. Advances in the Applications of Nonstandard Finite Difference Schemes. World Scientific, Singapore; (2005).
[18] Mickens RE. Applications of Nonstandard Finite Difference Schemes. World Scientific, Singapore; (2000).
[19] Mickens RE. Nonstandard Finite Difference Models of Differential Equations. World Scientific, Singapore; (1994).
[20] Mickens RE. Nonstandard finite difference schemes for differential equations. J. Diff. Equ. Appl; (2002). 8, 823–847.
[21] Van Den Driessche P, Watmough J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of desease transmission. Math.Biosci; (2002). 180, 29–48.
[22] Gjorgjieva J, Smith K, Chowell G, Sanchez F, Snyder J and Castillo-Chavez C. The role of vaccination in the control of SARS. Math.Biosci.Eng; (2005). 2, 753–769.
[23] Castillo-Chavez C and Song B. Dynamical model of tuberclosis and their applications. Math.Biosci.Eng; (2004). 1, 361–404.
[24] LaSalle JP. The Stability of Dynamical Systems, Regional Conference Series in Applied Mathematics. SIAM, Philadelphia. (1976).
[25] Vidyasagar M. Decomposition techniques for large-scale systems with nonadditive interactions Stability and stabilizability. IEEE Trans. Autom.Control; (1980). 25, 773–779.
[26] Cross GW. Three types of matrix stability. Linear Algebra Appl; (1978). 20, 253–263.
[27] Goh BS. Global stability in two species interactions. J.Math.Biol; (1976). 3, 313–318.
[28] Redheffer R. Volterra multipliers ii. SIAM J.Alg.Disc.Math; (1985). 6, 612–623.
[29] Berge T, Lubuma JM-S, Moremedi GM, Morris N, Kondera-Shava R. A simple mathematical model for Ebola in Africa. J.Biol.Dynamics; (2017). 11:1, 42–74.
[30] Castillo-Chavez C, et al. On the computation of Ro and its role on global stability ,(2001).
[31] Liu S, Pang L, Ruan S, Zhang X. Global dynamics of avian influenza epidemic models with psychological effect. Comput.Math.Methods.Med; (2015). Article ID 913726.
[32] Manyombe MLM, Mbang J, Lubuma J, Tsanou B. Global dynamics of a vaccination model for infectious deseases with asymptomatic carriers. Math.Biosci.Eng; (2016). Volume 13, Number 4, 813–840.
[33] Meksianis Z, Bertha S, Tambaru D. A Nonstandard Finite Difference Scheme for Water-Related Disease Mathematical Model. Appl. Math. Inf. Sci; (2019). 13, No. 4, 545–551.
[34] Mukandavire Z, Liao S, Wang J, Gaff H, Smith DL, Morris JG. Estimating the reproductive numbers for the 2008-2009 cholera outbreaks in Zimbabwe. P. Nat. Acad.Sci.USA; (2011). 108, 8767–8772.
[35] Tsanou B, Bowong S, Lubuma J, Mbang J. Assessing the impact of the environmental contam-

ination on the transmission of Ebola virus disease (EVD). J.Appl.Math.Comput; (2017). 55:1-2, 205–243.