
*Research article***Kayo–Kengne–Akgül fractional multi-delay model for avian influenza: Hopf bifurcation and optimal control analysis****Saeed M. Alamry***

Department of Mathematics and Statistics, College of Science, Taif University, P. O. Box 11099, Taif 21944, Saudi Arabia

* **Correspondence:** Email: sm.alamri@tu.edu.sa.

Abstract: This work develops a Kayo–Kengne–Akgül (KKA) fractional multi-delay framework for modeling avian-influenza transmission. The proposed formulation considers the interaction between bird and human populations while accounting for two biologically meaningful delays: an avian infectious-pressure delay and a human transmission-response delay. By incorporating the KKA fractional derivative, the model is able to represent memory-dependent effects that influence the spread of infection. The well-posedness of the system is verified by proving positivity and boundedness of solutions. The disease-free and endemic steady states are obtained, and the threshold quantity governing disease persistence is computed through the next-generation matrix method. Because the equilibria do not explicitly depend on time, their algebraic forms remain consistent with those of the corresponding classical delayed model. Nevertheless, the fractional operator alters the stability criteria and the Hopf bifurcation analysis through the associated KKA characteristic equation. In addition, an optimal-control formulation is presented by introducing avian biosecurity and contact-reduction measures, infected-bird culling, and awareness-based intervention as control measures. The theoretical findings are supported using a KKA-adapted Adams–Bashforth numerical method. Finally, numerical experiments demonstrate how the fractional order, delay parameters, and control interventions affect the evolution of avian influenza.

Keywords: avian influenza; Kayo–Kengne–Akgül derivative; fractional-order model; time delay; Hopf bifurcation; optimal control; Adams–Bashforth scheme

Mathematics Subject Classification: 34D20, 34K20, 34K60, 92C60, 92D45

1. Introduction

Avian influenza represents an important zoonotic disease with considerable implications for poultry populations and human health. Among its different subtypes, H7N9 has received significant attention

because it is mainly associated with birds but can also cause severe infection in humans. Although this virus is often reported as low pathogenic in poultry, human infections may lead to serious respiratory illness and even pandemic-level concerns [1,2]. Different preventive and control actions have been used to mitigate avian influenza outbreaks, including restrictions on live poultry markets, removal of infected birds, isolation procedures, vaccination programs, and public health education [3–5]. Nevertheless, the disease dynamics are highly intricate, as transmission is influenced by avian-human interactions, environmental reservoirs, latent periods, and delayed treatment responses.

Mathematical modeling plays an important role in studying infectious-disease spread and in comparing different prevention and control policies. Since the seminal contribution of Kermack and McKendrick [6], compartmental epidemic models have been widely adopted to represent disease evolution through interacting population classes and to determine critical threshold measures such as the basic reproduction number. In avian-influenza research, deterministic models have been used to explore infection pathways linking birds and humans. Chong et al. [7] analyzed an avian-influenza model involving a half-saturated incidence rate, while Iwami et al. [8] developed an avian-human influenza epidemic framework for studying interspecies transmission. More recent studies have incorporated logistic growth, nonlinear incidence, live-bird-market transmission, and different intervention strategies to better represent the biological and epidemiological features of avian influenza [9,10].

In epidemic modeling, delay terms are introduced to reflect the fact that many biological and clinical events do not take place immediately. For avian influenza, these delays may correspond to the incubation stage in birds, the period needed for symptom development, diagnostic lag, or delayed treatment in humans. Delay differential equations are therefore effective for incorporating latent and response intervals into the disease model. It is well known that the presence of delays can modify equilibrium stability and may produce sustained oscillations via Hopf bifurcation [11–13]. In particular, multi-delay models provide a more realistic framework when more than one biological delay affects the disease dynamics. However, most existing delayed avian-influenza models are formulated using integer-order derivatives, which describe only the instantaneous rate of change and do not fully capture memory-dependent effects in disease transmission.

Fractional-order modeling has become an important tool for studying systems whose evolution is influenced by historical information. In epidemiology, such models are particularly useful because they can account for memory-dependent effects in infection dynamics, providing greater flexibility than traditional integer-order models [14–16]. In biological systems, memory effects are important because the present transmission rate may depend on previous exposure, past infection levels, delayed immune response, environmental persistence, and the cumulative effect of control measures. Therefore, fractional derivatives offer a more realistic mathematical framework for infectious disease modeling, especially when the disease process involves nonlocal temporal behavior.

Recent developments across nonlinear dynamical systems, control theory, fractional calculus, and biomedical modeling have demonstrated the importance of time-dependent effects and advanced analytical techniques in complex systems. Kandil analyzed Hopf curves arising from time-delayed active control in a two-degree-of-freedom nonlinear dynamical system [17]. Kandil and El-Ganaini subsequently examined the influence of time delay on the control of rotating-blade vibrations [18]. The performance of a time-delayed proportional-derivative controller for rotating-blade vibrations was further investigated by Kandil and El-Gohary [19]. Eissa et al. studied a nonlinear

magnetic-levitation system controlled using a time-delayed proportional-derivative controller [20]. Chen et al. considered finite-time topology identification in delayed complex dynamical networks, further demonstrating the significance of delayed interactions in complex systems [21]. From the perspective of fractional calculus, Li et al. established results concerning existence, uniqueness, and continuous dependence on initial and final values for Liouville–Caputo fractional difference equations [22]. In biomedical research, Zhuang et al. evaluated the efficacy and mortality associated with ceftazidime/avibactam-based treatment regimens for carbapenem-resistant Gram-negative bacterial infections [23]. Zhang et al. analyzed reported cases of hemophagocytic lymphohistiocytosis following COVID-19 vaccination [24]. Wang et al. investigated immune and inflammatory microenvironment characteristics in noncancer end-stage liver disease [25]. In mathematical epidemiology, fractional-order approaches have also been increasingly applied to infectious-disease dynamics. Fractional modeling has been employed to investigate Lassa fever transmission and its associated memory-dependent behavior [26]. Related fractional formulations have been developed for the study of Zika virus dynamics [27]. Computational and parameter-estimation techniques have also been incorporated into fractional models of typhoid fever [28]. Fractional-order modeling combined with computational methods has further been applied to poliomyelitis transmission dynamics [29]. Hybrid fractional and machine-learning approaches have been considered for the analysis of Zika virus transmission [30]. Similar computational frameworks have also been employed for rabies transmission dynamics [31]. These studies collectively demonstrate the broad applicability of delay mechanisms, fractional memory, stability analysis, nonlinear control, and computational techniques in the investigation of complex dynamical and biological systems. The Caputo, Caputo–Fabrizio, Atangana–Baleanu–Caputo, and Kayo–Kengne–Akgül (KKA) fractional operators differ primarily in the memory kernels used to weight previous system states. The Caputo derivative employs a power-law kernel, the Caputo–Fabrizio derivative uses a nonsingular exponential kernel, the Atangana–Baleanu–Caputo derivative is based on a nonsingular Mittag–Leffler kernel, and the KKA derivative employs a generalized exponential kernel. In the present model, the fractional order ρ regulates the temporal weighting of previous epidemic states through the KKA memory kernel.

The KKA memory mechanism and the discrete delays have distinct roles in the proposed avian-influenza framework. The fractional operator incorporates distributed dependence on the previous epidemic trajectory, whereas τ_1 and τ_2 represent specific transmission-related time lags. The associated KKA fractional integral and KKA-adapted Adams–Bashforth discretization provide the analytical and numerical framework used in this study [32]. Consequently, the combined formulation permits the effects of fractional memory and discrete delays on stability, Hopf bifurcation, and epidemic control to be investigated within a unified model.

Existing fractional avian-influenza studies provide important foundations for the present work but generally address different modeling questions. Ye et al. [33] developed a Caputo fractional avian-human influenza system with logistic avian growth and established well-posedness, equilibrium existence, and stability, but their formulation did not incorporate the multiple discrete-delay mechanisms considered here. More recently, a Caputo fractional avian-influenza model for poultry farms incorporated data-driven analysis, emphasizing parameter estimation and real-data interpretation. Other recent bird-flu work has incorporated vaccination and treatment within a fractional framework together with spectral and machine-learning-based numerical techniques. In contrast, the present study focuses on the interaction of a generalized KKA memory kernel with

coupled avian-human transmission, multiple explicit delays, delay-dependent spectral stability switching, sensitivity analysis, and optimal control. Thus, its contribution is complementary rather than a replacement for existing Caputo or other fractional avian-influenza formulations.

Motivated by the above studies, we construct a KKA fractional version of a multi-delay avian-influenza model. In this formulation, the bird population is classified into two epidemiological groups, namely susceptible birds and infected birds, whereas the human population is represented by susceptible, exposed, infected, and recovered classes. The model includes two separate delay terms. The first represents a lag in avian infectious pressure in the bird-to-bird transmission pathway, while the second represents a human transmission-response lag in the human-to-human infection pathway. After substituting the classical integer-order derivative with the KKA fractional operator of order ρ , the resulting system is able to reflect both delayed responses and memory-driven transmission behavior.

The principal novelty of the present study lies in the unified integration of KKA fractional memory, multiple epidemiological delays, coupled avian-human transmission, and intervention strategies within a single mathematical framework. In contrast to classical integer-order delay models, where historical dependence is represented only through discrete lagged states, the proposed formulation simultaneously incorporates nonlocal KKA memory and distinct delays associated with avian and human transmission processes. The model explicitly couples avian-avian, avian-human, and human-human infection pathways, thereby allowing the fractional order ρ and the delay parameters to jointly regulate transient behavior and oscillatory dynamics. From the analytical perspective, the study establishes positivity and boundedness of solutions, characterizes the biologically feasible disease-free and endemic equilibria, and derives the threshold quantity $R_0 = \max R_1, R_2$ using the next-generation matrix approach. A further contribution is the development of zero-delay stability conditions and delay-induced Hopf-type spectral switching through the KKA characteristic equation, providing insight into how fractional memory and multiple delays influence the loss or recovery of stability. In addition, a delayed fractional optimal-control problem is formulated using three biologically meaningful interventions: avian biosecurity measures that reduce effective bird-to-bird transmission, culling of infected birds, and educational awareness programs that decrease avian-to-human exposure. Finally, a KKA-adapted Adams–Bashforth numerical framework is employed to validate the theoretical results and to quantify the individual and combined effects of fractional order, delays, and control measures. These developments provide an integrated theoretical and computational framework for understanding how distributed fractional memory and distinct epidemiological delays interact to shape avian-human influenza transmission and its control.

The structure of the paper is arranged as follows. Section 2 establishes the main qualitative properties of the model, including positivity, boundedness, equilibrium solutions, and the basic reproduction number. Section 3 is devoted to the analysis of local stability and Hopf bifurcation for different delay scenarios. The sensitivity of the basic reproduction number is examined in Section 4. Section 5 develops the optimal control formulation and studies the corresponding control system. Section 6 presents the numerical discretization together with simulation outcomes. Finally, Section 7 discusses the biological meaning of the results and provides the concluding remarks.

2. Model formulation

In this section, a KKA fractional multi-delay epidemic model is developed to describe the spread of avian influenza. The avian population is represented by a susceptible-infected (SI) compartmental structure, whereas the human population is described using a susceptible-exposed-infected-recovered (SEIR) compartmental framework. The KKA fractional operator is incorporated to account for memory-dependent behavior in the transmission process. In addition, two discrete transmission-related delays are included: an avian infectious-pressure delay and a human transmission-response delay.

Let $N_a(t)$ and $N_h(t)$ represent the total numbers of birds and humans at time t , respectively. The bird population consists of susceptible birds $S_a(t)$ and infected birds $I_a(t)$, and hence $N_a(t) = S_a(t) + I_a(t)$. The human population is classified into susceptible individuals $S_h(t)$, exposed individuals $E_h(t)$, infected individuals $I_h(t)$, and recovered individuals $R(t)$, so that $N_h(t) = S_h(t) + E_h(t) + I_h(t) + R(t)$. All recruited birds and humans are assumed to join the susceptible compartments. Transmission can occur within the bird population, from infected birds to humans, and within the human population. In this formulation, infected birds remain infectious without recovery, whereas infected humans may recover and move to the immune class.

To formulate the fractional version of the model, we first recall the KKA fractional derivative. Let $0 < \rho \leq 1$ and let f be sufficiently smooth. Then the KKA derivative is expressed as

$${}_0^{KKA}D_t^\rho f(t) = \frac{KA(\rho)}{1-\rho} \int_0^t f'(\xi) e_{\rho,\rho} \left(-\frac{\rho}{1-\rho}(t-\xi) \right) d\xi,$$

where $KA(\rho)$ denotes the KKA normalization function. Following Kayo and Akgül [32], it is taken as

$$KA(\rho) = 1 - \rho + \frac{\rho}{\Gamma(\rho)}, \quad 0 < \rho \leq 1,$$

with the endpoint normalization $A(0) = KA(1) = 1$. The function $e_{\rho,\rho}(\cdot)$ denotes the generalized exponential kernel, which for $0 < \rho < 1$ is given by

$$e_{\rho,\rho} \left(-\frac{\rho}{1-\rho}s \right) = \sum_{j=0}^{\infty} \frac{\left(-\frac{\rho}{1-\rho} \right)^j}{\Gamma(\rho j + \rho)} s^{\rho j + \rho - 1}, \quad s > 0.$$

Therefore, in the present model,

$$e_{\rho,\rho} \left(-\frac{\rho}{1-\rho}(t-\xi) \right) = \sum_{j=0}^{\infty} \frac{\left(-\frac{\rho}{1-\rho} \right)^j}{\Gamma(\rho j + \rho)} (t-\xi)^{\rho j + \rho - 1}.$$

The limiting case $\rho = 1$ is interpreted through the corresponding classical integer-order derivative. These definitions make explicit the normalization and memory kernel employed throughout the analytical and numerical developments.

Moreover, the corresponding fractional integral is defined by

$${}_0^{KKA}I_t^\rho f(t) = \frac{\rho}{KA(\rho)} \int_0^t f(\xi) d\xi + \frac{1-\rho}{KA(\rho)\Gamma(1-\rho)} \int_0^t f(\xi)(t-\xi)^{-\rho} d\xi.$$

Here, the order ρ characterizes the memory intensity of the fractional avian-influenza system. When ρ approaches the classical value $\rho = 1$, the proposed KKA fractional model coincides with the standard integer-order delayed formulation. Accordingly, ρ and the discrete delays have distinct interpretations. The parameter ρ determines the distributed memory of the entire previous trajectory through the KKA kernel, whereas τ_1 and τ_2 represent specific epidemiological lags. Thus, decreasing ρ modifies how strongly previous epidemic states influence the present dynamics without introducing an additional epidemiological compartment or a new transmission pathway.

We consider two discrete time delays associated with delayed transmission effects. The parameter $\tau_1 \geq 0$ represents an avian infectious-pressure delay: The current incidence among susceptible birds depends on the infectious-bird population at the earlier time $t - \tau_1$. Since the avian subsystem contains no exposed class, τ_1 is not interpreted as a true incubation-stage transition. The parameter $\tau_2 \geq 0$ represents a human transmission-response delay, so that the current force of human-to-human infection depends on the infectious-human population at time $t - \tau_2$. This formulation retains two biologically meaningful delay mechanisms while ensuring that delayed quantities do not appear as direct negative removals from a current population compartment. Thus, the avian-influenza model under the KKA fractional framework takes the following form:

$$\begin{aligned}
 {}_0^{KKA}D_t^\rho S_a(t) &= mS_a(t) \left(1 - \frac{S_a(t)}{k}\right) - \beta_1 S_a(t)I_a(t - \tau_1) - d_1 S_a(t), \\
 {}_0^{KKA}D_t^\rho I_a(t) &= \beta_1 S_a(t)I_a(t - \tau_1) - (\mu_a + \delta + d_2)I_a(t), \\
 {}_0^{KKA}D_t^\rho S_h(t) &= \Lambda - [\beta_2 I_h(t - \tau_2) + \beta_3 I_a(t)]S_h(t) - \mu_h S_h(t), \\
 {}_0^{KKA}D_t^\rho E_h(t) &= (\beta_2 I_h(t - \tau_2) + \beta_3 I_a(t))S_h(t) - (\mu_h + k_1 + \sigma)E_h(t), \\
 {}_0^{KKA}D_t^\rho I_h(t) &= \sigma E_h(t) - (\mu_h + \alpha + \gamma)I_h, \\
 {}_0^{KKA}D_t^\rho R(t) &= k_1 E_h(t) + \gamma I_h(t) - \mu_h R(t).
 \end{aligned} \tag{2.1}$$

The initial history functions are assumed to satisfy

$$S_a(\theta) = \Psi_1(\theta), \quad I_a(\theta) = \Psi_2(\theta), \quad S_h(\theta) = \Psi_3(\theta), \quad E_h(\theta) = \Psi_4(\theta), \quad I_h(\theta) = \Psi_5(\theta), \quad R(\theta) = \Psi_6(\theta), \tag{2.2}$$

where $\Psi_i \in C([-\tau, 0], \mathbb{R}_+)$, $\Psi_i(\theta) > 0$, $i = 1, 2, \dots, 6$, with $\tau = \max\{\tau_1, \tau_2\}$. In the present formulation, the delay τ_2 acts exclusively through the human-to-human transmission pathway. Specifically, the current force of human infection depends on the previous infectious-human population $I_h(t - \tau_2)$, whereas recovery from the infected-human compartment occurs at the current rate $\gamma I_h(t)$. Thus, τ_2 is interpreted as a human transmission-response delay rather than as a delayed removal or recovery mechanism. This formulation preserves the quasi-positivity of the epidemiological system without requiring an additional viability condition on the delayed history.

All model parameters are taken to be non-negative. The avian population grows with intrinsic rate m , while k denotes its environmental carrying capacity. Human recruitment is represented by Λ . The parameters β_1 , β_2 , and β_3 describe, respectively, transmission among birds, transmission among humans, and infection of susceptible humans due to contact with infected birds. Natural mortality in birds and humans is given by μ_a and μ_h , respectively. The additional mortality caused by the disease is denoted by δ for infected birds and by α for infected humans. The removal rates of susceptible and

infected birds through culling are represented by d_1 and d_2 , respectively. Furthermore, σ denotes the rate at which exposed humans become infectious, k_1 represents the recovery rate of exposed humans, and γ denotes the recovery rate of infected humans, while τ_2 represents the human transmission-response delay appearing in the force of infection. Since the recovered class does not influence the dynamics of the remaining compartments, the subsequent theoretical analysis can be carried out using the following reduced KKA fractional system:

$$\begin{aligned} {}_0^{KKA}D_t^\rho S_a(t) &= mS_a(t) \left(1 - \frac{S_a(t)}{k}\right) - \beta_1 S_a(t) I_a(t - \tau_1) - d_1 S_a(t), \\ {}_0^{KKA}D_t^\rho I_a(t) &= \beta_1 S_a(t) I_a(t - \tau_1) - a_1 I_a(t), \\ {}_0^{KKA}D_t^\rho S_h(t) &= \Lambda - [\beta_2 I_h(t - \tau_2) + \beta_3 I_a(t)] S_h(t) - \mu_h S_h(t), \\ {}_0^{KKA}D_t^\rho E_h(t) &= (\beta_2 I_h(t - \tau_2) + \beta_3 I_a(t)) S_h(t) - (a_2 + \sigma) E_h(t), \\ {}_0^{KKA}D_t^\rho I_h(t) &= \sigma E_h(t) - a_3 I_h(t), \end{aligned} \quad (2.3)$$

where $a_1 = \mu_a + \delta + d_2$, $a_2 = \mu_h + k_1$, and $a_3 = \mu_h + \alpha + \gamma$.

The system (2.3) will be used to investigate positivity, boundedness, equilibrium points, the basic reproduction number, stability, and Hopf bifurcation. The full system (2.1) will be used later when the optimal control problem is formulated.

2.1. Positivity, boundedness, and equilibrium analysis

In this subsection, we examine the biological validity of the proposed KKA fractional-order avian-influenza model. More precisely, we show that the solutions of the system remain non-negative and ultimately bounded, which ensures that the model is meaningful from an epidemiological point of view. After that, the disease-free steady state, the basic reproduction number, and the endemic steady states are obtained. Since the KKA fractional derivative vanishes for constant functions, the equilibrium equations keep the same algebraic form as those of the associated integer-order multi-delay system. Nevertheless, the fractional order ρ plays an important role in the dynamic behavior of the model, especially in relation to transient responses, rates of convergence, and stability characteristics.

Lemma 2.1. *Let $y(t)$ be a non-negative continuous function satisfying a KKA fractional differential inequality of the form ${}_0^{KKA}D_t^\rho y(t) \leq a - by(t)$, $0 < \rho \leq 1$, where $a \geq 0$, $b > 0$, and $y(0) \geq 0$. Under the order-preserving property of the corresponding KKA Volterra integral formulation, comparison with the scalar problem*

$${}_0^{KKA}D_t^\rho z(t) = a - bz(t), \quad z(0) = y(0)$$

yields $0 \leq y(t) \leq \max\{y(0), \frac{a}{b}\}$, $t \geq 0$. Consequently,

$$\limsup_{t \rightarrow \infty} y(t) \leq \frac{a}{b}.$$

This scalar result will be used below to obtain invariant upper bounds for the aggregated avian and human populations.

Theorem 2.2. Assume that all model parameters are non-negative, $m > d_1$, and that the initial history functions are non-negative. Then the vector field associated with system (2.3) is quasi-positive on the non-negative state space. Consequently, solutions initiated from non-negative histories remain non-negative for all $t \geq 0$. Moreover, the solutions are ultimately bounded in the positively invariant region specified below: $[\beta_2 I_h(t - \tau_2) + \beta_3 I_a(t)] S_h(t) \geq 0$. Finally, because recovery is non-delayed,

$$F_5|_{I_h=0} = \sigma E_h(t) \geq 0. \quad (2.4)$$

Hence, the vector field points inward or is tangent to the non-negative state cone at every coordinate boundary, and no additional viability condition involving $I_h(t - \tau_2)$ is required.

Proof. Define the vector of state variables by

$$X(t) = (S_a(t), I_a(t), S_h(t), E_h(t), I_h(t))^T.$$

Using this notation, the KKA fractional system (2.3) can be rewritten in the abstract form

$${}_0^{KKA} D_t^\rho X(t) = F(t, X_t),$$

where

$$X_t = X(t + \theta), \quad -\tau \leq \theta \leq 0$$

denotes the delayed history of the solution. By operating on both sides with the corresponding KKA fractional integral, the model is transformed into the equivalent Volterra integral equation

$$X(t) = X(0) + \frac{\rho}{KA(\rho)} \int_0^t F(s, X_s) ds + \frac{1-\rho}{KA(\rho)\Gamma(1-\rho)} \int_0^t (t-s)^{-\rho} F(s, X_s) ds.$$

Since the kernels appearing in the KKA Volterra representation are non-negative for $0 < \rho \leq 1$, preservation of the non-negative state cone can be examined through the inward-pointing behavior of the vector field on its boundary. For the present delay system, this condition is evaluated with respect to the current state together with its delayed history. Because the delayed state variables enter the corresponding boundary components with non-negative coefficients, quasi-positivity can be verified on the full non-negative history space.

From the first equation of (2.3), when $S_a(t) = 0$, we obtain

$$F_1(t, X_t)|_{S_a=0} = 0.$$

Similarly, when $I_a(t) = 0$,

$$F_2(t, X_t)|_{I_a=0} = \beta_1 S_a(t) I_a(t - \tau_1) \geq 0.$$

For the susceptible human class, when $S_h(t) = 0$,

$$F_3(t, X_t)|_{S_h=0} = \Lambda \geq 0.$$

For the exposed human class, when $E_h(t) = 0$,

$$F_4(t, X_t)|_{E_h=0} = (\beta_2 I_h(t - \tau_2) + \beta_3 I_a(t)) S_h(t) \geq 0.$$

Finally, when $I_h(t) = 0$, the fifth component of the vector field satisfies

$$F_5(t, X_t)|_{I_h=0} = \sigma E_h(t) \geq 0.$$

Therefore, $F_i(t, X_t) \geq 0$ on each coordinate boundary whenever the remaining current and delayed state variables are non-negative. Hence, the vector field is quasi-positive on the non-negative history space, and no additional viability condition involving $I_h(t - \tau_2)$ is required. Consequently, $S_a(t), I_a(t), S_h(t), E_h(t), I_h(t) \geq 0, t \geq 0$.

To prove boundedness, consider first the aggregate avian population $N_a(t) = S_a(t) + I_a(t)$. Using the first two equations of (2.3) and the linearity of the KKA derivative, one obtains

$${}_0^{KKA}D_t^\phi N_a(t) = mS_a(t) \left(1 - \frac{S_a(t)}{k}\right) - d_1 S_a(t) - a_1 I_a(t).$$

Since

$$mS_a(t) \left(1 - \frac{S_a(t)}{k}\right) \leq \frac{mk}{4},$$

we obtain

$${}_0^{KKA}D_t^\phi N_a(t) \leq \frac{mk}{4} - d_a^* N_a(t), \quad d_a^* = \min\{d_1, a_1\}.$$

If $\phi = \frac{mk}{4}$, then the KKA fractional comparison theorem gives

$$0 \leq N_a(t) \leq \max \left\{ N_a(0), \frac{\phi}{d_a^*} \right\}.$$

Therefore,

$$\limsup_{t \rightarrow \infty} N_a(t) \leq \frac{\phi}{d_a^*}.$$

Next, define the reduced human population by $N_h(t) = S_h(t) + E_h(t) + I_h(t)$. Adding the equations corresponding to $S_h(t)$, $E_h(t)$, and $I_h(t)$ in (2.3) gives

$${}_0^{KKA}D_t^\phi N_h(t) = \Lambda - \mu_h S_h(t) - a_2 E_h(t) - a_3 I_h(t).$$

Since $a_2 = \mu_h + k_1 \geq \mu_h$, $a_3 = \mu_h + \alpha + \gamma \geq \mu_h$, we obtain

$${}_0^{KKA}D_t^\phi N_h(t) \leq \Lambda - \mu_h (S_h(t) + E_h(t) + I_h(t)) = \Lambda - \mu_h N_h(t).$$

Using the comparison principle for KKA fractional differential inequalities, we deduce

$$0 \leq N_h(t) \leq \max \left\{ N_h(0), \frac{\Lambda}{\mu_h} \right\}.$$

Therefore,

$$\limsup_{t \rightarrow \infty} N_h(t) \leq \frac{\Lambda}{\mu_h}.$$

Hence, all solution variables of system (2.3) are bounded in the positively invariant region

$$\Omega = \left\{ (S_a, I_a, S_h, E_h, I_h) \in \mathbb{R}_+^5 : 0 \leq N_a(t) \leq \frac{\phi}{d_a^*}, \quad 0 \leq N_h(t) \leq \frac{\Lambda}{\mu_h} \right\}.$$

This completes the proof. $\square$

The disease-free state of the KKA fractional system (2.3) is found by eliminating all infected compartments, that is, $I_a = E_h = I_h = 0$. Hence, the disease-free equilibrium is given by $E_0 = (S_a^0, 0, S_h^0, 0, 0)$, where

$$S_a^0 = \frac{k(m - d_1)}{m}$$

and

$$S_h^0 = \frac{\Lambda}{\mu_h}.$$

The threshold quantity associated with the spread of the disease is $\mathcal{R}_0 = \max\{\mathcal{R}_1, \mathcal{R}_2\}$, with

$$\mathcal{R}_1 = \frac{\beta_1 k(m - d_1)}{a_1 m}$$

and

$$\mathcal{R}_2 = \frac{\beta_2 \Lambda \sigma}{a_3 \mu_h (a_2 + \sigma)}.$$

Here, $a_3 = \mu_h + \alpha + \gamma$. It is worth noting that the fractional order ρ is not explicitly present in $\mathcal{R}_0$, since this threshold parameter is derived from the algebraic linearization of the infected subsystem at the disease-free state. However, the value of ρ still affects the temporal evolution of the solutions, particularly the memory-dependent transient response, convergence behavior, stability, and bifurcation characteristics.

We now compute the endemic equilibrium, denoted by $E^* = (S_a^*, I_a^*, S_h^*, E_h^*, I_h^*)$. At equilibrium, all KKA derivatives vanish and the delayed terms satisfy

$$I_a(t - \tau_1) = I_a^*$$

and

$$I_h(t - \tau_2) = I_h^*.$$

Therefore, the endemic equilibrium satisfies

$$\begin{aligned} 0 &= m S_a^* \left(1 - \frac{S_a^*}{k} \right) - \beta_1 S_a^* I_a^* - d_1 S_a^*, \\ 0 &= (\beta_1 S_a^* - a_1) I_a^*, \\ 0 &= \Lambda - (\beta_2 I_h^* + \beta_3 I_a^*) S_h^* - \mu_h S_h^*, \\ 0 &= (\beta_2 I_h^* + \beta_3 I_a^*) S_h^* - (a_2 + \sigma) E_h^*, \\ 0 &= \sigma E_h^* - a_3 I_h^*, \end{aligned}$$

where $a_1 = \mu_a + \delta + d_2$, $a_2 = \mu_h + k_1$, and $a_3 = \mu_h + \alpha + \gamma$.

For $I_a^* > 0$, the second equation gives

$$S_a^* = \frac{a_1}{\beta_1}.$$

Substituting this expression into the first equilibrium equation yields

$$I_a^* = \frac{(\mathcal{R}_1 - 1)(m - d_1)}{\beta_1 \mathcal{R}_1}.$$

Thus, $I_a^* > 0$, whenever $\mathcal{R}_1 > 1$ and $m > d_1$. From the last equilibrium equation, we have

$$E_h^* = \frac{a_3 I_h^*}{\sigma}.$$

Substituting this into the fourth equilibrium equation gives $-a_2 E_h^* + S_h^* (\beta_2 I_h^* + \beta_3 I_a^*) - \sigma E_h^* = 0$. Hence,

$$S_h^* = \frac{a_3 I_h^* (a_2 + \sigma)}{\sigma (\beta_2 I_h^* + \beta_3 I_a^*)}.$$

Using

$$I_a^* = \frac{(\mathcal{R}_1 - 1)(m - d_1)}{\beta_1 \mathcal{R}_1},$$

we obtain

$$S_h^* = \frac{a_3 I_h^* (a_2 + \sigma)}{\sigma \left(\beta_2 I_h^* + \frac{\beta_3 (\mathcal{R}_1 - 1)(m - d_1)}{\beta_1 \mathcal{R}_1} \right)}.$$

Substituting I_a^* , E_h^* , and S_h^* into the third equilibrium equation gives the following quadratic equation in I_h^* :

$$a_3 \beta_1 \beta_2 \mathcal{R}_1 (a_2 + \sigma) I^2 + B \left(\frac{A}{B} - \mathcal{R}_1 \right) I + \beta_3 \Lambda (1 - \mathcal{R}_1) \sigma (m - d_1) = 0, \quad (2.5)$$

where

$$A = a_3 \beta_3 (-(a_2 + \sigma))(m - d_1)$$

and

$$B = -a_3 \beta_1 \mu_h (a_2 + \sigma) + a_3 \beta_3 (a_2 + \sigma) (d_1 - m) + \beta_1 \beta_2 \Lambda \sigma.$$

The discriminant of (2.5) is

$$\Delta = B^2 \left(\mathcal{R}_1 - \frac{A}{B} \right)^2 + 4a_3 \beta_1 \beta_2 \beta_3 \Lambda (\mathcal{R}_1 - 1) \mathcal{R}_1 \sigma (a_2 + \sigma) (m - d_1).$$

Therefore, the positive roots of (2.5) are

$$I_{h+}^* = \frac{B \left(\mathcal{R}_1 - \frac{A}{B} \right) + \sqrt{\Delta}}{2a_3 \beta_1 \beta_2 \mathcal{R}_1 (a_2 + \sigma)}$$

and

$$I_{h-}^* = \frac{B\left(\mathcal{R}_1 - \frac{A}{B}\right) - \sqrt{\Delta}}{2a_3\beta_1\beta_2\mathcal{R}_1(a_2 + \sigma)}.$$

For each positive value of I_h^* , the corresponding endemic equilibrium is given by

$$E^* = E(I_h^*) = (S_a^*, I_a^*, S_h^*, E_h^*, I_h^*).$$

We denote the two possible endemic equilibria by $E_+^* = E(I_{h+}^*)$ and $E_-^* = E(I_{h-}^*)$.

The biological admissibility of the endemic equilibrium requires particular attention. From the avian equilibrium equations, we obtain

$$S_a^* = \frac{a_1}{\beta_1}$$

and

$$I_a^* = \frac{(R_1 - 1)(m - d_1)}{\beta_1 R_1}.$$

Since $m > d_1$, the condition $I_a^* > 0$ necessarily requires $\mathcal{R}_1 > 1$. Therefore, solutions of the algebraic equation for I_h^* obtained under $\mathcal{R}_1 < 1$ cannot be interpreted as biologically admissible full endemic equilibria when they are coupled with the above expression for I_a^* . In particular, the occurrence of two algebraic roots for I_h^* does not by itself establish backward bifurcation; positivity of all epidemiological compartments must also be satisfied.

For $\mathcal{R}_1 > 1$, the constant term of Eq (2.5) is negative, while its leading coefficient is positive. Hence, the two roots of the corresponding quadratic have opposite signs, and only one positive value of I_h^* is biologically admissible. Consequently, the model possesses a unique fully endemic equilibrium associated with persistent avian infection when $\mathcal{R}_1 > 1$.

This observation is important for the interpretation of the threshold quantity $\mathcal{R}_0 = \max\{\mathcal{R}_1, \mathcal{R}_2\}$. In models exhibiting genuine backward bifurcation, an endemic equilibrium may coexist with the disease-free state even when $\mathcal{R}_0 < 1$, so reducing $\mathcal{R}_0$ below unity is not sufficient for guaranteed elimination. Such a situation generally requires the reproduction number to be reduced below an additional critical value smaller than one. In the present model, however, the positivity conditions do not support such a biologically meaningful backward-bifurcation branch. Thus, $\mathcal{R}_0 = 1$ retains its conventional role as the principal invasion threshold, subject to the KKA spectral stability conditions derived below. The fractional order ρ does not change the algebraic existence conditions of the equilibria, although it modifies their transient behavior, stability regions, and delay-induced bifurcation thresholds.

3. Stability analysis

This section studies the local stability of the disease-free equilibrium of the proposed KKA fractional multi-delay avian-influenza system (2.3). In contrast with the corresponding classical integer-order model, the characteristic equation of the KKA fractional system is not written directly in terms of the eigenvalue λ . Instead, the eigenvalue enters through the KKA spectral function, while the delay terms remain in exponential form.

For $0 < \rho < 1$, define

$$\Theta_\rho(\lambda) = \frac{KA(\rho)}{1 - \rho} \frac{\lambda}{\lambda^\rho + \frac{\rho}{1 - \rho}}. \quad (3.1)$$

For the limiting case $\rho = 1$, we set $\Theta_1(\lambda) = \lambda$. Hence, if the perturbation is chosen in the form $e^{\lambda t}$, then the KKA fractional derivative generates the spectral term $\Theta_\rho(\lambda)$. Therefore, in the local stability analysis, the classical eigenvalue λ is replaced by $\Theta_\rho(\lambda)$, whereas the delay factors are given by $e^{-\lambda\tau_1}$ and $e^{-\lambda\tau_2}$. For $0 < \rho < 1$, the complex fractional power appearing in $\Theta_\rho(\lambda)$ is evaluated on the principal branch, $\lambda^\rho = \exp(\rho \operatorname{Log} \lambda)$ and $\operatorname{Log} \lambda = \ln |\lambda| + i \operatorname{Arg}(\lambda)$, where $-\pi < \operatorname{Arg}(\lambda) \leq \pi$. Thus, if $\lambda = re^{i\theta}$, with $r > 0$ and $-\pi < \theta \leq \pi$, then $\lambda^\rho = r^\rho e^{i\rho\theta}$. Throughout the following stability and bifurcation analysis, this branch convention is used consistently. For $\rho = 1$, $\Theta_1(\lambda) = \lambda$. The disease-free equilibrium of Eq (2.3) is

$$E_0 = (S_a^0, 0, S_h^0, 0, 0),$$

where

$$S_a^0 = \frac{k(m-d_1)}{m} \quad \text{and} \quad S_h^0 = \frac{\Lambda}{\mu_h}.$$

For simplicity, let $a_1 = \mu_a + \delta + d_2$, $a_2 = \mu_h + k_1$, and $a_3 = \mu_h + \alpha + \gamma$.

The basic reproduction number is $\mathcal{R}_0 = \max\{\mathcal{R}_1, \mathcal{R}_2\}$, where

$$\mathcal{R}_1 = \frac{\beta_1 S_a^0}{a_1} = \frac{\beta_1 k(m-d_1)}{a_1 m}$$

and

$$\mathcal{R}_2 = \frac{\beta_2 S_h^0 \sigma}{a_3(a_2 + \sigma)} = \frac{\beta_2 \Lambda \sigma}{a_3 \mu_h(a_2 + \sigma)}.$$

Linearizing system (2.3) around E_0 and substituting perturbations of the form $e^{\lambda t}$, we obtain the characteristic matrix

$$\Delta(\lambda) = \begin{pmatrix} \Theta_\rho + m - d_1 & \beta_1 S_a^0 e^{-\lambda\tau_1} & 0 & 0 & 0 \\ 0 & \Theta_\rho + a_1 - \beta_1 S_a^0 e^{-\lambda\tau_1} & 0 & 0 & 0 \\ 0 & \beta_3 S_h^0 & \Theta_\rho + \mu_h & 0 & \beta_2 S_h^0 e^{-\lambda\tau_2} \\ 0 & -\beta_3 S_h^0 & 0 & \Theta_\rho + a_2 + \sigma & -\beta_2 S_h^0 e^{-\lambda\tau_2} \\ 0 & 0 & 0 & -\sigma & \Theta_\rho + a_3 \end{pmatrix}, \quad (3.2)$$

where $\Theta_\rho = \Theta_\rho(\lambda)$. Therefore, the characteristic equation is

$$\det \Delta(\lambda) = 0. \quad (3.3)$$

After simplification, the characteristic equation becomes

$$(\Theta_\rho + m - d_1)(\Theta_\rho + \mu_h)(\Theta_\rho + a_1 - \beta_1 S_a^0 e^{-\lambda\tau_1}) \times [(\Theta_\rho + a_2 + \sigma)(\Theta_\rho + a_3) - \sigma \beta_2 S_h^0 e^{-\lambda\tau_2}] = 0. \quad (3.4)$$

The factor $e^{-\lambda\tau_2}$ arises exclusively from the delayed human-to-human transmission term $\beta_2 I_h(t - \tau_2) S_h(t)$. The infected-human removal and recovery term remains non-delayed and therefore contributes the current-state factor $\Theta_\rho(\lambda) + a_3$, where $a_3 = \mu_h + \alpha + \gamma$. Hence, the characteristic equation is fully consistent with systems (2.1) and (2.3).

Remark 3.1. At the disease-free equilibrium, the avian-to-human transmission coefficient β_3 appears in the linearized matrix but does not appear explicitly in the factorized characteristic equation. This happens because infected birds act as a source term for exposed humans, while the local persistence thresholds are determined by the avian-to-avian transmission route and the human-to-human transmission route. Therefore, the disease-free stability threshold is governed by $\mathcal{R}_1$ and $\mathcal{R}_2$.

Theorem 3.2. Assume that $\tau_1 = \tau_2 = 0$. Then the disease-free equilibrium E_0 of system (2.3) is locally asymptotically stable whenever

$$\mathcal{R}_0 = \max\{\mathcal{R}_1, \mathcal{R}_2\} < 1.$$

Moreover, E_0 is unstable whenever $\mathcal{R}_0 > 1$.

Proof. When $\tau_1 = \tau_2 = 0$, Eq (3.4) reduces to

$$(\Theta_\rho + m - d_1)(\Theta_\rho + \mu_h)(\Theta_\rho + a_1 - \beta_1 S_a^0) \times [(\Theta_\rho + a_2 + \sigma)(\Theta_\rho + a_3) - \sigma\beta_2 S_h^0] = 0. \quad (3.5)$$

The first two factors yield $\Theta_\rho = -(m - d_1)$, and $\Theta_\rho = -\mu_h$. Since $m > d_1$ and $\mu_h > 0$, both auxiliary roots are negative. The avian infection factor gives

$$\Theta_\rho = \beta_1 S_a^0 - a_1 = a_1(\mathcal{R}_1 - 1).$$

Thus, if $\mathcal{R}_1 < 1$, this auxiliary root is negative. The human infection factor can be written as

$$\Theta_\rho^2 + (a_2 + \sigma + a_3)\Theta_\rho + (a_2 + \sigma)a_3 - \sigma\beta_2 S_h^0 = 0.$$

Using

$$\mathcal{R}_2 = \frac{\sigma\beta_2 S_h^0}{a_3(a_2 + \sigma)},$$

we obtain

$$\Theta_\rho^2 + (a_2 + \sigma + a_3)\Theta_\rho + (a_2 + \sigma)a_3(1 - \mathcal{R}_2) = 0.$$

If $\mathcal{R}_2 < 1$, all coefficients are positive, and the Routh-Hurwitz criterion implies that both auxiliary roots have negative real parts. Therefore, when $\mathcal{R}_0 = \max\{\mathcal{R}_1, \mathcal{R}_2\} < 1$, all auxiliary roots $z = \Theta_\rho(\lambda)$ have strictly negative real parts. By the spectral mapping lemma established above, $\Re z < 0 \rightarrow \Re \lambda < 0$. Hence, all characteristic roots lie in the open left half-plane, and E_0 is locally asymptotically stable. Conversely, if $\mathcal{R}_0 > 1$, then either $\mathcal{R}_1 > 1$ or $\mathcal{R}_2 > 1$. In either case, the corresponding auxiliary equation possesses a positive real root $z > 0$. Since $\Theta_\rho: (0, \infty) \rightarrow (0, \infty)$ is continuous, strictly increasing, and onto, this positive auxiliary root corresponds to a unique positive real characteristic root $\lambda > 0$. Therefore, E_0 is unstable. On the other hand, if $\mathcal{R}_0 > 1$, then either $\mathcal{R}_1 > 1$ or $\mathcal{R}_2 > 1$. If $\mathcal{R}_1 > 1$, then $\Theta_\rho = a_1(\mathcal{R}_1 - 1) > 0$, which produces an unstable characteristic root. If $\mathcal{R}_2 > 1$, then the constant term of the human infection quadratic becomes negative, and hence the quadratic has one positive auxiliary root. Therefore, E_0 is unstable whenever $\mathcal{R}_0 > 1$. This completes the proof. $\square$

Next, we study the effect of the avian infectious-pressure delay τ_1 . From (3.4), the delay-dependent avian factor is

$$H_a(\lambda, \tau_1) = \Theta_\rho(\lambda) + a_1 - \beta_1 S_a^0 e^{-\lambda\tau_1}. \quad (3.6)$$

Let $\lambda = i\omega$, where $\omega > 0$. Then

$$\Theta_\rho(i\omega) = \Theta_R(\omega; \rho) + i\Theta_I(\omega; \rho).$$

Substituting $\lambda = i\omega$ into (3.6) gives

$$\Theta_R + i\Theta_I + a_1 - \beta_1 S_a^0 [\cos(\omega\tau_1) - i\sin(\omega\tau_1)] = 0.$$

Separating real and imaginary parts yields

$$\Theta_R(\omega; \rho) + a_1 - \beta_1 S_a^0 \cos(\omega\tau_1) = 0 \quad (3.7)$$

and

$$\Theta_I(\omega; \rho) + \beta_1 S_a^0 \sin(\omega\tau_1) = 0. \quad (3.8)$$

Hence, purely imaginary roots associated with τ_1 can occur only if

$$\mathcal{G}_a(\omega; \rho) = (\Theta_R(\omega; \rho) + a_1)^2 + \Theta_I^2(\omega; \rho) - (\beta_1 S_a^0)^2 = 0. \quad (3.9)$$

Theorem 3.3. Assume that $\mathcal{R}_0 < 1$. If Eq (3.9) has a positive root $\omega_a > 0$ and the transversality condition

$$\left. \frac{d \operatorname{Re}(\lambda)}{d\tau_1} \right|_{\lambda=i\omega_a, \tau_1=\tau_1^*} \neq 0$$

holds, then the disease-free equilibrium E_0 undergoes Hopf bifurcation at $\tau_1 = \tau_1^*$.

Proof. Let $\lambda = i\omega_a$, $\omega_a > 0$, be a purely imaginary root of (3.6). Define

$$C_a = \frac{\Theta_R(\omega_a; \rho) + a_1}{\beta_1 S_a^0}$$

and

$$S_a = -\frac{\Theta_I(\omega_a; \rho)}{\beta_1 S_a^0}.$$

Then the critical values of the avian infectious-pressure delay are

$$\tau_{1,n}^{(\rho)} = \frac{1}{\omega_a} [\operatorname{atan2}(S_a, C_a) + 2n\pi], \quad n = 0, 1, 2, \dots \quad (3.10)$$

The first positive critical delay is

$$\tau_1^* = \min_{n \geq 0} \{ \tau_{1,n}^{(\rho)} : \tau_{1,n}^{(\rho)} > 0 \}.$$

Differentiating

$$H_a(\lambda, \tau_1) = \Theta_\rho(\lambda) + a_1 - \beta_1 S_a^0 e^{-\lambda\tau_1} = 0$$

with respect to τ_1 , we obtain

$$\frac{d\lambda}{d\tau_1} = \frac{\beta_1 S_a^0 \lambda e^{-\lambda\tau_1}}{\Theta'_\rho(\lambda) + \beta_1 S_a^0 \tau_1 e^{-\lambda\tau_1}}.$$

If

$$\operatorname{Re} \left(\frac{d\lambda}{d\tau_1} \right) \Big|_{\lambda=i\omega_a, \tau_1=\tau_1^*} \neq 0,$$

then the conjugate pair of characteristic roots crosses the imaginary axis with non-zero speed. Therefore, Hopf bifurcation occurs at $\tau_1 = \tau_1^*$. This completes the proof. $\square$

We now consider the effect of the human transmission-response delay τ_2 . From Eq (3.4), the corresponding human delay-dependent factor is

$$H_h(\lambda, \tau_2) = (\Theta_\rho + a_2 + \sigma)(\Theta_\rho + a_3) - \sigma\beta_2 S_h^0 e^{-\lambda\tau_2}. \quad (3.11)$$

Let $\lambda = i\omega$, $\omega > 0$, and write

$$\Theta_\rho(i\omega) = \Theta_R(\omega; \rho) + i\Theta_I(\omega; \rho).$$

Define

$$P_h(\omega; \rho) = [\Theta_\rho(i\omega) + a_2 + \sigma][\Theta_\rho(i\omega) + a_3] = P_R(\omega; \rho) + iP_I(\omega; \rho),$$

where

$$P_R(\omega; \rho) = (\Theta_R + a_2 + \sigma)(\Theta_R + a_3) - \Theta_I^2$$

and

$$P_I(\omega; \rho) = \Theta_I(2\Theta_R + a_2 + \sigma + a_3).$$

Setting $\ell_0 = \sigma\beta_2 S_h^0$, Eq (3.11) can be written as $P_h(\omega; \rho) = \ell_0 e^{-i\omega\tau_2}$. Hence, purely imaginary characteristic roots associated with τ_2 can occur only if

$$G_h(\omega; \rho) = P_R^2(\omega; \rho) + P_I^2(\omega; \rho) - \ell_0^2 = 0. \quad (3.12)$$

Theorem 3.4. Assume that $R_0 < 1$. If Eq (3.12) admits a positive root $\omega_h > 0$, and the transversality condition

$$\left. \frac{d \operatorname{Re}(\lambda)}{d\tau_2} \right|_{\lambda=i\omega_h, \tau_2=\tau_2^*} \neq 0$$

holds, then the disease-free equilibrium E_0 undergoes a Hopf bifurcation at $\tau_2 = \tau_2^*$.

Proof. Let $\lambda = i\omega_h$, where $\omega_h > 0$, be a purely imaginary root of Eq (3.11). Then

$$P_R(\omega_h; \rho) + iP_I(\omega_h; \rho) = \ell_0 [\cos(\omega_h\tau_2) - i\sin(\omega_h\tau_2)].$$

Therefore,

$$\cos(\omega_h\tau_2) = \frac{P_R(\omega_h; \rho)}{\ell_0}, \quad \sin(\omega_h\tau_2) = -\frac{P_I(\omega_h; \rho)}{\ell_0}.$$

The corresponding critical delay values are

$$\tau_{2,n}^{(\rho)} = \frac{1}{\omega_h} \left[\operatorname{atan2} \left(-\frac{P_I(\omega_h; \rho)}{\ell_0}, \frac{P_R(\omega_h; \rho)}{\ell_0} \right) + 2n\pi \right], \quad n = 0, 1, 2, \dots \quad (3.13)$$

The first positive critical value is

$$\tau_2^* = \min_{n \geq 0} \{ \tau_{2,n}^{(\rho)} : \tau_{2,n}^{(\rho)} > 0 \}.$$

Differentiating $H_h(\lambda, \tau_2) = 0$ implicitly with respect to τ_2 , we obtain

$$\frac{d\lambda}{d\tau_2} = -\frac{\partial H_h / \partial \tau_2}{\partial H_h / \partial \lambda},$$

where

$$\frac{\partial H_h}{\partial \tau_2} = \ell_0 \lambda e^{-\lambda \tau_2}$$

and

$$\frac{\partial H_h}{\partial \lambda} = \Theta'_\rho(\lambda) [2\Theta_\rho + a_2 + \sigma + a_3] + \ell_0 \tau_2 e^{-\lambda \tau_2}.$$

Thus,

$$\frac{d\lambda}{d\tau_2} = -\frac{\ell_0 \lambda e^{-\lambda \tau_2}}{\Theta'_\rho(\lambda) [2\Theta_\rho + a_2 + \sigma + a_3] + \ell_0 \tau_2 e^{-\lambda \tau_2}}.$$

If

$$\operatorname{Re} \left(\frac{d\lambda}{d\tau_2} \right) \Big|_{\lambda=i\omega_h, \tau_2=\tau_2^*} \neq 0,$$

then the conjugate pair of characteristic roots crosses the imaginary axis transversally. Hence, the KKA fractional avian-influenza system undergoes Hopf bifurcation at $\tau_2 = \tau_2^*$. $\square$

Remark 3.5. The delay τ_1 represents the lag in avian infectious pressure and therefore appears in the bird-to-bird transmission term. It should not be interpreted as an explicit incubation-stage transition because the avian subsystem does not contain an exposed compartment. The delay τ_2 represents a human transmission-response lag and enters the human-to-human force of infection through $I_h(t - \tau_2)$. Both delays can affect local stability and may induce oscillatory behavior through their respective transmission pathways.

Remark 3.6. When $\rho = 1$, the KKA spectral function satisfies $\Theta_1(\lambda) = \lambda$. Consequently, the above characteristic equations reduce to those of the corresponding classical integer-order multi-delay avian-influenza model. For $0 < \rho < 1$, the critical delay values depend explicitly on the fractional order ρ , so the delay thresholds obtained for the integer-order model cannot be directly applied to the KKA fractional system.

3.1. Stability analysis of interior equilibria and Hopf bifurcation

We now study the endemic equilibrium of the proposed KKA fractional-order avian-influenza model with multiple delays. The analysis is concerned with its local stability properties and the emergence of Hopf bifurcation under suitable delay conditions. Let $E_F = (S_a^*, I_a^*, S_h^*, E_h^*, I_h^*)$ denote the fully endemic equilibrium satisfying $\mathcal{R}_1 > 1$. Since the KKA derivative of a constant is zero, the endemic equilibrium has the same algebraic form as in the integer-order model. However, the stability and Hopf bifurcation conditions are modified by the KKA fractional operator. For $0 < \rho < 1$, define the KKA spectral function

$$\Theta_\rho(\lambda) = \frac{KA(\rho)}{1 - \rho} \frac{\lambda}{\lambda^\rho + \frac{\rho}{1 - \rho}}, \quad (3.14)$$

and for the limiting case $\rho = 1$, we set $\Theta_1(\lambda) = \lambda$. Thus, in the characteristic equation of the KKA fractional model, each classical occurrence of λ is replaced by $\Theta_\rho(\lambda)$, whereas the discrete delay terms remain $e^{-\lambda \tau_1}$, $e^{-\lambda \tau_2}$, and $e^{-\lambda(\tau_1 + \tau_2)}$. Let

$$h_1 = \frac{1}{k}, \quad \text{and} \quad q_2 = -I_a^* \beta_1 - d_1 - h_1 m S_a^* + m(1 - h_1 S_a^*).$$

Linearizing system (2.3) around E^* and taking perturbations of the form $e^{\lambda t}$, the characteristic matrix becomes

$$\Delta^*(\lambda) = \begin{pmatrix} \Theta_\rho - q_2 & \beta_1 S_a^* e^{-\lambda \tau_1} & 0 & 0 & 0 \\ -\beta_1 I_a^* & \Theta_\rho + a_1 - \beta_1 S_a^* e^{-\lambda \tau_1} & 0 & 0 & 0 \\ 0 & \beta_3 S_h^* & \Theta_\rho + \mu_h + \beta_2 I_h^* + \beta_3 I_a^* & 0 & \beta_2 S_h^* e^{-\lambda \tau_2} \\ 0 & -\beta_3 S_h^* & -(\beta_2 I_h^* + \beta_3 I_a^*) & \Theta_\rho + a_2 + \sigma & -\beta_2 S_h^* e^{-\lambda \tau_2} \\ 0 & 0 & 0 & -\sigma & \Theta_\rho + a_3 \end{pmatrix}, \quad (3.15)$$

where $\Theta_\rho = \Theta_\rho(\lambda)$. Therefore, the characteristic equation at E^* is

$$\det \Delta_*(\lambda) = 0. \quad (3.16)$$

Equivalently, the characteristic equation can be expressed as

$$\begin{aligned} & \Theta_\rho^5 + \Omega_4 \Theta_\rho^4 + \Omega_3 \Theta_\rho^3 + \Omega_2 \Theta_\rho^2 + \Omega_1 \Theta_\rho + \Omega_0 + (\Phi_4 \Theta_\rho^4 + \Phi_3 \Theta_\rho^3 + \Phi_2 \Theta_\rho^2 + \Phi_1 \Theta_\rho + \Phi_0) e^{-\lambda \tau_1} \\ & + (\Pi_4 \Theta_\rho^4 + \Pi_3 \Theta_\rho^3 + \Pi_2 \Theta_\rho^2 + \Pi_1 \Theta_\rho + \Pi_0) e^{-\lambda \tau_2} + (\Sigma_3 \Theta_\rho^3 + \Sigma_2 \Theta_\rho^2 + \Sigma_1 \Theta_\rho + \Sigma_0) e^{-\lambda(\tau_1 + \tau_2)} = 0. \end{aligned} \quad (3.17)$$

For compactness, introduce

$$\begin{aligned} r &= a_1 - q_2, \quad s = -a_1 q_2, \quad c = \mu_h + \beta_2 I_h^* + \beta_3 I_a^*, \quad d = a_2 + \sigma, \\ \ell &= \beta_2 \sigma S_h^*, \quad \kappa = \beta_1 S_a^*, \quad \eta = \beta_1 I_a^* + q_2. \end{aligned} \quad (3.18)$$

Define

$$H_2 = c + d + a_3, \quad H_1 = cd + ca_3 + da_3, \quad H_0 = cda_3. \quad (3.19)$$

With these notations, the characteristic determinant associated with Eq (3.15) can be written in the factorized form

$$\det \Delta^*(\lambda) = [\Theta_\rho^2 + r\Theta_\rho + s + \kappa(\eta - \Theta_\rho)e^{-\lambda \tau_1}] \times [\Theta_\rho^3 + H_2 \Theta_\rho^2 + H_1 \Theta_\rho + H_0 - \ell(\Theta_\rho + \mu_h)e^{-\lambda \tau_2}]. \quad (3.20)$$

Expanding Eq (3.20) and comparing the result with Eq (3.15) gives the following coefficient families.

The zero-delay coefficients are

$$\begin{aligned} \Omega_4 &= r + H_2, \\ \Omega_3 &= s + rH_2 + H_1, \\ \Omega_2 &= sH_2 + rH_1 + H_0, \\ \Omega_1 &= sH_1 + rH_0, \\ \Omega_0 &= sH_0. \end{aligned} \quad (3.21)$$

The coefficients associated with $e^{-\lambda \tau_1}$ are

$$\begin{aligned} \Phi_4 &= -\kappa, \\ \Phi_3 &= \kappa(\eta - H_2), \\ \Phi_2 &= \kappa(\eta H_2 - H_1), \\ \Phi_1 &= \kappa(\eta H_1 - H_0), \\ \Phi_0 &= \kappa \eta H_0. \end{aligned} \quad (3.22)$$

The coefficients associated with $e^{-\lambda\tau_2}$ are

$$\begin{aligned}\Pi_4 &= 0, \\ \Pi_3 &= -\ell, \\ \Pi_2 &= -\ell(r + \mu_h), \\ \Pi_1 &= -\ell(s + r\mu_h), \\ \Pi_0 &= -\ell s\mu_h.\end{aligned}\tag{3.23}$$

Finally, the mixed-delay coefficients associated with $e^{-\lambda(\tau_1+\tau_2)}$ are

$$\begin{aligned}\Sigma_3 &= 0, \\ \Sigma_2 &= \kappa\ell, \\ \Sigma_1 &= \kappa\ell(\mu_h - \eta), \\ \Sigma_0 &= -\kappa\ell\eta\mu_h.\end{aligned}\tag{3.24}$$

Thus, the τ_2 -dependent contribution arises entirely from the delayed human-to-human transmission term $\beta_2 I_h(t - \tau_2)$, whereas recovery remains non-delayed through $a_3 = \mu_h + \alpha + \gamma$.

Thus, all coefficients appearing in Eq (3.17) are explicitly determined by the model parameters and the endemic equilibrium E^* . Moreover, the factorized representation in Eq (3.20) clearly separates the zero-delay contribution, the contribution associated with the first delay τ_1 , the contribution associated with the second delay τ_2 , and the mixed effect generated by the simultaneous presence of both delays. When $\rho = 1$, (3.17) reduces to the corresponding integer-order characteristic equation.

Theorem 3.7. *Let $\tau_1 = \tau_2 = 0$, and let E^* denote the biologically admissible fully endemic equilibrium satisfying $\mathcal{R}_1 > 1$. Under the Routh-Hurwitz conditions derived in (3.20), in particular $2\mu_h > \sigma$, the equilibrium E^* is locally asymptotically stable.*

Proof. For $\tau_1 = \tau_2 = 0$, the characteristic Eq (3.17) reduces to

$$\begin{aligned}& \left[(a_1 + \Theta_\rho)(I_a^*\beta_1 + d_1 + m(2h_1S_a^* - 1) + \Theta_\rho) - \beta_1S_a^*(d_1 + m(2h_1S_a^* - 1) + \Theta_\rho) \right] \\ & \times \left[\beta_2\sigma S_h^*(\Theta_\rho + \mu_h) - (a_2 + \Theta_\rho + \sigma)(a_3 + \Theta_\rho)(I_h^*\beta_2 + I_a^*\beta_3 + \Theta_\rho + \mu_h) \right] = 0,\end{aligned}\tag{3.25}$$

where $a_3 = \mu_h + \alpha + \gamma$.

The first factor in (3.25) gives the quadratic equation

$$\Theta_\rho^2 + \frac{m - d_1}{\mathcal{R}_1}\Theta_\rho + \frac{a_1(\mathcal{R}_1 - 1)(m - d_1)}{\mathcal{R}_1} = 0.\tag{3.26}$$

Since $\mathcal{R}_1 > 1$ and $m > d_1$, all coefficients appearing in (3.26) are positive. It then follows from the Routh-Hurwitz criterion for the auxiliary variable Θ_ρ that the corresponding roots possess negative real parts.

The second factor in (3.25) gives the cubic equation

$$\Theta_\rho^3 + A\Theta_\rho^2 + B\Theta_\rho + C = 0,\tag{3.27}$$

where

$$A = a_2 + a_3 + I_a^* \beta_3 + I_h^* \beta_2 + \mu_h + \sigma, B = (a_2 + a_3 + \sigma)(I_a^* \beta_3 + I_h^* \beta_2 + \mu_h) + a_3 \sigma + a_2 a_3 - \beta_2 \sigma S_h^*$$

and

$$C = a_3(a_2 + \sigma)(I_a^* \beta_3 + I_h^* \beta_2) + \mu_h(a_3 \sigma + a_2 a_3 - \beta_2 \sigma S_h^*).$$

For $\mathcal{R}_1 > 1$, we have

$$a_3 \sigma + a_2 a_3 - \beta_2 \sigma S_h^* > 0.$$

Therefore, $A > 0$, $B > 0$, and $C > 0$. Moreover, under the condition $2\mu_h > \sigma$, one obtains $AB - C > 0$. Thus, by the Routh–Hurwitz criterion, all roots of the cubic Eq (3.27) have negative real parts in the auxiliary variable Θ_ρ .

Consequently, all auxiliary roots $z = \Theta_\rho(\lambda)$ associated with the zero-delay characteristic equation have strictly negative real parts. By the spectral mapping property established above, no characteristic root with $\Re(\lambda) \geq 0$ can map to an auxiliary root satisfying $\Re z < 0$. Therefore, every characteristic root of the zero-delay endemic system satisfies $\Re(\lambda) < 0$, and the fully endemic equilibrium E^* is locally asymptotically stable. This completes the proof. $\square$

For the delay-dependent Hopf bifurcation analysis, define

$$\begin{aligned} P_0^*(\Theta_\rho) &= \Theta_\rho^5 + \Omega_4 \Theta_\rho^4 + \Omega_3 \Theta_\rho^3 + \Omega_2 \Theta_\rho^2 + \Omega_1 \Theta_\rho + \Omega_0, \\ P_1^*(\Theta_\rho) &= \Phi_4 \Theta_\rho^4 + \Phi_3 \Theta_\rho^3 + \Phi_2 \Theta_\rho^2 + \Phi_1 \Theta_\rho + \Phi_0, \\ P_2^*(\Theta_\rho) &= \Pi_4 \Theta_\rho^4 + \Pi_3 \Theta_\rho^3 + \Pi_2 \Theta_\rho^2 + \Pi_1 \Theta_\rho + \Pi_0 \end{aligned}$$

and

$$P_{12}^*(\Theta_\rho) = \Sigma_3 \Theta_\rho^3 + \Sigma_2 \Theta_\rho^2 + \Sigma_1 \Theta_\rho + \Sigma_0.$$

Then (3.17) becomes

$$P_0^*(\Theta_\rho) + P_1^*(\Theta_\rho)e^{-\lambda\tau_1} + P_2^*(\Theta_\rho)e^{-\lambda\tau_2} + P_{12}^*(\Theta_\rho)e^{-\lambda(\tau_1+\tau_2)} = 0. \quad (3.28)$$

Let $F(\lambda, \tau) = 0$ denote the characteristic equation obtained under the delay configuration being considered. A positive solution ω_c of the corresponding frequency equation provides only a candidate imaginary characteristic pair $\lambda = \pm i\omega_c$. To identify a genuine first stability boundary, we impose the following additional requirements:

(H1) $\alpha(0) < 0$, where

$$\alpha(\tau) = \sup \{ \Re \lambda : F(\lambda, \tau) = 0 \}$$

is the spectral abscissa;

(H2) $F(i\omega_c, \tau_c) = 0$, $\omega_c > 0$;

(H3) $\partial_\lambda F(i\omega_c, \tau_c) \neq 0$, so that the critical conjugate pair is simple;

(H4) $\tau_c = \min\{\tau_{j,n} > 0\}$, where the minimum is taken over all positive frequencies and all admissible delay branches;

(H5) $\left. \frac{d \operatorname{Re}(\lambda)}{d\tau} \right|_{\tau=\tau_c} \neq 0$.

The condition (H4), together with numerical characteristic-root continuation on $0 \leq \tau < \tau_c$, ensures that no earlier stability crossing is overlooked. Then define

$$\frac{d\lambda}{d\tau} = -\frac{F_\tau(\lambda, \tau)}{F_\lambda(\lambda, \tau)}.$$

Theorem 3.8. *Let $\tau_2 = 0$. Suppose that $G_1^*(\omega; \rho) = 0$ has a positive root $\omega_{10} > 0$, and that the transversality condition*

$$\left. \frac{d \operatorname{Re}(\lambda)}{d\tau_1} \right|_{\lambda=i\omega_{10}, \tau_1=\tau_{10}^{(\rho)}} \neq 0$$

holds. Then the endemic equilibrium E^* is locally asymptotically stable for $0 \leq \tau_1 < \tau_{10}^{(\rho)}$, $\tau_2 = 0$, provided that $\tau_{10}^{(\rho)}$ is the first positive spectral stability boundary satisfying conditions (H1)–(H5). At $\tau_1 = \tau_{10}^{(\rho)}$, a pair of complex conjugate characteristic roots crosses the imaginary axis, and the KKA fractional system undergoes a Hopf-type spectral stability switch near E^* .

Proof. Setting $\tau_2 = 0$ in Eq (3.28), the characteristic equation associated with the avian infectious-pressure delay can be written as

$$A_1^*(\lambda; \rho) + B_1^*(\lambda; \rho)e^{-\lambda\tau_1} = 0, \quad (3.29)$$

where $A_1^*(\lambda; \rho) = \Theta_\rho^2 + r\Theta_\rho + s$ and $B_1^*(\lambda; \rho) = \kappa(\eta - \Theta_\rho)$. Let $\lambda = i\omega$, $\omega > 0$, and write $\Theta_\rho(i\omega) = \Theta_R(\omega; \rho) + i\Theta_I(\omega; \rho)$. Define

$$A_1^*(i\omega; \rho) = A_{1R}^*(\omega; \rho) + iA_{1I}^*(\omega; \rho)$$

and

$$B_1^*(i\omega; \rho) = B_{1R}^*(\omega; \rho) + iB_{1I}^*(\omega; \rho),$$

where

$$A_{1R}^* = \Theta_R^2 - \Theta_I^2 + r\Theta_R + s, \quad A_{1I}^* = \Theta_I(2\Theta_R + r)$$

and

$$B_{1R}^* = \kappa(\eta - \Theta_R), \quad B_{1I}^* = -\kappa\Theta_I.$$

Substituting $\lambda = i\omega$ into (3.29) gives

$$A_1^*(i\omega; \rho) + B_1^*(i\omega; \rho) [\cos(\omega\tau_1) - i \sin(\omega\tau_1)] = 0.$$

Separating the real and imaginary parts yields

$$A_{1R}^* + B_{1R}^* \cos(\omega\tau_1) + B_{1I}^* \sin(\omega\tau_1) = 0 \quad (3.30)$$

and

$$A_{1I}^* + B_{1I}^* \cos(\omega\tau_1) - B_{1R}^* \sin(\omega\tau_1) = 0. \quad (3.31)$$

Therefore,

$$\cos(\omega\tau_1) = -\frac{A_{1R}^* B_{1R}^* + A_{1I}^* B_{1I}^*}{(B_{1R}^*)^2 + (B_{1I}^*)^2} \quad \text{and} \quad \sin(\omega\tau_1) = \frac{B_{1R}^* A_{1I}^* - B_{1I}^* A_{1R}^*}{(B_{1R}^*)^2 + (B_{1I}^*)^2}.$$

Hence, purely imaginary characteristic roots can occur only if

$$G_1^*(\omega; \rho) = |A_1^*(i\omega; \rho)|^2 - |B_1^*(i\omega; \rho)|^2 = 0. \quad (3.32)$$

Let $\omega_{10} > 0$ be a positive root of Eq (3.32), and define

$$C_1^* = -\frac{A_{1R}^* B_{1R}^* + A_{1I}^* B_{1I}^*}{(B_{1R}^*)^2 + (B_{1I}^*)^2} \Big|_{\omega=\omega_{10}} \quad \text{and} \quad S_1^* = \frac{B_{1R}^* A_{1I}^* - B_{1I}^* A_{1R}^*}{(B_{1R}^*)^2 + (B_{1I}^*)^2} \Big|_{\omega=\omega_{10}}.$$

The corresponding critical avian infectious-pressure delays are

$$\tau_{1,n}^{(\rho)} = \frac{1}{\omega_{10}} [\text{atan2}(S_1^*, C_1^*) + 2n\pi], \quad n = 0, 1, 2, \dots \quad (3.33)$$

The first positive critical delay is therefore

$$\tau_{10}^{(\rho)} = \min_{n \geq 0} \{\tau_{1,n}^{(\rho)} : \tau_{1,n}^{(\rho)} > 0\}.$$

Define

$$H_1^*(\lambda, \tau_1) = A_1^*(\lambda; \rho) + B_1^*(\lambda; \rho)e^{-\lambda\tau_1}.$$

Differentiating $H_1^*(\lambda, \tau_1) = 0$ with respect to τ_1 gives

$$\frac{d\lambda}{d\tau_1} = -\frac{\partial H_1^*/\partial \tau_1}{\partial H_1^*/\partial \lambda},$$

where

$$\frac{\partial H_1^*}{\partial \tau_1} = -\lambda B_1^*(\lambda; \rho)e^{-\lambda\tau_1}$$

and

$$\frac{\partial H_1^*}{\partial \lambda} = (A_1^*)'(\lambda; \rho) + (B_1^*)'(\lambda; \rho)e^{-\lambda\tau_1} - \tau_1 B_1^*(\lambda; \rho)e^{-\lambda\tau_1}.$$

If

$$\text{Re}\left(\frac{d\lambda}{d\tau_1}\right) \Big|_{\lambda=i\omega_{10}, \tau_1=\tau_{10}^{(\rho)}} \neq 0,$$

then the transversality condition is satisfied. Hence, a conjugate pair of characteristic roots crosses the imaginary axis at $\tau_1 = \tau_{10}^{(\rho)}$, producing a Hopf-type spectral stability switch near E^* . $\square$

Theorem 3.9. Let $\tau_1 = 0$. Suppose that $G_2^*(\omega; \rho) = 0$ has a positive root $\omega_{20} > 0$, and that the transversality condition

$$\frac{d \text{Re}(\lambda)}{d\tau_2} \Big|_{\lambda=i\omega_{20}, \tau_2=\tau_{20}^{(\rho)}} \neq 0$$

holds. Then the endemic equilibrium E^* is locally asymptotically stable for $0 \leq \tau_2 < \tau_{20}^{(\rho)}$, $\tau_1 = 0$, provided that $\tau_{20}^{(\rho)}$ is the first positive spectral stability boundary satisfying conditions (H1)–(H5). At $\tau_2 = \tau_{20}^{(\rho)}$, a pair of complex conjugate characteristic roots crosses the imaginary axis, and the KKA fractional system undergoes a Hopf-type spectral stability switch near E^* .

Proof. Setting $\tau_1 = 0$ in Eq (3.28), we obtain

$$A_2^*(\lambda; \rho) + B_2^*(\lambda; \rho)e^{-\lambda\tau_2} = 0, \quad (3.34)$$

where $A_2^*(\lambda; \rho) = P_0^*(\Theta_\rho) + P_1^*(\Theta_\rho)$, and $B_2^*(\lambda; \rho) = P_2^*(\Theta_\rho) + P_{12}^*(\Theta_\rho)$. Here, P_0^* , P_1^* , P_2^* , and P_{12}^* are constructed using the corrected coefficient families in Eqs (3.21)–(3.24).

Let $\lambda = i\omega$, $\omega > 0$,

$$A_2^*(i\omega; \rho) = A_{2R}^*(\omega; \rho) + iA_{2I}^*(\omega; \rho), \quad \text{and} \quad B_2^*(i\omega; \rho) = B_{2R}^*(\omega; \rho) + iB_{2I}^*(\omega; \rho).$$

Substitution into Eq (3.34) gives

$$A_2^*(i\omega; \rho) + B_2^*(i\omega; \rho) [\cos(\omega\tau_2) - i \sin(\omega\tau_2)] = 0.$$

Separating the real and imaginary parts yields

$$A_{2R}^* + B_{2R}^* \cos(\omega\tau_2) + B_{2I}^* \sin(\omega\tau_2) = 0 \quad (3.35)$$

and

$$A_{2I}^* + B_{2I}^* \cos(\omega\tau_2) - B_{2R}^* \sin(\omega\tau_2) = 0. \quad (3.36)$$

Therefore,

$$\cos(\omega\tau_2) = -\frac{A_{2R}^* B_{2R}^* + A_{2I}^* B_{2I}^*}{(B_{2R}^*)^2 + (B_{2I}^*)^2} \quad \text{and} \quad \sin(\omega\tau_2) = \frac{B_{2R}^* A_{2I}^* - B_{2I}^* A_{2R}^*}{(B_{2R}^*)^2 + (B_{2I}^*)^2}.$$

Hence, purely imaginary roots can occur only if

$$G_2^*(\omega; \rho) = |A_2^*(i\omega; \rho)|^2 - |B_2^*(i\omega; \rho)|^2 = 0. \quad (3.37)$$

Let $\omega_{20} > 0$ be a positive root of Eq (3.37), and define

$$C_2^* = -\frac{A_{2R}^* B_{2R}^* + A_{2I}^* B_{2I}^*}{(B_{2R}^*)^2 + (B_{2I}^*)^2} \Big|_{\omega=\omega_{20}},$$

$$S_2^* = \frac{B_{2R}^* A_{2I}^* - B_{2I}^* A_{2R}^*}{(B_{2R}^*)^2 + (B_{2I}^*)^2} \Big|_{\omega=\omega_{20}}.$$

The corresponding critical human transmission-response delays are

$$\tau_{2,n}^{(\rho)} = \frac{1}{\omega_{20}} [\text{atan2}(S_2^*, C_2^*) + 2n\pi], \quad n = 0, 1, 2, \dots \quad (3.38)$$

The first positive critical value is

$$\tau_{20}^{(\rho)} = \min_{n \geq 0} \{ \tau_{2,n}^{(\rho)} : \tau_{2,n}^{(\rho)} > 0 \}.$$

Define $H_2^*(\lambda, \tau_2) = A_2^*(\lambda; \rho) + B_2^*(\lambda; \rho)e^{-\lambda\tau_2}$. Differentiating $H_2^*(\lambda, \tau_2) = 0$ with respect to τ_2 gives

$$\frac{d\lambda}{d\tau_2} = -\frac{\partial H_2^* / \partial \tau_2}{\partial H_2^* / \partial \lambda},$$

where

$$\frac{\partial H_2^*}{\partial \tau_2} = -\lambda B_2^*(\lambda; \rho) e^{-\lambda \tau_2}$$

and

$$\frac{\partial H_2^*}{\partial \lambda} = (A_2^*)'(\lambda; \rho) + (B_2^*)'(\lambda; \rho) e^{-\lambda \tau_2} - \tau_2 B_2^*(\lambda; \rho) e^{-\lambda \tau_2}.$$

If

$$\operatorname{Re} \left(\frac{d\lambda}{d\tau_2} \right) \bigg|_{\lambda=i\omega_{20}, \tau_2=\tau_{20}^{(\rho)}} \neq 0,$$

the transversality condition holds. Hence, a conjugate pair crosses the imaginary axis at $\tau_2 = \tau_{20}^{(\rho)}$, producing a Hopf-type spectral stability switch near E^* . $\square$

Theorem 3.10. Let $\tau_1 = \tau_2 = \tau$. Suppose that $G_3^*(\omega; \rho) = 0$ has a positive root $\omega_0 > 0$, and that

$$\frac{d \operatorname{Re}(\lambda)}{d\tau} \bigg|_{\lambda=i\omega_0, \tau=\tau_0^{(\rho)}} \neq 0.$$

Then, provided $\tau_0^{(\rho)}$ is the first positive stability boundary satisfying (H1)–(H5), the endemic equilibrium E^* is locally asymptotically stable for $0 \leq \tau < \tau_0^{(\rho)}$, and the KKA fractional system undergoes a Hopf-type spectral stability switch near E^* at $\tau = \tau_0^{(\rho)}$.

Proof. Let $\tau_1 = \tau_2 = \tau$. Then Eq (3.28) becomes

$$D_0^*(\lambda; \rho) + D_1^*(\lambda; \rho) e^{-\lambda \tau} + D_2^*(\lambda; \rho) e^{-2\lambda \tau} = 0, \quad (3.39)$$

where $D_0^*(\lambda; \rho) = P_0^*(\Theta_\rho)$, $D_1^*(\lambda; \rho) = P_1^*(\Theta_\rho) + P_2^*(\Theta_\rho)$, and $D_2^*(\lambda; \rho) = P_{12}^*(\Theta_\rho)$, with all P_j^* evaluated using the corrected coefficient families in Eqs (3.21)–(3.24). Let $\lambda = i\omega$, $\omega > 0$, and put $Y = e^{-i\omega \tau}$. Then Eq (3.39) becomes

$$D_2^*(i\omega; \rho) Y^2 + D_1^*(i\omega; \rho) Y + D_0^*(i\omega; \rho) = 0. \quad (3.40)$$

If $D_2^*(i\omega; \rho) \neq 0$, its roots are

$$Y_{\pm}^*(\omega; \rho) = \frac{-D_1^*(i\omega; \rho) \pm \sqrt{[D_1^*(i\omega; \rho)]^2 - 4D_2^*(i\omega; \rho)D_0^*(i\omega; \rho)}}{2D_2^*(i\omega; \rho)}. \quad (3.41)$$

Since $Y = e^{-i\omega \tau}$, it is necessary that $|Y| = 1$. Therefore, purely imaginary characteristic roots can occur whenever, for at least one branch $Y_{\pm}^*$,

$$G_3^*(\omega; \rho) = |Y_{\pm}^*(\omega; \rho)|^2 - 1 = 0. \quad (3.42)$$

Let $\omega_0 > 0$ be a positive root of Eq (3.42), and let $Y_0^* = Y_{\pm}^*(\omega_0; \rho) = Y_R^* + iY_I^*$, $|Y_0^*| = 1$. Then the corresponding critical common-delay values are

$$\tau_n^{(\rho)} = \frac{-\operatorname{atan2}(Y_I^*, Y_R^*) + 2n\pi}{\omega_0}, \quad n = 0, 1, 2, \dots \quad (3.43)$$

The first positive critical value is

$$\tau_0^{(\rho)} = \min_{n \geq 0} \{ \tau_n^{(\rho)} : \tau_n^{(\rho)} > 0 \}.$$

Define

$$H_3^*(\lambda, \tau) = D_0^*(\lambda; \rho) + D_1^*(\lambda; \rho)e^{-\lambda\tau} + D_2^*(\lambda; \rho)e^{-2\lambda\tau}.$$

Then

$$\frac{d\lambda}{d\tau} = -\frac{\partial H_3^*/\partial \tau}{\partial H_3^*/\partial \lambda},$$

where

$$\frac{\partial H_3^*}{\partial \tau} = -\lambda D_1^* e^{-\lambda\tau} - 2\lambda D_2^* e^{-2\lambda\tau}$$

and

$$\frac{\partial H_3^*}{\partial \lambda} = (D_0^*)' + (D_1^*)'e^{-\lambda\tau} - \tau D_1^* e^{-\lambda\tau} + (D_2^*)'e^{-2\lambda\tau} - 2\tau D_2^* e^{-2\lambda\tau}.$$

If

$$\operatorname{Re} \left(\frac{d\lambda}{d\tau} \right) \bigg|_{\lambda=i\omega_0, \tau=\tau_0^{(\rho)}} \neq 0,$$

the transversality condition is satisfied, and a Hopf-type spectral stability switch occurs at $\tau = \tau_0^{(\rho)}$. $\square$

Remark 3.11. The critical values $\tau_{10}^{(\rho)}$, $\tau_{20}^{(\rho)}$, and $\tau_0^{(\rho)}$ depend on the fractional order ρ . Therefore, the critical delay values of the classical integer-order model should not be used directly for the KKA fractional-order model. They must be recomputed from (3.32), (3.37), and (3.42).

4. Sensitivity analysis

In this section, we investigate the sensitivity of the basic reproduction number with respect to the model parameters. Sensitivity analysis is important because it identifies the parameters that have the strongest influence on the spread or control of avian influenza. Although the KKA fractional order ρ does not appear explicitly in the basic reproduction number, it affects the transient memory-dependent behavior, the convergence rate, and the delay-induced stability switching of the proposed fractional model.

For the KKA fractional-order model, the basic reproduction number is given by $\mathcal{R}_0 = \max\{\mathcal{R}_1, \mathcal{R}_2\}$, where

$$\mathcal{R}_1 = \frac{\beta_1 k(m - d_1)}{a_1 m} \quad \text{and} \quad \mathcal{R}_2 = \frac{\beta_2 \Lambda \sigma}{a_3 \mu_h(a_2 + \sigma)}.$$

Here, $a_1 = \mu_a + \delta + d_2$, $a_2 = \mu_h + k_1$, and $a_3 = \mu_h + \alpha + \gamma$. The fractional order ρ does not occur in $\mathcal{R}_0$, because $\mathcal{R}_0$ is obtained from the algebraic infection subsystem at the disease-free equilibrium. Therefore, $F_\rho^{\mathcal{R}_0} = 0$. However, this does not mean that ρ has no biological effect. The parameter ρ controls the memory strength of the KKA fractional system and therefore influences the time evolution, oscillatory behavior, and critical delay values associated with Hopf bifurcation. The parameter values used in the numerical and sensitivity analyses are selected from avian-influenza-specific modeling literature and from the corresponding classical multi-delay avian-influenza formulation. The transmission coefficient $\beta_1 = 0.6$, describing infection among birds,

follows the baseline parameterization adopted in delayed avian-influenza models. The avian-to-human transmission coefficient $\beta_3 = 0.2$ is guided by the avian–human influenza framework of Iwami et al., in which transmission from infectious birds to susceptible humans is explicitly represented. The human disease-induced mortality parameter $\alpha = 0.3445$ is consistent with values used in H7N9 epidemic models, while the recovery parameter $\gamma = 0.61$ follows H7N9 avian-influenza modeling studies. These quantities are therefore treated as literature-supported baseline epidemiological parameters rather than arbitrary numerical choices. The parameters $m, d_1, d_2, \Lambda, \mu_h, \sigma$, and k_1 , for which the exact values are not estimated from a specific outbreak dataset in the present study, are retained as illustrative baseline values from the corresponding classical multi-delay model. Their purpose is to permit qualitative investigation of the effects of memory, delays, sensitivity, and control and to facilitate comparison between the classical and KKA fractional formulations. Accordingly, these values should not be interpreted as universal estimates for all H7N9 or other avian-influenza outbreaks.

Moreover, ρ is not an epidemiological rate but the KKA fractional-order parameter; it is varied over $0 < \rho \leq 1$ to quantify memory effects, with $\rho = 1$ corresponding to the classical integer-order limit. Dataset-specific estimation of the full parameter vector is left for future work.

Definition 4.1. Let $\mathcal{Y} = \mathcal{Y}(v_1, v_2, \dots, v_n)$ be a differentiable function of the parameter v_i . The normalized forward sensitivity index of $\mathcal{Y}$ with respect to v_i is defined by

$$F_{v_i}^{\mathcal{Y}} = \frac{\partial \mathcal{Y}}{\partial v_i} \frac{v_i}{\mathcal{Y}}, \quad i = 1, 2, \dots, n. \quad (4.1)$$

Since $\mathcal{R}_0 = \max\{\mathcal{R}_1, \mathcal{R}_2\}$, the sensitivity index of $\mathcal{R}_0$ is interpreted through the dominant reproduction component. For completeness, we compute the branch-wise sensitivity indices of $\mathcal{R}_1$ and $\mathcal{R}_2$. For the avian transmission component $\mathcal{R}_1$, we obtain

$$F_k^{\mathcal{R}_1} = F_{\beta_1}^{\mathcal{R}_1} = 1 > 0, \quad F_m^{\mathcal{R}_1} = \frac{d_1}{m - d_1} > 0, \quad F_{d_1}^{\mathcal{R}_1} = -\frac{d_1}{m - d_1} < 0 \quad (4.2)$$

and

$$F_{\mu_a}^{\mathcal{R}_1} = -\frac{\mu_a}{\mu_a + \delta + d_2} < 0, \quad F_{\delta}^{\mathcal{R}_1} = -\frac{\delta}{\mu_a + \delta + d_2} < 0, \quad F_{d_2}^{\mathcal{R}_1} = -\frac{d_2}{\mu_a + \delta + d_2} < 0. \quad (4.3)$$

For the human transmission component $\mathcal{R}_2$, we obtain

$$F_{\beta_2}^{\mathcal{R}_2} = F_{\Lambda}^{\mathcal{R}_2} = 1 > 0, \quad F_{\sigma}^{\mathcal{R}_2} = \frac{a_2}{a_2 + \sigma} > 0, \quad F_{k_1}^{\mathcal{R}_2} = -\frac{k_1}{a_2 + \sigma} < 0, \quad F_{\alpha}^{\mathcal{R}_2} = -\frac{\alpha}{a_3} < 0, \quad F_{\gamma}^{\mathcal{R}_2} = -\frac{\gamma}{a_3} < 0 \quad (4.4)$$

and

$$F_{\mu_h}^{\mathcal{R}_2} = \frac{\alpha + \gamma}{\alpha + \gamma + \mu_h} - \frac{\mu_h}{k_1 + \mu_h + \sigma} - 2 < 0. \quad (4.5)$$

Thus, the parameters $k, m, \beta_1, \beta_2, \Lambda$, and σ have positive sensitivity indices. Increasing any of these parameters increases the corresponding reproduction component and therefore enhances the possibility of disease persistence. On the other hand, the parameters $d_1, \mu_a, \delta, d_2, \mu_h, \alpha, \gamma$, and k_1 have negative

sensitivity indices. Increasing these parameters decreases the corresponding reproduction component and therefore helps to reduce disease transmission (see Table 1).

Table 1. Parameter values used for the sensitivity analysis of the KKA fractional model.

Parameter	Value	Reference	Parameter	Value	Reference
m	3	[9]	δ	0.5	[9]
k	5	[9]	d_2	0.02	Assumed
β_1	0.6	[9]	Λ	0.4	Assumed
d_1	0.02	Assumed	β_2	0.003	[35]
μ_a	0.06	[9]	β_3	0.2	[35]
μ_h	0.15	Assumed	σ	0.6	Assumed
k_1	0.3	Assumed	α	0.3445	[34]
γ	0.61	[36]	ρ	$0 < \rho \leq 1$	Assumed

Using the parameter values in Table 1, the corresponding normalized sensitivity indices are listed in Table 2.

Table 2. Normalized sensitivity indices of the reproduction components.

Parameter	k	d_1	m	β_1	μ_a	δ	d_2
Index	1	-0.0067	0.0067	1	-0.1034	-0.8621	-0.0345
Parameter	β_2	Λ	σ	μ_h	α	γ	k_1
Index	1	1	0.4286	-1.2787	-0.3119	-0.5523	-0.2857

Table 2 shows that the most influential positive parameters are k , β_1 , β_2 , and Λ , each with sensitivity index equal to 1. Therefore, a 10% increase in any of these parameters leads to an approximate 10% increase in the corresponding reproduction component. The parameter σ also has a positive effect, with sensitivity index 0.4286, which indicates that increasing the progression rate from exposed to infected humans increases the human infection component. Among the negative indices, μ_h has the largest magnitude, with $F_{\mu_h}^{\mathcal{R}_2} = -1.2787$. This means that a 10% increase in the natural death rate of the human population produces an approximate 12.787% decrease in the human reproduction component. Similarly, the disease-induced death rate in birds δ has a strong negative index, $F_{\delta}^{\mathcal{R}_1} = -0.8621$, which indicates that increasing disease-induced removal of infected birds reduces the avian infection component.

The culling parameters d_1 and d_2 also have negative sensitivity indices. The parameter d_1 reduces the susceptible avian population, while d_2 removes infected birds from the active transmission process. Therefore, increasing avian culling rates decreases the reproduction component and helps control the spread of avian influenza. This confirms that culling of susceptible and infected birds can be an effective intervention strategy when applied appropriately. It is important to note that the fractional order ρ has zero direct sensitivity index with respect to $\mathcal{R}_0$, namely $F_{\rho}^{\mathcal{R}_0} = 0$.

Figure 1 illustrates the combined effects of selected epidemiological parameters on the basic reproduction number R_0 through surface and contour plots. In particular, Figure 1a examines the effects of the susceptible- and infected-bird culling rates, while Figure 1b shows the influence of the carrying capacity and avian transmission rate. Furthermore, Figures 1c and 1d illustrate the effects of

the susceptible-bird culling rate and transmission rate, and the carrying capacity and avian natural mortality rate, respectively. These graphical results complement the sensitivity analysis by illustrating how variations in key parameters influence the disease transmission threshold.

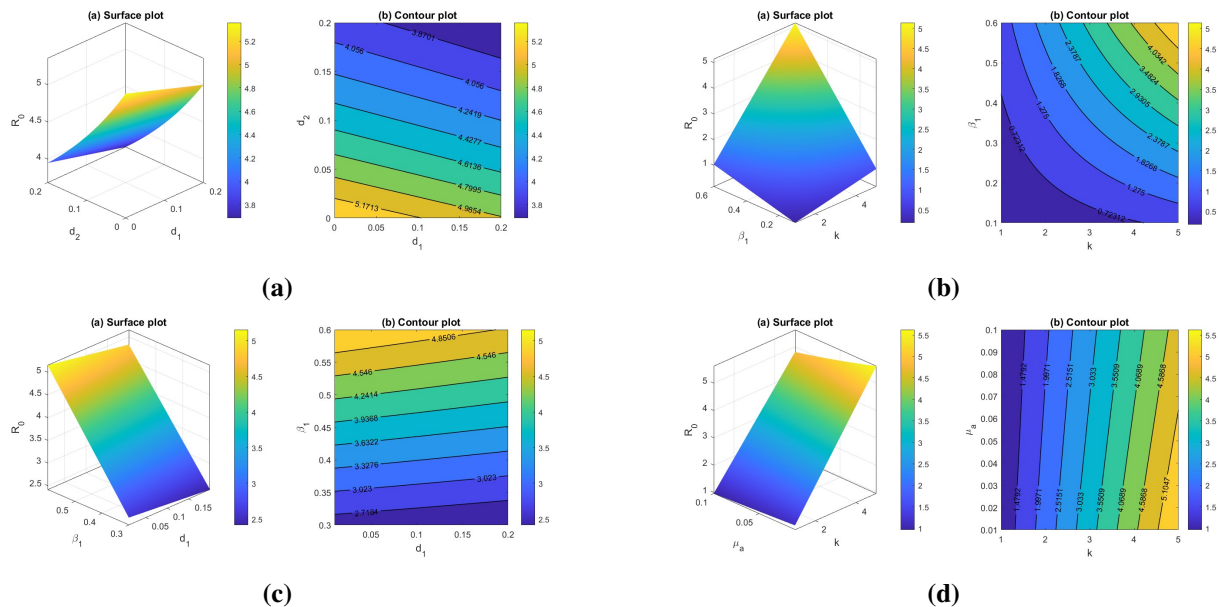


Figure 1. Surface and contour plots of the basic reproduction number R_0 for selected epidemiological parameter pairs.

Nevertheless, ρ should be included in the numerical simulations because it controls the memory effect of the KKA fractional model. Smaller values of ρ generally strengthen memory effects and may slow convergence to equilibrium, change oscillation amplitudes, and shift the critical delay values at which Hopf bifurcation occurs. Hence, while the threshold value R_0 remains algebraically unchanged, the dynamical response of the system is strongly influenced by the KKA fractional order.

5. KKA fractional delay model with optimal control

In this section, we formulate an optimal control problem for the proposed KKA fractional-order multi-delay avian-influenza model. The aim is to reduce the number of infected birds and infected humans while also minimizing the costs associated with the implementation of control measures. The control strategies considered in this study are avian biosecurity measures for reducing effective bird-to-bird transmission, culling of infected birds, and educational campaigns aimed at reducing risky human exposure to infected poultry.

5.1. Formulation

Let $v_1(t)$ – $v_3(t)$ be time-dependent control variables defined on the interval $[0, T]$. Their epidemiological interpretations are as follows:

- $v_1(t)$ represents the intensity of avian biosecurity and contact-reduction measures aimed at reducing effective transmission among birds. Such measures may include improved hygiene and

disinfection, movement restriction, separation of flocks, and reduction of potentially infectious contacts.

- $v_2(t)$ represents the culling or removal intensity of infected birds.
- $v_3(t)$ represents the intensity of educational campaigns and public-awareness measures aimed at reducing risky contact between humans and infected birds.

The controls satisfy $0 \leq v_i(t) \leq 1$, $i = 1, 2, 3$. In particular, $v_1(t) = 0$ represents the absence of additional biosecurity intervention, whereas $v_1(t) = 1$ represents the maximum assumed reduction in effective avian transmission under the control framework. Similarly, $v_2(t) = 0$ and $v_3(t) = 0$ correspond to the absence of the respective interventions.

Using the KKA fractional derivative, the controlled fractional multi-delay model is given by

$$\begin{aligned}
 {}_0^{KKA}D_t^\rho S_a(t) &= mS_a(t) \left(1 - \frac{S_a(t)}{k}\right) - (1 - v_1(t))\beta_1 S_a(t)I_a(t - \tau_1) - d_1 S_a(t), \\
 {}_0^{KKA}D_t^\rho I_a(t) &= (1 - v_1(t))\beta_1 S_a(t)I_a(t - \tau_1) - (\mu_a + \delta + d_2 + v_2(t))I_a(t), \\
 {}_0^{KKA}D_t^\rho S_h(t) &= \Lambda - [\beta_2 I_h(t - \tau_2) + (1 - v_3(t))\beta_3 I_a(t)]S_h(t) - \mu_h S_h(t), \\
 {}_0^{KKA}D_t^\rho E_h(t) &= [\beta_2 I_h(t - \tau_2) + (1 - v_3(t))\beta_3 I_a(t)]S_h(t) - (\mu_h + \sigma + k_1)E_h(t), \\
 {}_0^{KKA}D_t^\rho I_h(t) &= \sigma E_h(t) - (\mu_h + \alpha + \gamma)I_h(t), \\
 {}_0^{KKA}D_t^\rho R(t) &= k_1 E_h(t) + \gamma I_h(t) - \mu_h R(t).
 \end{aligned} \tag{5.1}$$

The initial history functions are given by

$$S_a(\theta) = \Psi_1(\theta), \quad I_a(\theta) = \Psi_2(\theta), \quad S_h(\theta) = \Psi_3(\theta), \quad E_h(\theta) = \Psi_4(\theta), \quad I_h(\theta) = \Psi_5(\theta), \quad R(\theta) = \Psi_6(\theta), \tag{5.2}$$

where $\Psi_i \in C([- \tau, 0], \mathbb{R}_+)$, $\Psi_i(\theta) > 0$, $i = 1, 2, \dots, 6$, and $\tau = \max\{\tau_1, \tau_2\}$. The objective functional is chosen to minimize the number of infected birds, infected humans, and exposed humans, while also reducing the implementation costs of the control strategies. Therefore, we define

$$J(v_1, v_2, v_3) = \int_0^T \left[A_1 I_a(t) + A_2 E_h(t) + A_3 I_h(t) + \frac{1}{2} (B_1 v_1^2(t) + B_2 v_2^2(t) + B_3 v_3^2(t)) \right] dt. \tag{5.3}$$

Here, A_1 , A_2 , and A_3 are non-negative weight constants associated with the epidemiological burden of infected birds, exposed humans, and infected humans, respectively. The constants B_1 , B_2 , and B_3 are positive weight constants associated with the costs of applying the controls $v_1(t)$, $v_2(t)$, and $v_3(t)$, respectively. The admissible control set is defined by

$$\mathcal{U} = \{(v_1, v_2, v_3) : v_i(t) \text{ is Lebesgue measurable on } [0, T], \quad 0 \leq v_i(t) \leq 1, \quad i = 1, 2, 3\}. \tag{5.4}$$

The optimal control problem is to find $(v_1^*, v_2^*, v_3^*) \in \mathcal{U}$ such that

$$J(v_1^*, v_2^*, v_3^*) = \min_{(v_1, v_2, v_3) \in \mathcal{U}} J(v_1, v_2, v_3), \tag{5.5}$$

subject to the controlled KKA fractional system (5.1) and the initial history functions (5.2).

The control $v_1(t)$ reduces the effective avian-to-avian transmission rate from β_1 to $(1 - v_1(t))\beta_1$, representing the effect of intensified biosecurity and reduced infectious contacts among birds. The

control $v_2(t)$ directly removes infected birds from the transmission process through culling, whereas $v_3(t)$ reduces the effective avian-to-human transmission rate β_3 by replacing it with $(1 - v_3(t))\beta_3$. Therefore, the three controls represent complementary interventions acting respectively on transmission prevention in poultry, removal of infectious birds, and reduction of human exposure. When $\rho = 1$, the KKA fractional controlled system (5.1) reduces to the corresponding classical integer-order delay optimal control model.

5.2. Existence and characterization of the optimal control

This subsection establishes the existence of an optimal control for the KKA fractional controlled systems (5.1)–(5.4) and derives the corresponding necessary optimality conditions. Since the state system is governed by a left-sided KKA fractional derivative, the adjoint system is described in terms of a right-sided KKA fractional derivative. For completeness, we distinguish explicitly between the left- and right-sided KKA operators used in the optimality system. Let

$$K_\rho(s) = e_{\rho,\rho} \left(-\frac{\rho}{1-\rho} s \right), \quad c_\rho = \frac{KA(\rho)}{1-\rho}, \quad 0 < \rho < 1.$$

The left-sided Caputo-type KKA derivative governing the state variables is defined by

$${}^{C,KKA}D_{0+}^\rho x(t) = c_\rho \int_0^t x'(\xi) K_\rho(t - \xi) d\xi. \quad (5.6)$$

The corresponding right-sided Riemann-Liouville-type KKA operator, which appears naturally in the adjoint system, is defined by

$${}^{RL,KKA}D_{T-}^\rho y(t) = -c_\rho \frac{d}{dt} \int_t^T y(\xi) K_\rho(\xi - t) d\xi. \quad (5.7)$$

For comparison, the right-sided Caputo-type KKA derivative is

$${}^{C,KKA}D_{T-}^\rho y(t) = -c_\rho \int_t^T y'(\xi) K_\rho(\xi - t) d\xi. \quad (5.8)$$

These two right-sided operators satisfy

$${}^{RL,KKA}D_{T-}^\rho y(t) = {}^{C,KKA}D_{T-}^\rho y(t) + c_\rho y(T) K_\rho(T - t). \quad (5.9)$$

Consequently, under the transversality condition $y(T) = 0$, the two right-sided forms coincide. The left- and right-sided operators are connected through the integration-by-parts identity

$$\int_0^T y(t) {}^{C,KKA}D_{0+}^\rho x(t) dt = \int_0^T x(t) {}^{RL,KKA}D_{T-}^\rho y(t) dt - c_\rho x(0) \int_0^T y(\xi) K_\rho(\xi) d\xi. \quad (5.10)$$

In the variational derivation of the optimality conditions, the state variation has fixed initial history and therefore vanishes at the initial endpoint. Hence, the boundary contribution in Eq (5.10) vanishes. Moreover, the transversality conditions $\lambda_i(T) = 0$ imply that the right-sided Riemann-Liouville-type and Caputo-type KKA derivatives coincide for the adjoint variables.

Theorem 5.1. *There exists an optimal control $v^*(t) = (v_1^*(t), v_2^*(t), v_3^*(t)) \in \mathcal{U}$ with corresponding optimal state variables $S_a^*(t)$, $I_a^*(t)$, $S_h^*(t)$, $E_h^*(t)$, $I_h^*(t)$, and $R^*(t)$, such that*

$$J(v_1^*, v_2^*, v_3^*) = \min_{(v_1, v_2, v_3) \in \mathcal{U}} J(v_1, v_2, v_3),$$

subject to the controlled KKA fractional system (5.1).

Proof. The admissible control set $\mathcal{U}$ is non-empty, closed, convex, and bounded in $L^\infty([0, T])^3$. Moreover, the right-hand side of the controlled system (5.1) is continuous with respect to the state variables and controls, and it is bounded by a linear function of the state variables on the positively invariant region. Hence, for every admissible control $v = (v_1, v_2, v_3) \in \mathcal{U}$, the KKA fractional system admits a bounded non-negative solution. The integrand of the objective functional (5.3) is given by

$$L(t, X, v) = A_1 I_a(t) + A_2 E_h(t) + A_3 I_h(t) + \frac{1}{2} (B_1 v_1^2(t) + B_2 v_2^2(t) + B_3 v_3^2(t)),$$

where $B_i > 0$, $i = 1, 2, 3$. This integrand is convex with respect to the control variables. Furthermore, there exist constants $\omega_1 > 0$, $\omega_2 > 0$, and $\nu > 1$ such that

$$L(t, X, v) \geq \omega_1 (|v_1|^2 + |v_2|^2 + |v_3|^2)^{\nu/2} - \omega_2.$$

Thus, the objective functional is coercive and lower semicontinuous with respect to the controls. Therefore, by the standard existence result for optimal control problems with bounded state variables and convex control costs, there exists an optimal control

$$v^* = (v_1^*, v_2^*, v_3^*) \in \mathcal{U}.$$

This completes the proof. □

The controlled system employs the same delay convention as the uncontrolled model. In particular, τ_2 acts exclusively in the human-to-human transmission term through $I_h(t - \tau_2)$, whereas recovery from the infected-human compartment remains non-delayed. The corresponding Hamiltonian is

$$\begin{aligned} H = & A_1 I_a + A_2 E_h + A_3 I_h + \frac{1}{2} (B_1 v_1^2 + B_2 v_2^2 + B_3 v_3^2) \\ & + \lambda_1 \left[m S_a \left(1 - \frac{S_a}{k} \right) - (1 - v_1) \beta_1 S_a I_a(t - \tau_1) - d_1 S_a \right] \\ & + \lambda_2 [(1 - v_1) \beta_1 S_a I_a(t - \tau_1) - (\mu_a + \delta + d_2 + v_2) I_a] \\ & + \lambda_3 [\Lambda - (\beta_2 I_h(t - \tau_2) + (1 - v_3) \beta_3 I_a) S_h - \mu_h S_h] \\ & + \lambda_4 [(\beta_2 I_h(t - \tau_2) + (1 - v_3) \beta_3 I_a) S_h - (\mu_h + \sigma + k_1) E_h] \\ & + \lambda_5 [\sigma E_h - (\mu_h + \alpha + \gamma) I_h] + \lambda_6 [k_1 E_h + \gamma I_h - \mu_h R]. \end{aligned} \quad (5.11)$$

Let

$$\chi_{[0, T - \tau_j]}(t) = \begin{cases} 1, & 0 \leq t \leq T - \tau_j, \\ 0, & \text{otherwise,} \end{cases} \quad j = 1, 2.$$

The characteristic functions appear because the delayed terms $I_a(t - \tau_1)$ and $I_h(t - \tau_2)$ generate advanced adjoint terms.

Using the above integration-by-parts identity, the necessary optimality conditions follow from the fractional Pontryagin principle for delayed systems. In the notation of the present problem, the adjoint relation takes the general form

$${}^{RL,KKA}D_{T-}^{\rho}\lambda(t) = H_X(t) + \sum_{j=1}^2 \chi_{[0,T-\tau_j]}(t) H_{X_{\tau_j}}(t + \tau_j), \quad \lambda(T) = 0, \quad (5.12)$$

where H_X denotes differentiation of the Hamiltonian with respect to the current state and $H_{X_{\tau_j}}$ denotes differentiation with respect to the delayed state. The second term in Eq (5.12) explains the appearance of advanced adjoint quantities evaluated at $t + \tau_j$. The stationarity conditions with respect to the controls, followed by projection onto the admissible interval, give the optimal-control characterizations stated below.

Theorem 5.2. *Let $v^*(t) = (v_1^*(t), v_2^*(t), v_3^*(t))$ be an optimal control with corresponding optimal state variables S_a^* , I_a^* , S_h^* , E_h^* , I_h^* , and R^* . The adjoint variables satisfy the following right-sided KKA fractional system:*

$$\begin{aligned} {}^{RL,KKA}D_{T-}^{\rho}\lambda_1(t) &= \lambda_1 \left[m - \frac{2mS_a^*}{k} - d_1 - (1 - v_1^*)\beta_1 I_a^*(t - \tau_1) \right] + \lambda_2(1 - v_1^*)\beta_1 I_a^*(t - \tau_1), \\ {}^{RL,KKA}D_{T-}^{\rho}\lambda_2(t) &= A_1 - \lambda_2(\mu_a + \delta + d_2 + v_2^*) - \lambda_3(1 - v_3^*)\beta_3 S_h^* + \lambda_4(1 - v_3^*)\beta_3 S_h^* \\ &\quad + \chi_{[0,T-\tau_1]}(t)\beta_1(1 - v_1^*(t + \tau_1))S_a^*(t + \tau_1) \times [\lambda_2(t + \tau_1) - \lambda_1(t + \tau_1)], \\ {}^{RL,KKA}D_{T-}^{\rho}\lambda_3(t) &= -\lambda_3[\beta_2 I_h^*(t - \tau_2) + (1 - v_3^*)\beta_3 I_a^* + \mu_h] + \lambda_4[\beta_2 I_h^*(t - \tau_2) + (1 - v_3^*)\beta_3 I_a^*], \\ {}^{RL,KKA}D_{T-}^{\rho}\lambda_4(t) &= A_2 - \lambda_4(\mu_h + \sigma + k_1) + \sigma\lambda_5 + k_1\lambda_6, \\ {}^{RL,KKA}D_{T-}^{\rho}\lambda_5(t) &= A_3 - (\mu_h + \alpha + \gamma)\lambda_5(t) + \gamma\lambda_6(t) + \chi_{[0,T-\tau_2]}(t)\beta_2 S_h^*(t + \tau_2) \times [\lambda_4(t + \tau_2) - \lambda_3(t + \tau_2)], \\ {}^{RL,KKA}D_{T-}^{\rho}\lambda_6(t) &= -\mu_h\lambda_6(t). \end{aligned} \quad (5.13)$$

The transversality conditions are

$$\lambda_i(T) = 0, \quad i = 1, 2, \dots, 6. \quad (5.14)$$

Furthermore, the optimal controls are characterized by

$$v_1^*(t) = \min \left\{ 1, \max \left\{ 0, \frac{\beta_1 S_a^*(t) I_a^*(t - \tau_1) [\lambda_2(t) - \lambda_1(t)]}{B_1} \right\} \right\}, \quad (5.15)$$

$$v_2^*(t) = \min \left\{ 1, \max \left\{ 0, \frac{\lambda_2(t) I_a^*(t)}{B_2} \right\} \right\} \quad (5.16)$$

and

$$v_3^*(t) = \min \left\{ 1, \max \left\{ 0, \frac{\beta_3 I_a^*(t) S_h^*(t) (\lambda_4(t) - \lambda_3(t))}{B_3} \right\} \right\}. \quad (5.17)$$

Proof. The Hamiltonian associated with the KKA fractional optimal control problem is given by (5.11). Applying the fractional Pontryagin maximum principle for systems involving delay, the

adjoint equations are obtained by differentiating the Hamiltonian with respect to the state variables. Since the state equations are written with a left-sided KKA derivative, the adjoint system is expressed using the right-sided KKA derivative ${}^{KKA}_0 D_t^\rho$. The delayed terms generate additional advanced contributions in the adjoint system. In particular, $I_a(t - \tau_1)$ appears in the first two state equations and therefore contributes

$$\chi_{[0, T-\tau_1]}(t) \beta_1 [1 - v_1^*(t + \tau_1)] S_a^*(t + \tau_1) [\lambda_2(t + \tau_1) - \lambda_1(t + \tau_1)]$$

to the adjoint equation for λ_2 . Similarly, $I_h(t - \tau_2)$ appears in the susceptible-human and exposed-human equations and contributes

$$\chi_{[0, T-\tau_2]}(t) \beta_2 S_h^*(t + \tau_2) [\lambda_4(t + \tau_2) - \lambda_3(t + \tau_2)]$$

to the adjoint equation for λ_5 . Since recovery from the infected-human compartment is non-delayed, its contribution consists only of the current terms

$$-(\mu_h + \alpha + \gamma) \lambda_5(t) + \gamma \lambda_6(t).$$

To characterize the optimal controls, the stationarity conditions

$$\frac{\partial H}{\partial v_i} = 0, \quad i = 1, 2, 3,$$

give

$$\begin{aligned} \frac{\partial H}{\partial v_1} &= B_1 v_1 + \beta_1 S_a I_a(t - \tau_1) (\lambda_1 - \lambda_2), \\ \frac{\partial H}{\partial v_2} &= B_2 v_2 - \lambda_2 I_a, \\ \frac{\partial H}{\partial v_3} &= B_3 v_3 + \beta_3 I_a S_h (\lambda_3 - \lambda_4). \end{aligned}$$

Hence, the unconstrained controls are

$$\begin{aligned} v_1 &= \frac{\beta_1 S_a(t) I_a(t - \tau_1) [\lambda_2(t) - \lambda_1(t)]}{B_1}, \\ v_2 &= \frac{\lambda_2(t) I_a(t)}{B_2}, \quad v_3 = \frac{\beta_3 I_a(t) S_h(t) [\lambda_4(t) - \lambda_3(t)]}{B_3}. \end{aligned}$$

Projecting these expressions onto $[0, 1]$ yields Eqs (5.15)–(5.17). The proof is complete. $\square$

Substituting the optimal controls into the corrected controlled system gives

$$\begin{aligned} {}^{KKA}_0 D_t^\rho S_a^* &= m S_a^* \left(1 - \frac{S_a^*}{k} \right) - (1 - v_1^*) \beta_1 S_a^* I_a^*(t - \tau_1) - d_1 S_a^*, \\ {}^{KKA}_0 D_t^\rho I_a^* &= (1 - v_1^*) \beta_1 S_a^* I_a^*(t - \tau_1) - (\mu_a + \delta + d_2 + v_2^*) I_a^*, \\ {}^{KKA}_0 D_t^\rho S_h^* &= \Lambda - [\beta_2 I_h^*(t - \tau_2) + (1 - v_3^*) \beta_3 I_a^*] S_h^* - \mu_h S_h^*, \\ {}^{KKA}_0 D_t^\rho E_h^* &= [\beta_2 I_h^*(t - \tau_2) + (1 - v_3^*) \beta_3 I_a^*] S_h^* - (\mu_h + \sigma + k_1) E_h^*, \\ {}^{KKA}_0 D_t^\rho I_h^* &= \sigma E_h^* - (\mu_h + \alpha + \gamma) I_h^*, \\ {}^{KKA}_0 D_t^\rho R^* &= k_1 E_h^* + \gamma I_h^* - \mu_h R^*. \end{aligned} \tag{5.18}$$

The complete optimality system consists of the state system (5.18), the adjoint system (5.13), the transversality condition (5.14), and the control characterizations (5.15)–(5.17).

5.3. Numerical solution of the optimality system

The optimality system is nonlinear and couples the state equations, which are subject to prescribed initial histories, with the adjoint equations, which satisfy terminal conditions. Therefore, the system is solved iteratively by a forward-backward sweep procedure. For a fixed fractional order ρ , the state variables are first advanced from $t = 0$ to $t = T$ using the KKA Adams–Bashforth discretization. Using the resulting state trajectory, the adjoint variables are then computed backward from the terminal conditions $\lambda_i(T) = 0$ (see Algorithm 1).

Algorithm 1 Forward-backward sweep for the KKA optimal-control system.

1: Input:

fractional order ρ , final time T , step size h , delays τ_1, τ_2 , tolerance ε , relaxation parameter ω , and maximum iterations $N_{\max}$.

2: Set $t_n = nh, n = 0, \dots, N$, where $N = T/h$.

3: Initialize admissible controls $v_1^{(0)}(t_n), v_2^{(0)}(t_n), v_3^{(0)}(t_n) \in [0, 1]$.

4: Set iteration counter $k = 0$.

5: repeat
(a) Forward sweep:

Using $v_1^{(k)}, v_2^{(k)}$, and $v_3^{(k)}$, solve the state system from $t = 0$ to T using the KKA Adams–Bashforth scheme, including the prescribed history functions for delayed states.

(b) Terminal conditions:

Set $\lambda_i(T) = 0, i = 1, \dots, 6$.

(c) Backward sweep:

Solve the adjoint system (5.13) backward from T to 0. Evaluate $\lambda_i(t + \tau_j)$ from the stored adjoint grid when $t + \tau_j \leq T$; otherwise set the corresponding term to zero.

(d) Control update:

Compute $v_{1,\text{new}}, v_{2,\text{new}}$, and $v_{3,\text{new}}$ from (5.15)–(5.17).

(e) Projection:

$$v_{i,\text{new}} = \min\{1, \max\{0, v_{i,\text{new}}\}\}, i = 1, 2, 3.$$

(f) Relaxation:

$$v_i^{(k+1)} = \omega v_{i,\text{new}} + (1 - \omega)v_i^{(k)}, i = 1, 2, 3.$$

(g) Convergence error:

$$E_k = \max_i \max_n |v_i^{(k+1)}(t_n) - v_i^{(k)}(t_n)|.$$

(h) Set $k = k + 1$.

6: until $E_k < \varepsilon$ or $k = N_{\max}$
7: Compute the final state, adjoint, and control trajectories and evaluate the adjoint residual.

8: Output: optimal states, adjoints, controls, iteration count, convergence error, and adjoint residual.

The advanced adjoint terms generated by the delays are evaluated from the stored adjoint trajectory whenever $t + \tau_j \leq T$, and are set to zero outside the interval through the characteristic functions already appearing in (5.13). After each forward and backward sweep, the controls are recalculated from Eqs (5.15)–(5.17), projected onto $[0, 1]$, and combined with the previous controls through a relaxation parameter $0 < \omega \leq 1$. The procedure is repeated until the controls criterion $E^{(k)} < \varepsilon$ is satisfied, where

$\varepsilon > 0$ is a prescribed numerical tolerance and converge,

$$E^{(k)} = \max_{i=1,2,3} \max_{0 \leq n \leq N} |v_i^{(k+1)}(t_n) - v_i^{(k)}(t_n)|.$$

This criterion measures the maximum absolute change in the control variables between two consecutive forward–backward sweep iterations. The stopping criterion was based on the maximum change in the controls between two successive iterations, $E^{(k)} < \varepsilon$, whereas the discrete adjoint residual R_λ was evaluated separately as an a posteriori numerical diagnostic.

Let the adjoint equations symbolically be

$${}^{KKA}D_{T-}^\rho \lambda_i(t) = G_i(t, X, \lambda, v), \quad i = 1, \dots, 6.$$

Define the discrete adjoint defect by

$$r_{i,n} = \left({}^{KKA}D_{T-}^\rho \lambda_i \right)_n - G_i(t_n, X_n, \lambda_n, v_n),$$

and report

$$\mathfrak{R}_\lambda = \max_{1 \leq i \leq 6} \max_{0 \leq n \leq N} |r_{i,n}|.$$

The quantity $\mathfrak{R}_\lambda$ measures the maximum discrete defect of the adjoint equations over the complete computational interval.

6. Numerical simulations

In this section, numerical simulations are carried out to support the analytical results obtained for the proposed KKA fractional multi-delay avian-influenza model. The simulations illustrate the effects of the fractional order ρ , the avian infectious-pressure delay τ_1 , the human transmission-response delay τ_2 , and the proposed optimal control strategies on the disease dynamics. Unless otherwise stated, the initial conditions for the reduced system are taken as $S_a(0) = 0.1$, $I_a(0) = 0.1$, $S_h(0) = 0.1$, $E_h(0) = 0.1$, and $I_h(0) = 0.1$. The parameter values used in the numerical experiments are listed in Table 1. The fractional order is selected from $\rho \in \{0.75, 0.85, 0.95, 1\}$. The case $\rho = 1$ corresponds to the classical integer-order model, whereas $0 < \rho < 1$ describes the memory-dependent KKA fractional model.

For the KKA fractional system

$${}_0^{KKA}D_t^\rho X(t) = F(t, X(t), X(t - \tau_1), X(t - \tau_2)),$$

the numerical approximation is obtained using the KKA Adams–Bashforth method. Let $t_n = nh$, where $h > 0$ is the time-step size. Then the discrete approximation is written as

$$\begin{aligned} X_{n+1} = X_n &+ \frac{\rho h}{2KA(\rho)} (3F_n - F_{n-1}) + \frac{(1-\rho)F_n}{hKA(\rho)\Gamma(2-\rho)} \left[2ht_{n+1}^{1-\rho} - ht_n^{1-\rho} - \mathcal{A}t_{n+1}^{2-\rho} + \mathcal{A}t_n^{2-\rho} \right] \\ &- \frac{(1-\rho)F_{n-1}}{hKA(\rho)\Gamma(2-\rho)} \left[ht_{n+1}^{1-\rho} - \mathcal{A}t_{n+1}^{2-\rho} + \mathcal{A}t_n^{2-\rho} \right], \end{aligned} \quad (6.1)$$

where

$$\mathcal{A} = \frac{1-\rho}{2-\rho}.$$

For the delay terms, we use $I_a(t_n - \tau_1) \approx I_{a,n-\ell_1}$ and $I_h(t_n - \tau_2) \approx I_{h,n-\ell_2}$, where $\ell_1 = \frac{\tau_1}{h}$ and $\ell_2 = \frac{\tau_2}{h}$. If ℓ_1 or ℓ_2 is not an integer, linear interpolation is applied.

To assess the numerical robustness of the KKA Adams–Bashforth discretization, a mesh-refinement study is performed using successively smaller time steps. Let $X_h(t)$ denote the numerical solution obtained with time step h . Since an exact solution is not available for the fractional-delay system, the solution on the next finer grid is used as the reference. The relative refinement error is defined by

$$E_{\text{rel}}(h) = \frac{\max_{0 \leq t_n \leq T} \|X_h(t_n) - X_{h/2}(t_n)\|_{\infty}}{\max_{0 \leq t_n \leq T} \|X_{h/2}(t_n)\|_{\infty}}. \quad (6.2)$$

The corresponding empirical refinement rate is calculated from

$$p_h = \log_2 \left(\frac{E_{\text{rel}}(h)}{E_{\text{rel}}(h/2)} \right). \quad (6.3)$$

The same model parameters, initial history functions, delay values, and final integration time are used at every refinement level. Within each time-series comparison, the same numerical step size and a common simulation interval are used for all displayed state variables unless explicitly stated otherwise. Parameter values distinguishing individual panels are given directly in the corresponding captions. For bifurcation diagrams, only post-transient asymptotic extrema are retained, and theoretically determined critical delay values are indicated directly in the plots.

First, we verify the disease-free and endemic stability results in the zero-delay case. For $\beta_1 = 0.11$ and $\tau_1 = \tau_2 = 0$, the basic reproduction number satisfies

$$\mathcal{R}_0 = \max\{\mathcal{R}_1, \mathcal{R}_2\} < 1.$$

Thus, the disease-free equilibrium

$$E_0 = (S_a^0, 0, S_h^0, 0, 0)$$

is locally asymptotically stable. The numerical solution confirms that the infected bird population $I_a(t)$, the exposed human population $E_h(t)$, and the infected human population $I_h(t)$ decay to zero as $t \rightarrow \infty$. On the other hand, for $\beta_1 = 0.6$, we obtain $\mathcal{R}_0 > 1$, and the system admits a positive endemic equilibrium

$$E^* = (S_a^*, I_a^*, S_h^*, E_h^*, I_h^*).$$

For $\tau_1 = \tau_2 = 0$, the solution trajectories approach E^* , which confirms the local stability result for the endemic equilibrium. These two cases are illustrated in Figure 2, where panel (a) corresponds to $\beta_1 = 0.11$, with $\mathcal{R}_0 < 1$, and panel (b) corresponds to $\beta_1 = 0.6$, with $\mathcal{R}_0 > 1$. Both simulations are performed under zero-delay conditions, $\tau_1 = \tau_2 = 0$, using the remaining parameter values listed in Table 1.

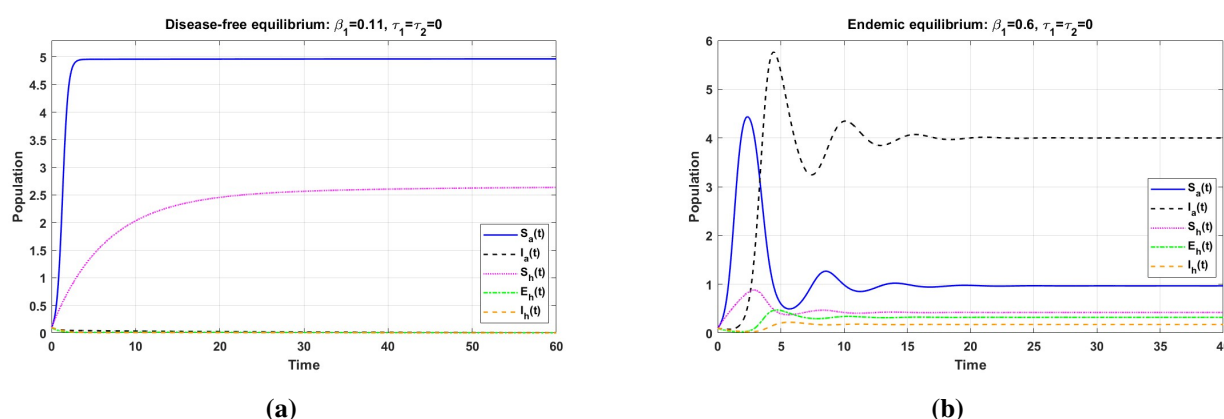


Figure 2. Time-series solutions of the KKA fractional avian-influenza model under zero-delay conditions.

To examine the influence of the fractional order, the system is simulated for different values of ρ . The numerical results show that the fractional order mainly affects the transient response of the solution. Smaller values of ρ produce stronger memory effects, which slow down the convergence toward the equilibrium and may increase the duration of transient oscillations. As $\rho \rightarrow 1$, the solution approaches the behavior of the corresponding classical integer-order model. Figure 3 shows the bifurcation of the state variables with respect to the delay parameter τ_1 .

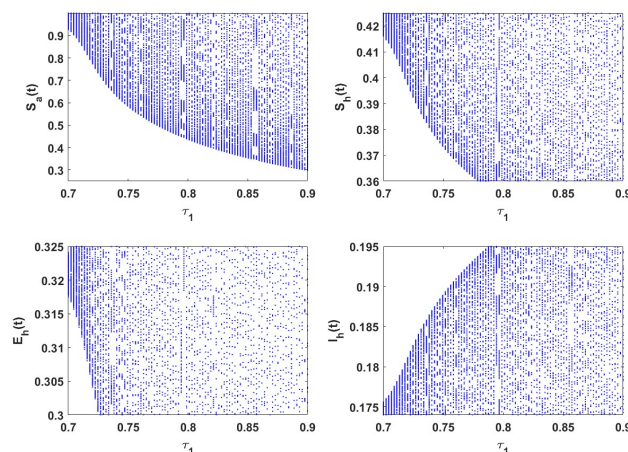


Figure 3. Bifurcation diagrams of the state variables with respect to the avian infectious-pressure delay τ_1 .

Next, we investigate delay-induced Hopf bifurcation caused by the avian infectious-pressure delay τ_1 . We first set $\tau_2 = 0$ and vary τ_1 . The critical value obtained from the KKA characteristic equation is $\tau_{10}^{(\rho)} = 0.7141$. When $\tau_1 = 0.71 < \tau_{10}^{(\rho)}$, the oscillations are damped and all state variables converge to the endemic equilibrium. However, when $\tau_1 = 0.74 > \tau_{10}^{(\rho)}$, persistent oscillations appear in $S_a(t)$, $I_a(t)$, $S_h(t)$, $E_h(t)$, and $I_h(t)$. This indicates that the endemic equilibrium loses stability and a Hopf bifurcation occurs as τ_1 crosses the critical value. These results are shown in Figure 4.

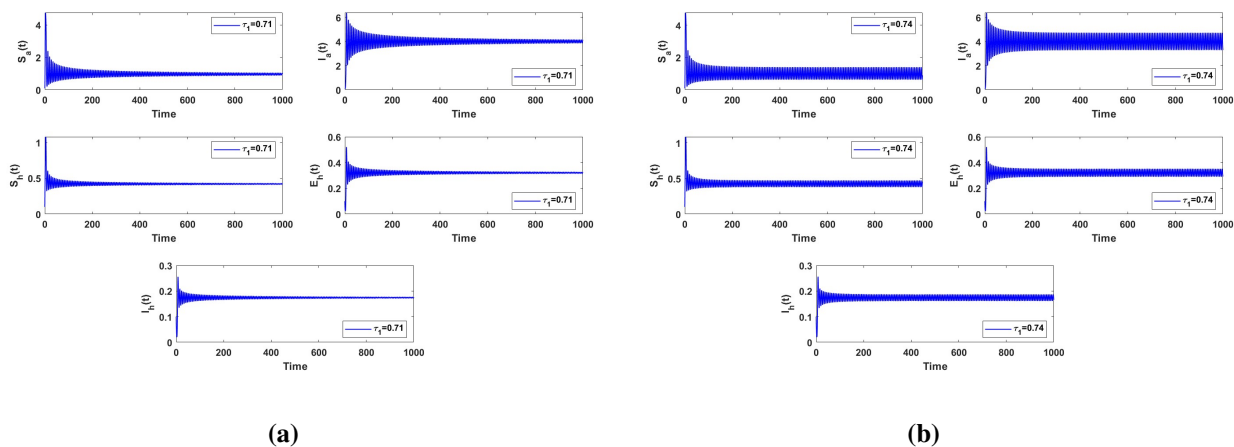


Figure 4. Effects of the avian infectious-pressure delay on the dynamics near the endemic equilibrium.

We then study the effect of the human transmission-response delay τ_2 by setting $\tau_1 = 0$. The critical human transmission-response delay is $\tau_{20}^{(\rho)} = 7.034$. For $\tau_2 = 6.20 < \tau_{20}^{(\rho)}$, the numerical trajectories exhibit damped oscillations and converge to the endemic equilibrium. When $\tau_2 = 7.10 > \tau_{20}^{(\rho)}$, persistent oscillations are observed, showing that the endemic equilibrium becomes unstable through Hopf bifurcation. τ_2 represents a human transmission-response delay, and it acts through the delayed human-to-human infection term $\beta_2 I_h(t - \tau_2) S_h(t)$. Consequently, its influence is observed in the human compartments $S_h(t)$, $E_h(t)$, and $I_h(t)$, whereas the avian subsystem is not directly affected by τ_2 , since no human-to-avian transmission pathway is included in the model. These results are illustrated in Figure 5.

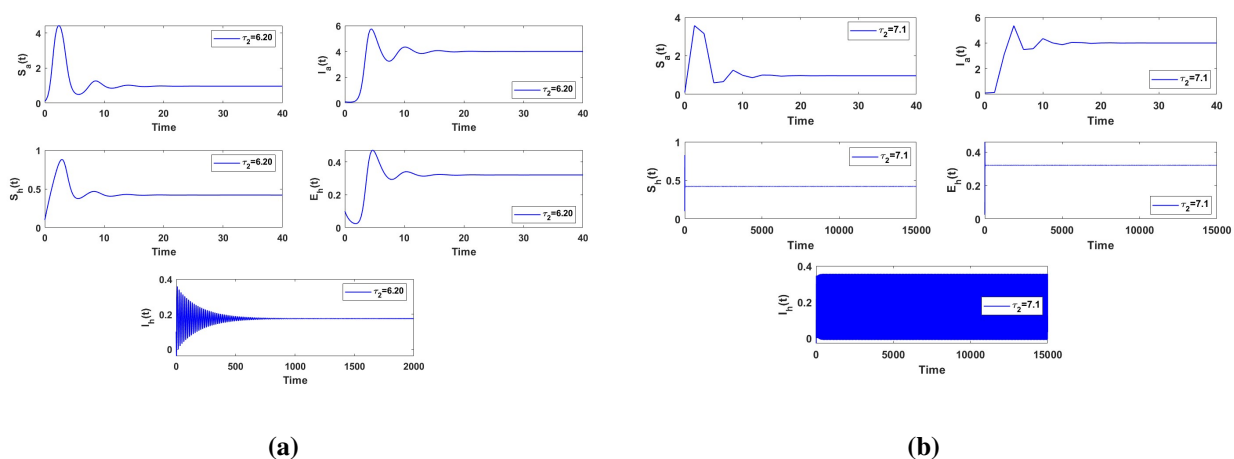


Figure 5. Effects of the human transmission-response delay on the dynamics near the endemic equilibrium.

To distinguish asymptotic oscillatory behavior from transient oscillations, the bifurcation diagrams are constructed from post-transient local extrema rather than from the complete time-series samples. For each value of the delay parameter, the KKA fractional-delay system is integrated on $0 \leq t \leq T_{\text{end}}$

using the numerical step size $h = 0.025$. The initial interval $0 \leq t < T_{\text{tr}}$ is discarded as transient, and only the solution over $T_{\text{tr}} \leq t \leq T_{\text{end}}$ is retained for the bifurcation analysis. For each delay value, a retained sample $X(t_n)$ is classified as a local maximum if $X(t_{n-1}) < X(t_n) > X(t_{n+1})$, and as a local minimum if $X(t_{n-1}) > X(t_n) < X(t_{n+1})$. Only these post-transient extrema are plotted against the corresponding delay value. When the trajectory converges to an equilibrium and no distinct extrema remain, the asymptotic upper and lower values coincide, producing a single branch in the diagram. In contrast, persistent oscillations generate separated upper and lower branches.

The adequacy of the transient length was checked by repeating the extraction with a longer discarded interval and confirming that the resulting extrema were unchanged to the plotting accuracy. Figure 6 presents the bifurcation diagrams obtained from the asymptotic local extrema after removal of the transient interval described above. In panel 6(a), the post-transient maxima and minima of $I_a(t)$ are plotted against the avian transmission delay τ_1 . For delay values below the first spectral stability boundary $\tau_{10}^{(\rho)}$, the upper and lower extrema collapse to the endemic-equilibrium value, indicating convergence to the steady state. Beyond the critical delay, distinct upper and lower extrema appear, indicating persistent oscillatory behavior. The theoretically calculated value $\tau_{10}^{(\rho)}$ is indicated by a vertical dashed line. In panel 6(b), the same procedure is applied to $I_h(t)$ while varying the human transmission-response delay τ_2 . The theoretical first critical value $\tau_{20}^{(\rho)}$ is again shown by a vertical dashed line. The numerical onset of separated asymptotic extrema can therefore be compared directly with the critical value obtained from the characteristic-root analysis.

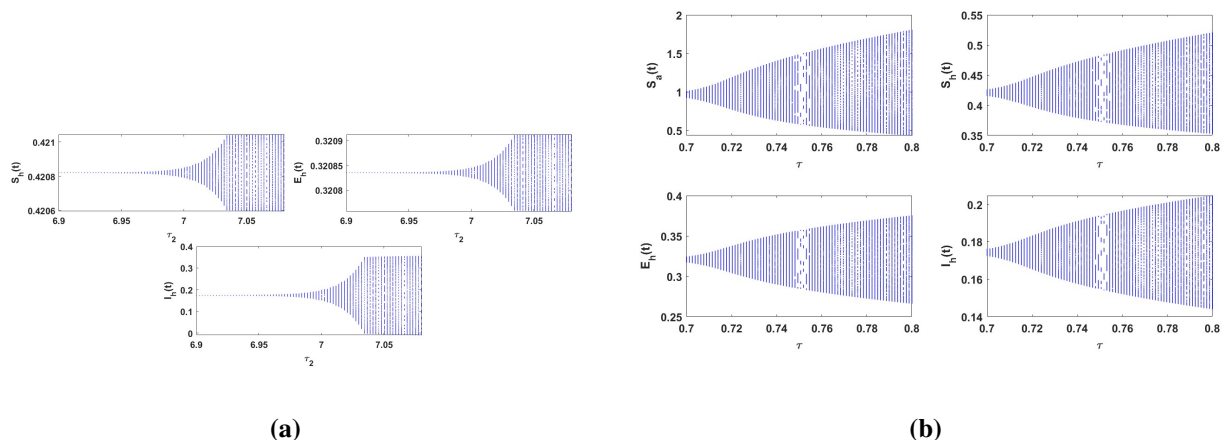


Figure 6. Bifurcation diagrams of infected populations based on post-transient local extrema.

For the equal-delay case $\tau_1 = \tau_2 = \tau$, the numerical simulations are performed for two delay values, $\tau = 5$ and $\tau = 35$. In both cases, the state variables show transient oscillatory behavior and then approach the endemic equilibrium. These simulations indicate that, for the selected parameter set, the endemic equilibrium remains stable under the considered common-delay values. Nevertheless, larger delays can increase the duration and amplitude of transient oscillations. In Figure 7, panel (a) corresponds to the equal-delay case $\tau_1 = \tau_2 = 5$, whereas panel (b) corresponds to $\tau_1 = \tau_2 = 35$. Both cases exhibit transient oscillations followed by convergence toward the endemic equilibrium.

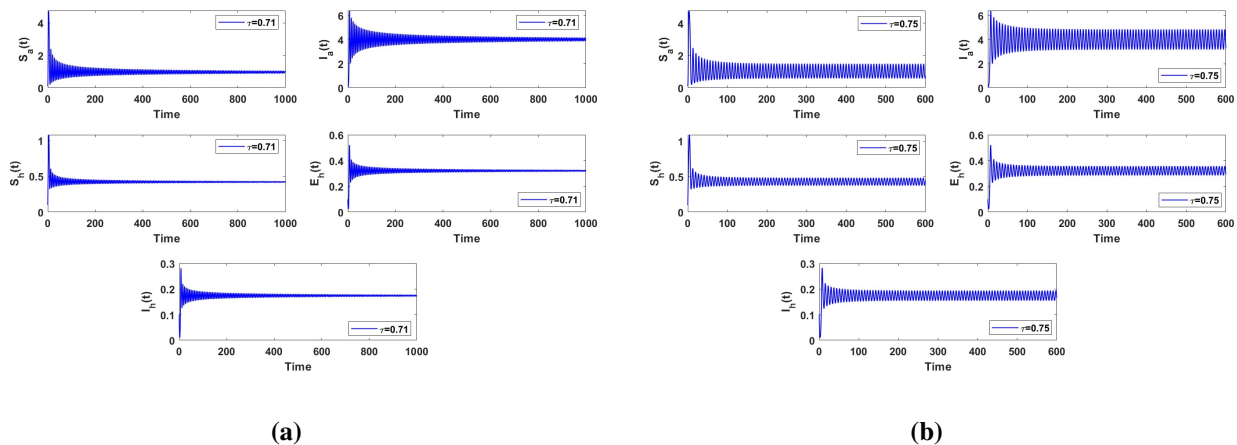


Figure 7. Time-series solutions of the KKA fractional avian-influenza model under equal-delay conditions.

The phase portraits provide a geometric interpretation of the delay-induced dynamics. In the case $\tau_2 = 0$, the trajectory corresponding to $\tau_1 = 0.71 < \tau_{10}^{(\rho)}$ spirals toward the endemic equilibrium, whereas for $\tau_1 = 0.74 > \tau_{10}^{(\rho)}$, a stable limit cycle appears. This confirms the occurrence of Hopf bifurcation caused by the avian infectious-pressure delay. For the common-delay case $\tau_1 = \tau_2 = \tau$, the phase portraits for $\tau = 5$ and $\tau = 35$ show how increasing the delay changes the transient trajectory before convergence. These results are displayed in Figure 8. Figure 8a presents phase portraits in the (S_a, I_a, E_h) space for $\tau_1 = 0.71$ and $\tau_1 = 0.74$, with $\tau_2 = 0$. Figure 8b illustrates the equal-delay cases $\tau_1 = \tau_2 = 5$ and $\tau_1 = \tau_2 = 35$, highlighting the influence of the common delay on the transient dynamics.

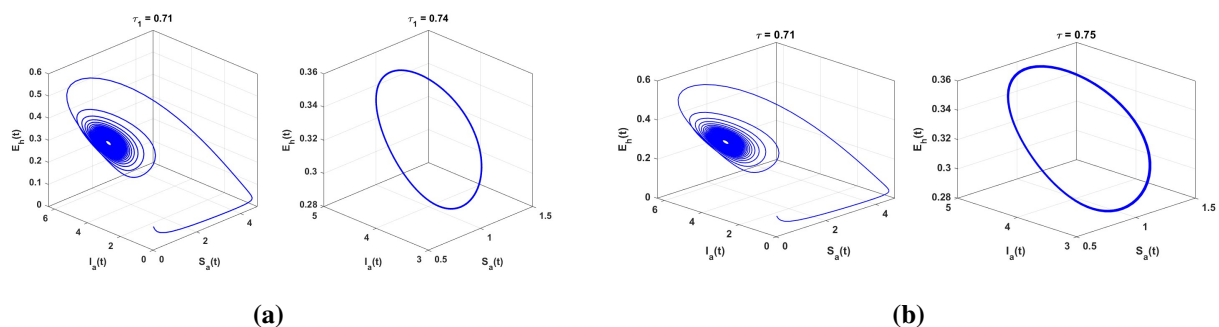


Figure 8. Phase portraits of the KKA fractional avian-influenza model under different delay scenarios.

It is important to emphasize that the critical delay values $\tau_{10}^{(\rho)}$, $\tau_{20}^{(\rho)}$, and $\tau_0^{(\rho)}$ depend on the fractional order ρ . Therefore, the critical delay values of the classical integer-order model cannot be directly used for the KKA fractional model. They must be recalculated from the corresponding KKA characteristic equations for each chosen value of ρ .

6.1. Numerical validation of the KKA Adams–Bashforth scheme

Before presenting the epidemiological simulations, we examine the numerical reliability of the KKA Adams–Bashforth discretization. The convergence and stability properties of the underlying KKA Adams–Bashforth formulation have been established in [32]. Nevertheless, since the present problem additionally contains nonlinear transmission terms and discrete delays, a step-size refinement test is performed to verify the accuracy of its implementation for the proposed avian-influenza system.

Let

$$X_h(t) = (S_{a,h}(t), I_{a,h}(t), S_{h,h}(t), E_{h,h}(t), I_{h,h}(t))^T$$

denote the numerical solution obtained with time step h . Since an exact analytical solution of the nonlinear KKA fractional-delay system is not available, the numerical difference between two successive refinements is measured by

$$E_h = \max_{0 \leq t_n \leq T} |X_h(t_n) - X_{h/2}(t_n)|^\infty.$$

The corresponding empirical convergence rate is calculated from

$$p_h = \log_2 \left(\frac{E_h}{E_{h/2}} \right).$$

For the refinement study, the system is solved using $h = 0.1, 0.05, 0.025$, and 0.0125 , with the same initial conditions and model parameters used in the subsequent simulations. A representative fractional order $\rho = 0.85$ is considered, and the remaining parameters are held fixed throughout the refinement procedure. Table 3 presents the maximum differences between successive numerical solutions. The decreasing values of E_h as h is reduced demonstrate numerical convergence, while the solutions obtained on the two finest grids show negligible differences on the scale of the reported simulations. As an additional independent validation, we consider the limiting case $\rho = 1$ with $\tau_1 = \tau_2 = 0$. Since $KA(1) = 1$, all terms in Eq (6.1) multiplied by $1 - \rho$ vanish, and the KKA Adams–Bashforth discretization reduces to the classical two-step Adams–Bashforth method. The numerical solution obtained from Eq (6.1) is therefore compared with a high-accuracy reference solution of the corresponding integer-order model computed using an adaptive numerical solver with stringent relative and absolute error tolerances. The two solutions exhibit close agreement over the entire simulation interval, and the maximum absolute discrepancy over all compartments is 2.4315×10^{-4} . These mesh-refinement and classical-limit benchmark tests support the reliability of the numerical implementation employed in the subsequent fractional-order, delay-induced bifurcation, and optimal-control simulations.

Table 3. Step-size refinement test for the KKA Adams–Bashforth scheme for $\rho = 0.85$.

The error E_h denotes the maximum difference between solutions obtained on two successive grids.

h	Absolute error E_h	Relative error	Empirical rate p_h
0.1000	$2.35294576 \times 10^{-1}$	$3.97148821 \times 10^{-2}$	–
0.0500	$1.06579581 \times 10^{-1}$	$1.80038022 \times 10^{-2}$	1.142537
0.0250	$4.82264522 \times 10^{-2}$	$8.14887987 \times 10^{-3}$	1.144034
0.0125	Reference/fine grid	Reference/fine grid	–

The step-size refinement results in Table 3 show a systematic decrease in both the absolute and relative errors as the temporal mesh is refined. In particular, the relative difference between the solutions obtained with $h = 0.025$ and $h = 0.0125$ is approximately 8.15×10^{-3} , corresponding to less than 1%. The empirical refinement rates are approximately 1.14, indicating consistent numerical convergence of the KKA Adams–Bashforth discretization for the considered fractional-delay test case $\rho = 0.85$, $\tau_1 = 0.5$, and $\tau_2 = 1$.

6.2. Numerical illustration of optimal control

In this subsection, numerical simulations are presented for the KKA fractional optimal control model. The objective is to compare the uncontrolled dynamics with different controlled scenarios and to evaluate the effectiveness of each intervention strategy. For the optimal-control simulations, we use $m = 1.5$, $k = 1$, $\beta_1 = 0.35$, and $\delta = 0.2$, with $\tau_1 = \tau_2 = 5$. The remaining parameter values are taken from Table 1. The initial values are chosen as $S_a(0) = 5$, $I_a(0) = 5$, for the avian population, and $S_h(0) = 0.3$, $E_h(0) = 0.2$, $I_h(0) = 0.1$, $R(0) = 0.1$, for the human population. The weight constants in the objective functional are selected as $A_1 = 10$, $A_2 = 10$, $A_3 = 60$, and $B_1 = 10$, $B_2 = 10$, $B_3 = 80$. Before comparing the individual control strategies, we assessed the convergence of the forward–backward sweep procedure for the fractional orders used in the simulations. The same initial control guess, time step, relaxation parameter, and stopping tolerance were employed for all values of ρ . Table 4 reports the number of iterations required for convergence, the final control-update error E^k , and the maximum adjoint residual R_λ .

Table 4. Numerical diagnostics of the forward-backward sweep algorithm for different fractional orders. A run is classified as converged only when $E^{(k)} < \varepsilon$ before reaching $N_{\max} = 500$.

ρ	Iterations	Final E	R_λ
0.75	19	4.7684×10^{-7}	0
0.85	500	0.33333	25047
0.95	500	0.26597	300.54
1.00	500	0.26802	357.04

Table 4 shows that the convergence behavior of the forward-backward sweep algorithm depends strongly on the fractional order. For $\rho = 0.75$, the control-update error decreases below the prescribed tolerance after 19 iterations, and the corresponding adjoint residual is negligible. Therefore, this case satisfies the adopted numerical convergence criterion. In contrast, for $\rho = 0.85$, 0.95 , and 1.00 , the algorithm reaches the maximum number of 500 iterations while the final control-update errors remain substantially above the prescribed tolerance. The corresponding adjoint residuals are also non-negligible. Consequently, these three computations are not classified as converged solutions of the forward–backward sweep algorithm under the numerical settings used here. They are therefore reported only as diagnostic evidence of the sensitivity of the iterative procedure to the fractional order and should not be interpreted as fully converged optimal-control solutions.

The results in Table 4 provide the numerical convergence diagnostics for the forward-backward sweep procedure. The number of iterations varies with the fractional order, reflecting the influence of the KKA memory term on the coupling between the forward state system and the backward adjoint

system. In the absence of control, $v_1(t) = v_2(t) = v_3(t) = 0$, the disease persists in both the avian and human populations. Therefore, control strategies are introduced to reduce infection levels and limit transmission. We consider four control scenarios, as shown in Figure 9. Panels (a), (b), and (c) correspond to avian biosecurity control v_1 , infected-bird culling v_2 , and educational awareness control v_3 , respectively, while panel (d) presents the combined application of all three controls. The corresponding population trajectories and control profiles are displayed using the remaining parameter values listed in Table 1.

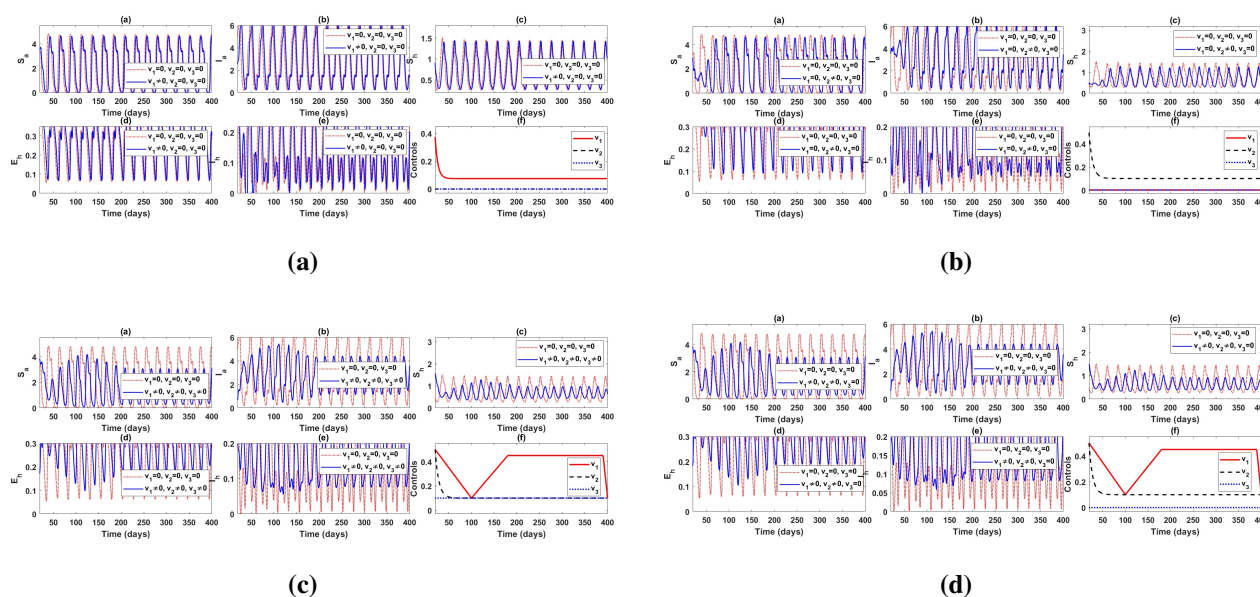


Figure 9. Effects of individual and combined optimal control strategies on avian-influenza transmission.

Strategy 1: avian biosecurity only

In this strategy, only the avian biosecurity control is applied: $v_1(t) \neq 0$, $v_2(t) = 0$, and $v_3(t) = 0$. The control v_1 reduces effective transmission among birds by replacing β_1 with $(1 - v_1(t))\beta_1$. Consequently, fewer susceptible birds acquire infection, leading to a reduction in the infected avian population and, indirectly, in avian-to-human exposure.

Strategy 2: infected-bird culling only

In this strategy, only infected-bird culling is used: $v_1(t) = 0$, $v_2(t) \neq 0$, and $v_3(t) = 0$. The simulations show that $v_2(t)$ directly decreases the infected bird class $I_a(t)$. Since infected birds contribute to avian-to-human transmission, reducing $I_a(t)$ also decreases the exposed human class $E_h(t)$ and the infected human class $I_h(t)$. Therefore, infected-bird culling is more effective than susceptible-bird culling alone in reducing the disease burden.

Strategy 3: educational awareness only

In this strategy, only the educational campaign control is applied: $v_1(t) = 0$, $v_2(t) = 0$, and $v_3(t) \neq 0$. The purpose of the $v_3(t)$ is to reduce the effective avian-to-human transmission rate by replacing β_3

with $(1 - v_3(t))\beta_3$. The numerical results indicate that this strategy reduces human exposure by limiting risky contact between humans and infected birds. Hence, $E_h(t)$ and $I_h(t)$ decrease compared with the uncontrolled case. However, because $v_3(t)$ does not directly remove infected birds, the infected avian population may persist.

Strategy 4: combined control strategy

In this strategy, all three controls are applied simultaneously: $v_1(t) \neq 0$, $v_2(t) \neq 0$, and $v_3(t) \neq 0$. The combined strategy simultaneously applies avian biosecurity, infected-bird culling, and educational awareness. The biosecurity control $v_1(t)$ reduces bird-to-bird transmission, $v_2(t)$ directly removes infectious birds, and $v_3(t)$ reduces avian-to-human exposure.

The numerical simulations indicate that infected-bird culling $v_2(t)$ plays the dominant role in reducing infection because it directly targets the main infectious avian compartment. Susceptible-bird culling $v_1(t)$ helps reduce the pool of birds available for infection, while the education control $v_3(t)$ is particularly useful for reducing avian-to-human transmission. The best outcome is achieved when the three controls are applied together.

The KKA fractional order ρ also influences the controlled dynamics. Smaller values of ρ introduce stronger memory effects, which may delay the impact of the control measures and prolong the time required to reduce the infection. As $\rho \rightarrow 1$, the controlled system approaches the behavior of the corresponding classical integer-order controlled model.

7. Conclusions

In this paper, we formulated and analyzed a KKA fractional-order multi-delay model for avian-influenza transmission. The model extends the classical delayed framework by incorporating the KKA fractional operator to capture memory effects. The two delays represent distinct transmission-memory mechanisms: τ_1 describes a lag in avian infectious pressure, whereas τ_2 describes a lag in the effect of previous human infectious prevalence on current human-to-human transmission. This interpretation avoids attributing an explicit incubation transition to the SI avian subsystem and eliminates the positivity difficulty associated with delayed removal from the infected-human compartment. The non-negativity and boundedness of solutions were established, and the disease-free and endemic equilibria were derived. The basic reproduction number was obtained as $\mathcal{R}_0 = \max\{\mathcal{R}_1, \mathcal{R}_2\}$. Since the equilibrium equations are algebraic, ρ does not appear explicitly in $\mathcal{R}_0$. However, it affects the transient behavior, convergence rate, oscillations, and critical delay values. The local stability of the equilibria was investigated using the KKA characteristic equations. For the zero-delay case, stability conditions were obtained through the Routh-Hurwitz criterion. In the delay case, the critical delay values depend on the model parameters and the fractional order ρ . Hopf bifurcation may occur when the avian infectious-pressure delay, human transmission-response delay, or common delay exceeds its critical value, producing periodic outbreaks. Sensitivity analysis showed that transmission rates, carrying capacity, recruitment rate, and progression rate increase $\mathcal{R}_0$, whereas culling rates, death rates, recovery-related parameters, and disease-induced death rates reduce it. Although ρ has zero direct sensitivity with respect to $\mathcal{R}_0$, it strongly influences the memory-dependent disease dynamics. An optimal control problem was formulated using three epidemiologically motivated interventions: avian biosecurity for reducing bird-to-bird transmission, culling of infected

birds, and educational campaigns for reducing avian-to-human exposure. The numerical simulations demonstrate that these interventions act through complementary mechanisms, with infected-bird culling directly reducing the infectious reservoir and biosecurity limiting the generation of new avian infections. The combined intervention strategy produces the greatest reduction in disease prevalence among the considered scenarios. Educational campaigns reduce avian-to-human transmission, depending on cost and transmission intensity. The numerical results support the theoretical analysis and confirm the effect of ρ on disease dynamics. Smaller values of ρ produce stronger memory effects, slower convergence, and modified oscillations, while $\rho \rightarrow 1$ recovers the classical integer-order delayed model. Thus, the KKA fractional framework provides a flexible approach for studying avian-influenza dynamics with memory, multiple delays, Hopf bifurcation, and optimal control. Future work may include vaccination, environmental contamination, stochastic effects, seasonal transmission, spatial diffusion, and parameter estimation using real data.

Use of Generative-AI tools declaration

The authors declares he has not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The author declares no conflict of interest regarding the publication of this manuscript.

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