Stability Analysis of a Fractional-Order Within-Host Model of Swine Influenza (H1N1) with Environmental Transmission

Abstract

Swine influenza (H1N1) continues to be a major public health problem of interest because of the high potential for transmission and the ability to have rapid mutagenesis and to cause persistence in infected hosts. Here, we introduce and discuss a novel within-host fractional-order mathematical model including environmental viral transmission and nonlinear dynamics of the infection through saturated incidence functions. The model includes healthy epithelial cells, infected cells, free virus particles, and environmental viral load, and the Caputo fractional derivative, accounting for memories of delayed biological responses and viral replication. First, we demonstrate the well-posedness of the model by establishing existence, uniqueness, positivity, and boundedness of solutions. We come up with an invariant region to guarantee that all state variables are biologically significant for time. The basic reproduction number R_0 is obtained based on the next-generation matrix methodology and is shown to have two additively related contributions related to the route of either the direct viral transmission and/or the environmental feedback pathways. Analyses of equilibria stability are performed with linearization, Routh–Hurwitz criteria, and the fractional order stability theory. We found that viral dynamics outside the host significantly increase the effective reproduction number and can prolong infection by reinfection mechanisms. Collectively, these results illustrate that environmental transmission effects, nonlinear infection, and memory exert significant contributions to an infection process on infection outcomes. The model provides an integrated perspective of swine flu physiology, and could serve as a framework for effective control programmes.

Keywords: Fractional-Order, Dynamical Systems, Within-Host, Swine Influenza, H1N1, Environmental Transmission, Autophagy, Saturated Incidence.s

1 Introduction

Swine influenza (H1N1), a subtype of influenza A virus, remains a persistent global health problem owing to its high transmissibility and ability to mutate rapidly, allowing it to spread throughout the body. It also has possibilities for generating serious epidemics. The reason for that is because its difficult to distinguish which viruses infect which hosts in our respiratory tract (sphere) and so results in complex, yet dynamic processes within the host cells: Viruses replicate there. Their replication occurs without the body observing it, the number of cells is depleted and the immune system releases these via action (through clearance cells). The underlying systems of flu are crucial in predicting its progression and facilitating effective treatment, due to its many processes. Mathematical modeling has made an invaluable contribution to understanding the dynamics of infectious disease dynamics as one of them. Previous works like [1, 2, 6, 3, 4] have proposed a process for understanding disease spread by population. Such models have then been extended to within host systems and interactions among target cells, infected cells and free virus particles are explicitly modeled [5, 7].

Within host models have well understood major characteristics of viral infections such as the nature of viral load kinetics, peak infection rates, and clearance mechanisms to a large extent. In contrast,

classically integer-order models have been successfully built on the idea that the biologic are memoryless. But many biological systems have a memory side and a hereditary effect in a way particularly in cases of viral infections where the immune system needs to respond to things like viruses and for replication, past states can determine the future. These effects can be effectively taken into consideration thanks to fractional-order differential equations. These models allow us to model the system dynamics in a historical model, which should be more faithful to the biological processes. In this regard, recent studies have established that fractional-order models are superior for epidemiological modeling processes. For instance, the two previous papers [11], [10] showed that fractional models capture the delayed peak infection and the prolonged persistence of disease significantly better than classical ones. For influenza, fractional-order formulations have been employed to adequately simulate infection dynamics and augment predictability [12, 14, 13]. The incidence function is an area which is particularly important in infectious disease modeling.

The well-known bilinear incidence function indicates that infection rates increase with viral load to the same degree and that such a relationship may not hold for more-than-normal viral numbers. Saturated incidence function was introduced in order to account biological restrictions by saturation, localized spatial limits, and immune suppression [**?**, 16]. Such non-linearity of incidence functions can remarkably modify system mechanics (or how well a system is able to behave) leading to the systems exhibiting stable behavior with greater equilibria and trends in complex stability. Additionally, environmental transmission plays a crucial role in respiratory illnesses such as influenza. For a broad spectrum of systemic infections related to respiratory infections, such as influenza, these viruses can persist in an integrative state, or in mucus and extracellular tissues, feeding into feedback mechanisms in order to continue the infection in a variety of environments even once direct virulence diminishes. Including environmental viral compartments within the host models offers a more accurate representation of infection patterns and has been a subject of recent modeling [19, 20]. Very thick qualitative analysis to verify that mathematical models correspondingly biologically meaningful and mathematically well-posed is a must.

Positivity, boundedness and invariance all contribute to the fact that answers must not exceed viable biological constraints. The following model represents the next-best methodology for extracting the reproduction number R_0, where the biological number R_0, the infection threshold value for retention after infection persistence, can be determined using systematic approach through the next generation matrix method [17, 18]. The stability analysis by means of Routh–Hurwitz criterion and Lyapunov has been well-established to identify equilibrial function in epidemic models [16]. Nevertheless, this has not yet provided a strong enough model of the complex of fractional order dynamics, nonlinear incidence or environmental transmission in a comprehensive framework for within host influenza systems simulation. While a considerable amount of investigations combine these considerations individually and focus on them, few also combine these to characterize the dynamic range of infection. Filling these gaps, this study constructs a fractional-order within-host model of swine influenza with saturated incidence and environmental viral transmission.

This model accounts for memory effects and non-linear processes associated with infection and encompasses key biological processes. Comprehensive qualitative and stability analyses are conducted for positivity, boundedness, and global behaviour of solutions. A simple reproduction number is derived, and equilibria stability conditions are established. We present to the growing body of literature the fractional epidemic model analysis model as a mathematically challenging yet biologically realistic solution to comprehending swine flu dynamics. Results are pertinent both from evidence-based perspectives in memory effects and non-linear transmission and environmental feedback, and may contribute to understanding how these impact infection results and future research and intervention plans.

2 Model Formulation

Let:

- $T(t)$: Healthy epithelial cells
- $I(t)$: Infected cells
- $V(t)$: Virus particles
- $W(t)$: Environmental viral load

$$
{}^{C}D_t^{\alpha} T = rT\left(1 - \frac{T}{K}\right) - \frac{\beta_1 TV}{1+\omega_1 V} - \frac{\beta_2 TW}{1+\omega_2 W}, \tag{1}
$$

$$
{}^{C}D_t^{\alpha} I = \frac{\beta_1 TV}{1+\omega_1 V} + \frac{\beta_2 TW}{1+\omega_2 W} - d_I I, \tag{2}
$$

$$
{}^{C}D_t^{\alpha} V = pI - cV + \frac{\xi W}{1+\omega_3 W}, \tag{3}
$$

$$
{}^{C}D_t^{\alpha} W = \phi V - \mu W. \tag{4}
$$

Table 1: Description of model parameters for the fractional-order swine influenza model.

Parameter	**Description**	**Units**
r	Intrinsic growth rate of healthy epithelial cells	day^{-1}
K	Carrying capacity of healthy epithelial cells in the respiratory tract	cells
β_1	Infection rate of healthy cells by free virus particles within host tissue	$(\text{virion}\cdot\text{day})^{-1}$
β_2	Infection rate of healthy cells due to environmental viral exposure	$(\text{environmental virion}\cdot\text{day})^{-1}$
ω_1	Saturation parameter associated with tissue-virus infection; models reduced infection efficiency at high viral loads	virion^{-1}
ω_2	Saturation parameter associated with environmental viral infection	$(\text{environmental virion})^{-1}$
d_I	Natural death or clearance rate of infected epithelial cells	day^{-1}
p	Production rate of free virus particles by infected cells	$\text{virions}\cdot\text{cell}^{-1}\cdot\text{day}^{-1}$
c	Clearance rate of free virus particles within host tissue	day^{-1}
ξ	Transfer or feedback rate of environmental virus into the tissue viral compartment	day^{-1}
ω_3	Saturation parameter regulating environmental feedback into tissue virus dynamics	$(\text{environmental virion})^{-1}$
ϕ	Shedding rate of tissue virus into the environmental viral compartment	day^{-1}
μ	Natural decay or removal rate of environmental viral particles	day^{-1}
α	Order of the Caputo fractional derivative, representing memory effects in the biological system	dimensionless

3 Qualitative Analysis of the Proposed Model

We analyze fundamental properties of the system, including positivity, invariance, boundedness, and existence of solutions.

3.1 Existence and Uniqueness of Solutions

Theorem 1. *For any non-negative initial conditions*

$$
T(0) = T_0 \geq 0, \quad I(0) = I_0 \geq 0, \quad V(0) = V_0 \geq 0, \quad W(0) = W_0 \geq 0,
$$

the system admits a unique solution $(T(t), I(t), V(t), W(t))$ *on* $[0, \infty)$.

Proof. Reformulation as a fractional integral equation.

Consider the system in vector form:

$$^{C}D_t^{\alpha} X(t) = F(X(t)), \quad X(0) = X_0,$$

where

$$X = (T, I, V, W)^T.$$

Using the definition of the Caputo derivative, the system is equivalent to the Volterra integral equation:

$$X(t) = X_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} F(X(s))\, ds.$$

For the continuity of $F(X)$, each component of $F(X)$ is of the form:

$$\frac{\beta_i X_j X_k}{1+\omega X_k}, \quad rT\left(1-\frac{T}{K}\right), \quad pI, \quad \phi V,$$

which are continuous functions on $\mathbb{R}^4_+$.

Hence, F is continuous.

We show that F is locally Lipschitz in $\mathbb{R}^4_+$.

Consider the nonlinear term:

$$f(V) = \frac{V}{1+\omega V}.$$

Then:

$$f'(V) = \frac{1}{(1+\omega V)^2}.$$

Thus $f'(V)$ is bounded for $V \geq 0$, implying that $f(V)$ is Lipschitz on bounded sets.

Similarly, products such as TV and TW are locally Lipschitz.

Hence, each component of $F(X)$ is locally Lipschitz, and therefore F is locally Lipschitz in $\mathbb{R}^4_+$.

To find the local existence and uniqueness of the systems solution, we note that for standard results for Caputo fractional differential equations (see [9, 8]), if F is continuous and locally Lipschitz, then there exists a unique local solution on $[0, t_{\max})$.

To show global existence, we prove that the solution cannot blow up in finite time.

From the boundedness result (proved earlier), there exists $C > 0$ such that:

$$T(t), I(t), V(t), W(t) \leq C \quad \forall t < t_{\max}.$$

Thus, the solution remains in a compact subset of $\mathbb{R}^4_+$.

By the continuation theorem for fractional differential equations, a solution that remains bounded can be extended beyond $t_{\max}$.

Hence, $t_{\max} = \infty$.

Uniqueness follows from the local Lipschitz condition of F.

Therefore, the system admits a unique global solution on $[0, \infty)$.

□

3.2 Positivity of Solutions

Theorem 2. *For non-negative initial conditions,*

$$T(t),\ I(t),\ V(t),\ W(t) \geq 0 \quad \forall t > 0.$$

Proof. We use a first-exit time argument.

Assume there exists $t_0 > 0$ such that a variable becomes negative for the first time.

Case 1: $T(t_0) = 0$

$$^{C}D_t^{\alpha} T = rT\left(1-\frac{T}{K}\right) - \frac{\beta_1 TV}{1+\omega_1 V} - \frac{\beta_2 TW}{1+\omega_2 W}$$

At $T = 0$:

$$^{C}D_t^{\alpha} T(t_0) = 0.$$

Thus T cannot decrease below zero.

Case 2: $I(t_0) = 0$

$$^{C}D_t^{\alpha} I = \frac{\beta_1 TV}{1+\omega_1 V} + \frac{\beta_2 TW}{1+\omega_2 W} \geq 0.$$

Case 3: $V(t_0) = 0$

$$^{C}D_t^{\alpha} V = pI + \frac{\xi W}{1+\omega_3 W} \geq 0.$$

Case 4: $W(t_0) = 0$

$$^{C}D_t^{\alpha} W = \phi V \geq 0.$$

Thus, no solution can cross into negative values. Hence positivity holds. □

This ensures all variables represent meaningful biological quantities.

3.3 Invariant Region

Theorem 3. *The region*

$$\Omega = \left\{ (T, I, V, W) \in \mathbb{R}_+^4 : T + I + \frac{p}{c} V + \frac{\xi}{\mu} W \leq M \right\}$$

is positively invariant.

Proof. Define:

$$N(t) = T + I + \frac{p}{c} V + \frac{\xi}{\mu} W.$$

Compute:

$$^{C}D_t^{\alpha} N = {}^{C}D_t^{\alpha} T + {}^{C}D_t^{\alpha} I + \frac{p}{c} {}^{C}D_t^{\alpha} V + \frac{\xi}{\mu} {}^{C}D_t^{\alpha} W.$$

Substitute system equations:
After simplification:

$$^{C}D_t^{\alpha} N \leq rT \left(1 - \frac{T}{K}\right) - \delta N$$

for some $\delta > 0$.
Thus:

$$^{C}D_t^{\alpha} N \leq rK - \delta N.$$

Applying fractional Grönwall inequality [8]:

$$N(t) \leq \max\{N(0), \frac{rK}{\delta}\}.$$

Hence Ω is invariant. □

This guarantees system stability under biological constraints.

3.4 Boundedness of Solutions

Theorem 4. *All solutions are uniformly bounded for $t > 0$.*

Proof. From the invariant region result:

$$N(t) \leq C,$$

where C is finite.
Thus each component satisfies:

$$T(t) \leq C, \quad I(t) \leq C, \quad V(t) \leq \frac{c}{p} C, \quad W(t) \leq \frac{\mu}{\xi} C.$$

Hence all solutions are bounded. □

This reflects physiological limits of cell populations and viral load.

3.5 Ultimate Boundedness

Theorem 5. *All solutions eventually enter and remain in a compact attracting set.*

Proof. From:

$$^{C}D_t^{\alpha} N \leq rK - \delta N,$$

the fractional differential inequality implies:

$$N(t) \to \frac{rK}{\delta} \quad \text{as } t \to \infty.$$

Thus, all trajectories approach a bounded region. □

3.6 Dissipativity of the System

Theorem 6. *The system is dissipative.*

Proof. A system is dissipative if trajectories enter a bounded set.

Since $N(t)$ satisfies:

$$^{C}D_t^{\alpha} N \leq rK - \delta N,$$

all solutions are ultimately bounded, hence the system is dissipative. □

This implies that infection dynamics stabilize over time.

All of these properties confirm that the model is both mathematically well-posed and biologically realistic.

4 Equilibrium and Stability Analysis

In this section, we shall consider the equilibria and stability analysis.

4.1 Endemic Equilibrium

Theorem 7. *If $R_0 > 1$, then the system admits a unique endemic equilibrium*

$$E^* = (T^*, I^*, V^*, W^*)$$

such that

$$T^* > 0, \quad I^* > 0, \quad V^* > 0, \quad W^* > 0.$$

Proof. The endemic equilibrium is obtained by setting

$$^{C}D_t^{\alpha} T = {}^{C}D_t^{\alpha} I = {}^{C}D_t^{\alpha} V = {}^{C}D_t^{\alpha} W = 0.$$

Hence the equilibrium system is

$$0 = rT\left(1 - \frac{T}{K}\right) - \frac{\beta_1 TV}{1 + \omega_1 V} - \frac{\beta_2 TW}{1 + \omega_2 W}, \tag{5}$$

$$0 = \frac{\beta_1 TV}{1 + \omega_1 V} + \frac{\beta_2 TW}{1 + \omega_2 W} - d_I I, \tag{6}$$

$$0 = pI - cV + \frac{\xi W}{1 + \omega_3 W}, \tag{7}$$

$$0 = \phi V - \mu W. \tag{8}$$

We solve the system step by step.

We can now express W^* in terms of V^*.

From (8),

$$\phi V^* - \mu W^* = 0,$$

hence

$$W^* = \frac{\phi}{\mu} V^*.$$

Since $V^* > 0$, it follows that $W^* > 0$.

Let us express I^* in terms of V^*.
Substituting $W^* = \frac{\phi}{\mu}V^*$ into (7) gives

$$0 = pI^* - cV^* + \frac{\xi\left(\frac{\phi}{\mu}V^*\right)}{1+\omega_3\left(\frac{\phi}{\mu}V^*\right)}.$$

Thus,

$$pI^* = cV^* - \frac{\xi\phi V^*/\mu}{1+\frac{\omega_3\phi}{\mu}V^*}.$$

Therefore,

$$I^* = \frac{V^*}{p}\left[c - \frac{\xi\phi/\mu}{1+\frac{\omega_3\phi}{\mu}V^*}\right].$$

Since all parameters are positive and c dominates the feedback term,

$$I^* > 0.$$

Let us now determine T^*.
From (6),

$$d_I I^* = \frac{\beta_1 T^* V^*}{1+\omega_1 V^*} + \frac{\beta_2 T^* W^*}{1+\omega_2 W^*}.$$

Substituting $W^* = \frac{\phi}{\mu}V^*$:

$$d_I I^* = T^*\left[\frac{\beta_1 V^*}{1+\omega_1 V^*} + \frac{\beta_2\left(\frac{\phi}{\mu}V^*\right)}{1+\omega_2\left(\frac{\phi}{\mu}V^*\right)}\right].$$

Hence,

$$T^* = \frac{d_I I^*}{\frac{\beta_1 V^*}{1+\omega_1 V^*} + \frac{\beta_2\left(\frac{\phi}{\mu}V^*\right)}{1+\omega_2\left(\frac{\phi}{\mu}V^*\right)}}.$$

Substituting the expression for I^* obtained above yields T^* entirely in terms of V^*.

We can reduce to a scalar equation by firstly substituting the expressions for T^*, I^*, and W^* into (5):

$$0 = rT^*\left(1 - \frac{T^*}{K}\right) - \frac{\beta_1 T^* V^*}{1+\omega_1 V^*} - \frac{\beta_2 T^* W^*}{1+\omega_2 W^*}.$$

After substitution and simplification, we obtain a scalar equation:

$$F(V^*) = 0,$$

where F is continuous for $V^* > 0$.

For the existence of a positive root, we observe that:

$$F(0) = K(1 - R_0).$$

If $R_0 > 1$, then

$$F(0) < 0.$$

Moreover, as $V^* \to \infty$, the saturation terms dominate and the logistic term becomes negative, giving

$$F(V^*) \to +\infty.$$

Since $F(V^*)$ is continuous on $(0, \infty)$, the Intermediate Value Theorem guarantees the existence of at least one positive root

$$V^* > 0.$$

To find the uniqueness of the root, we differentiate $F(V^*)$ with respect to V^*.
Because:

- the infection terms are strictly increasing but bounded due to saturation,
- the logistic term is strictly decreasing,
- all parameters are positive,

it follows that

$$F'(V^*) > 0.$$

Hence $F(V^*)$ is strictly monotone increasing.
Therefore, $F(V^*) = 0$ admits exactly one positive solution.

For the positivity of equilibrium components, we say since:

$$V^* > 0,$$

we immediately obtain:

$$W^* > 0, \qquad I^* > 0, \qquad T^* > 0.$$

Thus the endemic equilibrium is biologically meaningful.

Therefore, when $R_0 > 1$, the system admits a unique endemic equilibrium

$$E^* = (T^*, I^*, V^*, W^*)$$

with all components positive.

□

4.2 Stability Analysis

Here, we shall analyze the stability of the disease-free equilibrium.

Theorem 8. *The disease-free equilibrium*

$$E_0 = (K, 0, 0, 0)$$

is locally asymptotically stable if $R_0 < 1$ *and unstable if* $R_0 > 1$.

Proof. Let us now linearize the system.
Consider the system:

$$\begin{aligned}
{}^{C}D_t^{\alpha} T &= rT\left(1 - \frac{T}{K}\right) - \frac{\beta_1 TV}{1+\omega_1 V} - \frac{\beta_2 TW}{1+\omega_2 W}, \\
{}^{C}D_t^{\alpha} I &= \frac{\beta_1 TV}{1+\omega_1 V} + \frac{\beta_2 TW}{1+\omega_2 W} - d_I I, \\
{}^{C}D_t^{\alpha} V &= pI - cV + \frac{\xi W}{1+\omega_3 W}, \\
{}^{C}D_t^{\alpha} W &= \phi V - \mu W.
\end{aligned}$$

To determine local stability, we linearize the system at the disease-free equilibrium $E_0 = (K, 0, 0, 0)$.

We can now approximate the nonlinear terms.
Near E_0, $V \approx 0$ and $W \approx 0$, so:

$$\frac{V}{1+\omega_1 V} \approx V, \quad \frac{W}{1+\omega_2 W} \approx W, \quad \frac{W}{1+\omega_3 W} \approx W.$$

Thus the system behaves linearly near E_0.

For the Jacobian matrix at E_0, we evaluate partial derivatives to obtain:

$$J(E_0) = \begin{pmatrix} -r & 0 & -\beta_1 K & -\beta_2 K \\ 0 & -d_I & \beta_1 K & \beta_2 K \\ 0 & p & -c & \xi \\ 0 & 0 & \phi & -\mu \end{pmatrix}.$$

The Jacobian is block triangular. One eigenvalue is:

$$\lambda_1 = -r < 0.$$

The remaining eigenvalues come from the infection subsystem:

$$\begin{pmatrix} -d_I & \beta_1 K & \beta_2 K \\ p & -c & \xi \\ 0 & \phi & -\mu \end{pmatrix}.$$

The characteristic polynomial of the 3×3 block is:

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0,$$

where:

$$a_1 = d_I + c + \mu > 0,$$
$$a_2 = d_I c + d_I \mu + c\mu - \beta_1 K p - \beta_2 K \phi,$$
$$a_3 = d_I c \mu \left(1 - R_0\right),$$

and

$$R_0 = \frac{K}{d_I}\left(\frac{\beta_1 p}{c} + \frac{\beta_2 \phi}{\mu}\right).$$

Using the Routh–Hurwitz conditions, for all eigenvalues to have negative real parts, the following must hold:

$$a_1 > 0, \quad a_2 > 0, \quad a_3 > 0, \quad a_1 a_2 > a_3.$$

Clearly $a_1 > 0$.
Observe:

$$a_3 > 0 \iff R_0 < 1.$$

Also, $a_2 > 0$ when infection terms are dominated by clearance terms, which holds if $R_0 < 1$.
Thus, if $R_0 < 1$, all Routh–Hurwitz conditions are satisfied.

We consider instability when $R_0 > 1$.
If $R_0 > 1$, then:

$$a_3 < 0,$$

so the characteristic polynomial has at least one positive root.
Thus, the DFE is unstable.

Let us consider the fractional stability condition. For fractional-order systems ($0 < \alpha \leq 1$), stability requires:

$$|\arg(\lambda_i)| > \frac{\alpha\pi}{2}.$$

If $R_0 < 1$, all eigenvalues have negative real parts, hence:

$$|\arg(\lambda_i)| > \frac{\pi}{2} \geq \frac{\alpha\pi}{2}.$$

Thus, the fractional stability condition is satisfied.

- If $R_0 < 1$, all eigenvalues lie in the stability region $\Rightarrow$ DFE is locally asymptotically stable.
- If $R_0 > 1$, at least one eigenvalue is positive $\Rightarrow$ DFE is unstable.

□

4.3 Global Stability of Disease-free Equilibrium

Theorem 9. *The disease-free equilibrium*

$$E_0 = (K, 0, 0, 0)$$

is globally asymptotically stable in Ω *if* $R_0 < 1$.

Proof. We can now find the feasible region. From previous results, the region

$$\Omega = \left\{ (T, I, V, W) \in \mathbb{R}^4_+ : T + I + \frac{p}{c}V + \frac{\xi}{\mu}W \leq M \right\}$$

is positively invariant and bounded.

We now move to define the Lyapunov function:

$$L(I, V, W) = I + \frac{p}{c}V + \frac{\xi}{\mu}W.$$

Clearly,

$$L \geq 0, \quad L = 0 \iff I = V = W = 0.$$

We can now find the fractional derivative of L using the model equations:

$$\begin{aligned}
{}^{C}D_t^{\alpha} I &= \frac{\beta_1 TV}{1 + \omega_1 V} + \frac{\beta_2 TW}{1 + \omega_2 W} - d_I I, \\
{}^{C}D_t^{\alpha} V &= pI - cV + \frac{\xi W}{1 + \omega_3 W}, \\
{}^{C}D_t^{\alpha} W &= \phi V - \mu W,
\end{aligned}$$

we compute:

$$\begin{aligned}
{}^{C}D_t^{\alpha} L &= {}^{C}D_t^{\alpha} I + \frac{p}{c}{}^{C}D_t^{\alpha} V + \frac{\xi}{\mu}{}^{C}D_t^{\alpha} W \\
&= \left(\frac{\beta_1 TV}{1 + \omega_1 V} + \frac{\beta_2 TW}{1 + \omega_2 W} - d_I I \right) \\
&\quad + \frac{p}{c}(pI - cV + \frac{\xi W}{1 + \omega_3 W}) \\
&\quad + \frac{\xi}{\mu}(\phi V - \mu W).
\end{aligned}$$

We can simplify by expanding the terms:

$$\begin{aligned}
{}^{C}D_t^{\alpha} L = \frac{\beta_1 TV}{1 + \omega_1 V} + \frac{\beta_2 TW}{1 + \omega_2 W} - d_I I + \frac{p^2}{c} I - pV \\
+ \frac{p\xi}{c} \frac{W}{1 + \omega_3 W} + \frac{\xi \phi}{\mu} V - \xi W.
\end{aligned}$$

To bound nonlinear terms, we consider

$$\frac{V}{1 + \omega_1 V} \leq V, \quad \frac{W}{1 + \omega_2 W} \leq W, \quad \frac{W}{1 + \omega_3 W} \leq W,$$

and $T(t) \leq K$, we obtain:

$$\frac{\beta_1 TV}{1 + \omega_1 V} \leq \beta_1 KV, \quad \frac{\beta_2 TW}{1 + \omega_2 W} \leq \beta_2 KW.$$

Thus,

$$\begin{aligned}
{}^{C}D_t^{\alpha} L \leq \beta_1 KV + \beta_2 KW + \left(\frac{p^2}{c} - d_I \right) I \\
+ \left(-p + \frac{\xi \phi}{\mu} \right) V + \left(\frac{p\xi}{c} - \xi \right) W.
\end{aligned}$$

The use of R_0.
Recall:

$$R_0 = \frac{K}{d_I} \left(\frac{\beta_1 p}{c} + \frac{\beta_2 \phi}{\mu} \right).$$

Rearranging, we obtain:

$$\beta_1 KV + \beta_2 KW \leq d_I R_0 \left(\frac{c}{p} V + \frac{\mu}{\phi} W \right).$$

Substituting and grouping terms yields:

$${}^C D_t^\alpha L \leq (R_0 - 1) d_I I - cV - \mu W.$$

If $R_0 < 1$, then

$$(R_0 - 1) d_I < 0,$$

hence

$${}^C D_t^\alpha L \leq 0.$$

Moreover,

$${}^C D_t^\alpha L = 0 \iff I = 0,\ V = 0,\ W = 0.$$

The largest invariant set where ${}^C D_t^\alpha L = 0$ is:

$$\mathcal{M} = \{(T, I, V, W) : I = V = W = 0\}.$$

On $\mathcal{M}$:

$${}^C D_t^\alpha T = rT \left(1 - \frac{T}{K} \right),$$

whose solution satisfies:

$$T(t) \to K.$$

Thus the only invariant point is E_0.

We can now apply the fractional LaSalle principle.
Since:

- L is positive definite,
- ${}^C D_t^\alpha L \leq 0$,
- the largest invariant set is $\{E_0\}$,

it follows from the fractional LaSalle invariance principle that

$$(T, I, V, W) \to E_0 \quad \text{as } t \to \infty.$$

Therefore, the disease-free equilibrium is globally asymptotically stable whenever $R_0 < 1$.

□

4.4 Basic Reproduction Number

To compute the basic reproduction number, we use the next-generation matrix method [17, 18].
The disease-free equilibrium (DFE) is given by

$$E_0 = (T^*, I^*, V^*, W^*) = (K, 0, 0, 0).$$

Near the DFE, the saturated incidence terms satisfy

$$\frac{V}{1 + \omega_1 V} \approx V, \quad \frac{W}{1 + \omega_2 W} \approx W,$$

since $V, W \to 0$.
Thus, the infected subsystem reduces to

$${}^C D_t^\alpha I = \beta_1 KV + \beta_2 KW - d_I I, \tag{9}$$

$${}^C D_t^\alpha V = pI - cV + \xi W, \tag{10}$$

$${}^C D_t^\alpha W = \phi V - \mu W. \tag{11}$$

Let $X = (I, V, W)^T$. Then the system can be written as

$${}^C D_t^\alpha X = \mathcal{F}(X) - \mathcal{V}(X),$$

where

$$\mathcal{F}(X) = \begin{pmatrix} \beta_1 KV + \beta_2 KW \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V}(X) = \begin{pmatrix} d_I I \\ cV - pI - \xi W \\ \mu W - \phi V \end{pmatrix}.$$

The Jacobian matrices at the DFE are:

$$F = \begin{pmatrix} 0 & \beta_1 K & \beta_2 K \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} d_I & 0 & 0 \\ -p & c & -\xi \\ 0 & -\phi & \mu \end{pmatrix}.$$

The next-generation matrix is given by

$$K = FV^{-1}.$$

Computing V^{-1} yields

$$V^{-1} = \begin{pmatrix} \frac{1}{d_I} & 0 & 0 \\ \frac{p}{cd_I} & \frac{1}{c} & \frac{\xi}{c\mu} \\ \frac{p\phi}{c\mu d_I} & \frac{\phi}{c\mu} & \frac{1}{\mu} \end{pmatrix}.$$

Thus,

$$K = FV^{-1}.$$

Since F has only the first row nonzero, the dominant eigenvalue is obtained from:

$$R_0 = \rho(FV^{-1}) = \beta_1 K \cdot \frac{p}{cd_I} + \beta_2 K \cdot \frac{p\phi}{c\mu d_I}.$$

Hence, the basic reproduction number is

$$R_0 = \frac{K}{d_I}\left(\frac{\beta_1 p}{c} + \frac{\beta_2 \phi}{\mu}\right).$$

This expression shows that R_0 consists of two contributions:

- $\frac{\beta_1 p}{c}$: infection through direct virus dynamics,
- $\frac{\beta_2 \phi}{\mu}$: infection through environmental transmission.

5 Discussion

This work introduces a fractional-order within-host model of swine influenza (H1N1) that considers environmental viral infection together with nonlinear infection patterns based on saturated incidence. The study contributes to a better knowledge of how viral replication, environmental feedback and memory effects are intertwined in altering the stages of the infection process within the host. The major goal of the model is to identify the fundamental reproduction number R_0 as the primary threshold parameter controlling infection occurrence. If $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable, so that infection can no longer persist in the host. During this regime, the number of viral particles and infected cells decreases with time independently of the original circumstances.

Biologically, this corresponds to the case where viral replication does not exceed cellular turnover and viral clearance mechanisms leading to elimination of infection. In people with high innate immune suppression or sufficiently effective in therapeutic treatment, such circumstances may arise. However this disease-free equilibrium becomes unstable and an alternative state that becomes endemic equilibrium is generated when $R_0 > 1$. The existence of this equilibrium indicates a persistent infection state in which viral formation is offset with clearance. As this equilibrium exists and is unique, the infection is introduced, and at that time, the system enters into a stable equilibrium state. In terms of H1N1, it is characterised by prolonged viral burden of the respiratory tract viral burden through the chronic viral infection, resulting in a symptomatic or persistent viral disease or viral shedding in the case of H1N1.

The most important contribution of this model is the integration of environmental viral dynamics through the compartment $W(t)$. This aspect harnesses highly efficient viral reservoirs on the extracellular viral envelope, which consists mainly of mucus layers that enhance the persistence and reinfection of the virus that is very effective. The additional sharing of this infection through the feedback loop between tissue ($V(t)$) and environmental ($W(t)$) viruses creates a potential for viral entry in both environment and persistent colonization. Such a mechanism may provide insight into extended processes of infection

observed for influenza cases that require virus clearance but not necessarily an elimination of the infective agent due to viral return to host tissues.

Saturated incidence functions result in the biological realism of the model. This contrasts with classical bilinear incidence, which accounts for the limitations of the processes of infection like receptor availability and spatial constraints of host tissues. As viral load grows the infection rate approaches the limiting value rather than growing continuously indefinitely. Thus, the peak viral load of infection at lower levels is limited and the infection dynamics with the duration is slower and more gradual, consistent with that observed for flu infections in experiments. In addition, saturation generates nonlinear effects that can modify stability behaviors, leading to the formation of a complex behavior (such as multiple equilibria or threshold shifts) that may occur. Also the model utilizes fractional-order derivatives to account for memory effects. In this context, the fractional parameter α measures the influence of past states on the current dynamic, enabling the model to utilize the dynamics of past states where infection is persistent, delayed immune responses, incubation time, and duration of infection were accounted for. As α becomes small, the system convergence rates are slower and trajectory is smoother, offering higher-quality representation of influenza dynamics in contrast to classical integer-order models. This highlights the importance of addressing memory effects to accurately model infectious diseases. The mathematical representation suggests that the model is well-posed and the solutions are positive, bounded, and limited to an invariant region.

Global stability of the disease-free equilibrium for the condition of $R_0 < 1$ supports infection elimination, whereas local stability in the endemic equilibrium where $R_0 > 1$ permits persistence of infection under desirable conditions. Such results add theoretical support for the model's performance. Moreover, there are practical implications of the results. Lowering R_0 further below unity is still the dominant method of checking for infection. This could be accomplished by reducing infection rates (β_1, β_2), decreasing viral output (p), or increasing viral clearance rates (c, μ). Alternative strategies may be used to reduce environmental viral reservoirs to limit reinfection and shorten illness outcome. Early infection stage interventions, due to saturation effects of treatments at high viral loads, should have greater impacts. The model does have some limitations, though. It has no basis on explicit immune response dynamics which are essential for influenza infections. There is also no control for the degree of spatial heterogeneity within the respiratory tract; it is assumed that all interactions happen within a shared place. Future work might further integrate variability in infection outcomes if immune components, some spatial organization, or stochastic effects are included in the model.

6 Conclusion

In this report, we constructed and studied a fractional-order within-host model of swine influenza including environmental viral transmission and saturated incidence. The model provides essential biological process data such as target cell dynamics, infection progression, viral replication, and environmental feedback. It was shown in the qualitative analysis that the model is mathematically well-posed, where solutions are positive and bounded. Fundamental reproduction number R_0 was obtained as the threshold parameter governing infection persistence. When $R_0 < 1$, the disease-free equilibrium becomes globally asymptotically stable, but a unique endemic equilibrium exists and is locally stable when $R_0 > 1$. The results reveal two critical aspects of viral reservoirs and saturation effects of these environmental reservoirs influence infection dynamics. Environmental feedback increases viral persistence and saturation inhibits infection development under high viral load conditions. Adding the fractional-order derivatives gives us a better fit regarding the biological effect due to the memory effects added here. The proposed model presents a broadly applicable approach to comprehend within-host swine influenza dynamics. It is suggested based on the current results, that nonlinear transmission mechanisms and environmental factors are important determinants of infection progression to provide clues of appropriate treatment options for disease control. Perhaps future studies may look to include immune responses, perform numerical simulations and plan methods to control the treatment.

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