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# TRANSMISSION MODEL OF INFLUENZA A WITH ASYMPTOMATIC INFECTION AND ENVIRONMENTAL TRANSMISSION

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**Abstract:** The present study aims to investigate the impact of asymptomatically infected individuals and the presence of free virus in the environment on the transmission of influenza A virus. To this end, an infectious disease model of influenza A virus with asymptomatic infection and environmental transmission is established.Initially, the nonnegativity and boundedness of the global positive solution of the model are obtained, and the fundamental regeneration number of the model $R_0$, is determined by the method of the spectral radius of the next-generation operator.Utilising qualitative ordinary differential equations, stability theory and fluctuation priming, it is demonstrated that the disease-free equilibrium point is globally asymptotically stable at $R_0 < 1$. Furthermore, the consistent persistence of the disease is substantiated by constructing an auxiliary system at $R_0 > 1$. The validity of the theoretical results is substantiated by numerical simulations.The innovative aspect of this paper is the integration of asymptomatic infection and environmental transmission into a unified model. This comprehensive approach elucidates the transmission mechanism of influenza A within the population, thus offering a novel perspective through which to attain a more profound comprehension of the transmission of influenza A.
**Keywords:** A symptomatic infection; Multiple pathways of transmission; Basic regeneration number; Global asymptotic stabilization; Uniform persistence

## 1 INTRODUCTION

Influenza A, is an acute respiratory infection caused by influenza A viruses. Influenza A viruses are highly mutable and can spread rapidly among populations by droplet transmission, direct contact, and airborne aerosols[1-3]. Furthermore, the capacity of influenza A viruses to persist and propagate on environmental surfaces underscores the potential for their transmission under diverse environmental conditions, which in turn may shape their propagation within the population[4-5]. The rapid and widespread dissemination of influenza A viruses has profound consequences for public health, leading to significant morbidity and mortality, as well as substantial economic losses[6]. Historical precedent demonstrates the capacity of influenza A to spark pandemics, as evidenced by the H1N1 epidemic of 2009[7]. Consequently, the development of effective prevention and control strategies for influenza A is of paramount importance. The mutability of influenza A viruses poses a significant challenge in the implementation of traditional prevention and control measures, which often prove ineffective in fully containing their spread. This underscores the necessity for continuous exploration of novel prevention and control strategies.

Mathematical modeling has emerged as a valuable instrument in the study of infectious disease transmission, facilitating our understanding of the underlying mechanisms and enabling accurate epidemic forecasting. This, in turn, provides a robust scientific foundation for the development of effective public health policies. In recent years, with the advancement of computing capabilities and the development of mathematical theory, the field of infectious disease modeling has garnered significant attention[8]. Notably, in the context of respiratory infectious diseases, such as influenza A, the application of mathematical models has yielded noteworthy outcomes[9].

In light of the aforementioned discussion, the objective of this paper is to develop an infectious disease model of influenza A virus with asymptomatic infection and environmental transmission to study the kinetic behavior of influenza A transmission. The structure of this paper is outlined as follows: The initial section of this study is devoted to the modeling process. Subsequent sections are dedicated to the verification of two crucial properties of the global positive solution of the model: its nonnegativity and its boundedness. The third section involves the derivation of the fundamental regeneration number of the model and the demonstration of the existence and uniqueness of the disease-free equilibrium point. The fourth section focuses on the proof of the global asymptotic stability of the disease-free equilibrium point. The fifth section provides a rigorous justification for the model's consistent persistence. Ultimately, numerical simulations are implemented to validate the accuracy of the obtained results.

## 2 FORMULATION OF THE MODEL

The model under consideration in this paper consists of four human compartments: susceptible ($S$), asymptomatically infected ($A$), symptomatically infected ($I$), and recovered ($R$), and an environmental compartment: the free virus ($W$) contained in the environment and released by the infected person. A diagram illustrating the model's compartmental structure is presented in Figure 1.

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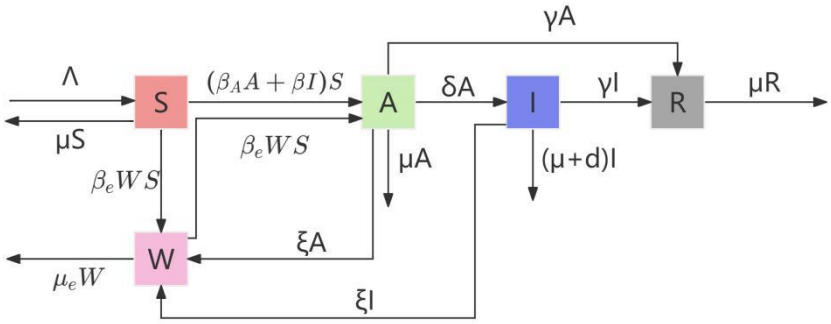


**Figure 1** Diagram of the Model's Chamber

The transmission model of Influenza A is as follows:

$$\begin{cases} \dfrac{dS(t)}{dt} = \Lambda - \beta_A A(t)S(t) - \beta I(t)S(t) - \beta_e W(t)S(t) - \mu S(t) \\ \dfrac{dA(t)}{dt} = \beta_A A(t)S(t) + \beta I(t)S(t) + \beta_e W(t)S(t) - \mu A(t) - \delta A(t) - \gamma A(t) \\ \dfrac{dI(t)}{dt} = \delta A(t) - \gamma I(t) - \mu I(t) - dI(t) \\ \dfrac{dW(t)}{dt} = \xi A(t) + \xi I(t) - \mu_e W(t) \\ \dfrac{dR(t)}{dt} = \gamma A(t) + \gamma I(t) - \mu R(t) \end{cases} \tag{1}$$

In the model, $\Lambda$ represents the birth rate of the population, $\mu$ denotes the natural death rate, d representative case fatality rate for symptomatic infections, $\beta_A$ and $\beta$ respectively represent the transmission rates from asymptomatic and symptomatic infected individuals to susceptible individuals. $\beta_e$ indicates the transmission rate caused by environmental factors, while δ represents the transfer rate from asymptomatic to symptomatic infected individuals. Additionally,$\mu_e$denotes the morbidity-related mortality rate of infected individuals induced by environmental factors. $\gamma$ represent the recovery rates of asymptomatic and symptomatic infected individuals, $\xi$ reflect the rate of viral shedding from asymptomatic and symptomatic infected individuals into the environment.

Given that the differential equations of $S(t), A(t), I(t), W(t)$ in the model do not include $R(t)$, $R(t)$ can be decoupled. Therefore, we consider the subsystem:

$$\begin{cases} \dfrac{dS(t)}{dt} = \Lambda - \beta_A A(t)S(t) - \beta I(t)S(t) - \beta_e W(t)S(t) - \mu S(t) \\ \dfrac{dA(t)}{dt} = \beta_A A(t)S(t) + \beta I(t)S(t) + \beta_e W(t)S(t) - \mu A(t) - \delta A(t) - \gamma A(t) \\ \dfrac{dI(t)}{dt} = \delta A(t) - \gamma I(t) - \mu I(t) - dI(t) \\ \dfrac{dW(t)}{dt} = \xi A(t) + \xi I(t) - \mu_e W(t) \end{cases} \tag{2}$$

For any solution of the system that satisfies the initial conditions $\big(S(0), A(0), I(0), W(0)\big)$ :

$$X(t) = \big(S(t), A(t), I(t), W(t)\big) \tag{3}$$

There exists $X(t) \in \Gamma$,among which,

$$\Gamma = \{(S, A, I, W) \in R_+^4 | S(t) + A(t) + I(t) + W(t) \leq \frac{\xi\Lambda}{\mu\mu_e} + \frac{\Lambda}{\mu}\} \tag{4}$$

## 3 THE NON-NEGATIVITY AND BOUNDEDNESS OF THE MODEL

**Theorem 1:** $\Gamma$ is the maximal positive invariant set of the model (2).

**Proof :** First, we prove the non-negativity. For any solution $\big(S(t), A(t), I(t), W(t)\big)$ that satisfies the initial conditions $\big(S(0), A(0), I(0), W(0)\big)$. Using the contradiction method, assume there exists $t_0$ such that $S(t_0) = 0$ and $\frac{dS(t_0)}{dt} < 0$ , Substituting into the first equation of model (2), we get $\frac{dS(t_0)}{dt} = \Lambda - \beta_A A(t_0)S(t_0) - \beta I(t_0)S(t_0) -$

$\beta_e W(t_0)S(t_0) - \mu S(t_0)$ , Therefore, $\frac{dS(t_0)}{dt} = \Lambda < 0$ , this contradicts $\Lambda > 0$ , thus the assumption is not valid. Therefore, $\frac{dS(t)}{dt} \geq 0$.

Considering $\frac{\mathrm{dA(t)}}{\mathrm{dt}}$, assume that at $\mathrm{t_0}$, $\mathrm{A(t_0)} = 0$ and $\frac{\mathrm{dA(t_0)}}{\mathrm{dt}} < 0$. At $\mathrm{t_1}$, we have $\mathrm{I(t_1)} = 0$ and $\frac{\mathrm{dI(t_1)}}{\mathrm{dt}} < 0$. At $\mathrm{t_2}$, we have $W(t_2) = 0$ and $\frac{\mathrm{dW(t_2)}}{\mathrm{dt}} < 0$ . Taking $\mathrm{t} = \min\{\mathrm{t_0}, \mathrm{t_1}, \mathrm{t_2}\}$, if $\mathrm{t} = \mathrm{t_0}$ , then $\mathrm{I(t)} \geq 0$ and $\mathrm{W(t)} \geq 0$ . Thus, $\frac{\mathrm{dA(t_0)}}{\mathrm{dt}} = \beta \mathrm{I(t_0)S(t_0)} + \beta_e \mathrm{W(t_0)S(t_0)} \geq 0$, Since $\frac{\mathrm{dA(t_0)}}{\mathrm{dt}} < 0$ leads to a contradiction, we conclude that $\frac{dA(t)}{dt} \geq 0$.

Similarly, for $\frac{dI(t)}{dt}$ and $\frac{\mathrm{dW(t)}}{\mathrm{dt}}$, the above method can be applied to demonstrate their non-negativity.In summary, all state variables in model (2) are non-negative.

Next, we verify the boundedness. Summing the first three equations in model (2), we obtain:

$$\mathrm{d(S + A + I)} = \Lambda - \mu \mathrm{S} - \mu \mathrm{A} - \gamma \mathrm{A} - \gamma \mathrm{I} - \mathrm{dI} - \mu \mathrm{I} \leq \Lambda - \mu(\mathrm{S + A + I}) \tag{5}$$

Thus,

$$S + A + I \leq \frac{\Lambda}{\mu} \tag{6}$$

Moreover, since

$$\mathrm{dW} = \xi(\mathrm{A + I}) - \mu_e \mathrm{W} \leq \frac{\xi \Lambda}{\mu} \tag{7}$$

it follows that:

$$\mathrm{W} \leq \frac{\xi \Lambda}{\mu \mu_e} \tag{8}$$

Consequently,

$$\mathrm{S + A + I + W} \leq \frac{\xi \Lambda}{\mu \mu_e} + \frac{\Lambda}{\mu} \tag{9}$$

Clearly, we already know that $S + A + I + W \geq 0$. Therefore, the state variables in model (2) are all bounded.

In conclusion, the theorem 1 is established. That is, Γ is the maximal positive invariant set of model (2).

## 4 EXISTENCE OF THE BASIC REPRODUCTION NUMBER AND THE DISEASE-FREE EQUILIBRIUM POINT

In model (2), when $I = 0$, according to the second and fourth equations, it follows that $A = 0$ and $\mathrm{W} = 0$. Substituting into the first equation yields $S = \frac{\Lambda}{\mu}$, Therefore, model (2) has a unique disease-free equilibrium point $E_0 = \left(\frac{\Lambda}{\mu}, 0,0,0\right)$.

Next, we will calculate the basic reproduction number using the next-generation matrix method [10].

Rewrite the equations in model (2):

$$\begin{cases} \dfrac{dA(t)}{dt} = \beta_{\mathrm{A}} A(t)S(t) + \beta I(t)S(t) + \beta_e W(t)S(t) - \mu A(t) - \delta A(t) - \gamma A(t) \\ \dfrac{dI(t)}{dt} = \delta A(t) - \gamma I(t) - \mu I(t) - dI(t) \\ \dfrac{dW(t)}{dt} = \xi A(t) + \xi I(t) - \mu_e W(t) \end{cases} \tag{10}$$

as,

$$\frac{dA}{dt} = \mathcal{F}_1\big(S(t), A(t), I(t), W(t)\big) - \mathcal{V}_1\big(S(t), A(t), I(t), W(t)\big) \tag{11}$$

$$\frac{dI}{dt} = \mathcal{F}_2\big(S(t), A(t), I(t), W(t)\big) - \mathcal{V}_2\big(S(t), A(t), I(t), W(t)\big) \tag{12}$$

$$\frac{dW}{dt} = \mathcal{F}_3\big(S(t), A(t), I(t), W(t)\big) - \mathcal{V}_3\big(S(t), A(t), I(t), W(t)\big) \tag{13}$$

where,

$$\mathscr{F}_1\big(S(t), A(t), I(t), W(t)\big) = \beta_A A(t)S(t) + \beta I(t)S(t) + \beta_e W(t)S(t) \tag{14}$$

$$\mathscr{F}_2\big(S(t), A(t), I(t), W(t)\big) = \delta A(t) \tag{15}$$

$$\mathscr{F}_3\big(S(t), A(t), I(t), W(t)\big) = \xi A(t) + \xi I(t) \tag{16}$$

$$\mathcal{V}_1\big(S(t), A(t), I(t), W(t)\big) = \mu A(t) + \delta A(t) + \gamma A(t) \tag{17}$$

$$\mathcal{V}_2\big(S(t), A(t), I(t), W(t)\big) = \gamma I(t) + \mu I(t) + dI(t) \tag{18}$$

$$\mathcal{V}_3\big(S(t), A(t), I(t), W(t)\big) = \mu_e W(t) \tag{19}$$

leads to the matrices $\mathscr{F} = \begin{pmatrix} \mathscr{F}_1 \\ \mathscr{F}_2 \\ \mathscr{F}_3 \end{pmatrix}$ and $\mathcal{V} = \begin{pmatrix} \mathcal{V}_1 \\ \mathcal{V}_2 \\ \mathcal{V}_3 \end{pmatrix}$.The Jacobian matrices at the equilibrium point $E_0 = \left(\frac{\Lambda}{\mu}, 0,0,0\right)$ are represented by

$$F = \begin{pmatrix} \frac{\partial \mathcal{F}_1}{\partial A} & \frac{\partial \mathcal{F}_1}{\partial I} & \frac{\partial \mathcal{F}_1}{\partial W} \\ \frac{\partial \mathcal{F}_2}{\partial A} & \frac{\partial \mathcal{F}_2}{\partial I} & \frac{\partial \mathcal{F}_2}{\partial W} \\ \frac{\partial \mathcal{F}_3}{\partial A} & \frac{\partial \mathcal{F}_3}{\partial I} & \frac{\partial \mathcal{F}_3}{\partial W} \end{pmatrix} = \begin{pmatrix} \beta_A & \beta & \beta_e \\ \delta & 0 & 0 \\ \xi & \xi & 0 \end{pmatrix} \tag{20}$$

and

$$V = \begin{pmatrix} \frac{\partial \mathcal{V}_1}{\partial A} & \frac{\partial \mathcal{V}_1}{\partial I} & \frac{\partial \mathcal{V}_1}{\partial W} \\ \frac{\partial \mathcal{V}_2}{\partial A} & \frac{\partial \mathcal{V}_2}{\partial I} & \frac{\partial \mathcal{V}_2}{\partial W} \\ \frac{\partial \mathcal{V}_3}{\partial A} & \frac{\partial \mathcal{V}_3}{\partial I} & \frac{\partial \mathcal{V}_3}{\partial W} \end{pmatrix} = \begin{pmatrix} \mu+\delta+\gamma & 0 & 0 \\ 0 & \gamma+\mu+d & 0 \\ 0 & 0 & \mu_e \end{pmatrix} \tag{21}$$

For the sake of computational simplicity, the matrices F and V are reformulated as:

$$F_1 = \begin{pmatrix} \beta_A & \beta & \beta_e \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} V_1 = \begin{pmatrix} \mu+\delta+\gamma & 0 & 0 \\ -\delta & \gamma+\mu+d & 0 \\ -\xi & -\xi & \mu_e \end{pmatrix} \tag{22}$$

After computation, the results yield:

$$V_1^{-1} = \begin{pmatrix} \frac{1}{\mu+\delta+\gamma} & 0 & 0 \\ \frac{-\delta}{(\mu+\delta+\gamma)(\gamma+\mu+d)} & \frac{1}{\gamma+\mu+d} & 0 \\ \frac{\delta\xi+\xi(\gamma+\mu+d)}{(\mu+\delta+\gamma)(\gamma+\mu+d)\mu_e} & \frac{\xi}{(\gamma+\mu+d)\mu_e} & \mu_e \end{pmatrix} \tag{23}$$

Define the next-generation matrix as $\mathrm{M} = \mathrm{FV}^{-1}$. Then, it follows that:

$$M = \begin{pmatrix} \frac{\beta_A(\gamma+\mu+d)\mu_e+\beta\delta\mu_e+\delta\xi\beta_e+\xi\beta_e(\gamma+\mu+d)}{(\mu+\delta+\gamma)(\gamma+\mu+d)\mu_e} & \frac{\beta\mu_e+\xi\beta_e}{(\gamma+\mu+d)\mu_e} & \frac{\beta_e}{\mu_e} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{24}$$

The basic reproduction number $R_0$ is defined as the spectral radius of the matrix M. Thus, $R_0$ is given by:

$$R_0 = \frac{\beta_A\mu_e(\gamma+\mu+d)+\beta\delta\mu_e+\xi\beta_e(\gamma+\mu+d+\delta)}{(\mu+\delta+\gamma)(\gamma+\mu+d)\mu_e} \cdot \frac{\Lambda}{\mu} \tag{25}$$

## 5 THE STABILITY OF THE DISEASE-FREE EQUILIBRIUM POINT

**Theorem 2**: When $R_0 < 1$, the disease-free equilibrium point $E_0 = \left(\frac{\Lambda}{\mu}, 0,0,0\right)$ is locally asymptotically stable.

**Proof**: The Jacobian matrix at $E_0$ for Model (2) is given by:

$$J(E_0) = \begin{bmatrix} -\mu & \frac{\beta_A\Lambda}{\mu} & \frac{\beta\Lambda}{\mu} & \frac{\beta_e\Lambda}{\mu} \\ 0 & \frac{\beta_A\Lambda}{\mu} - \mu - \delta - \gamma & \frac{\beta\Lambda}{\mu} & \frac{\beta_e\Lambda}{\mu} \\ 0 & \delta & -\gamma-\mu-d & 0 \\ 0 & \xi & \xi & -\mu_e \end{bmatrix} \tag{26}$$

It is evident that $J(E_0)$ has one negative eigenvalue $\lambda_1 = -\mu < 0$, The remaining eigenvalues satisfy the equation:

$$(\lambda - \frac{\beta_A\Lambda}{\mu} + \mu + \delta + \gamma)(\lambda + \gamma + \mu + \mathrm{d})(\lambda + \mu_e) = \frac{\beta_e\Lambda\xi}{\mu}(\delta + \lambda + \gamma + \mu + \mathrm{d}) + \frac{\beta\Lambda\xi}{\mu}(\lambda + \mu_e) \tag{27}$$

Assuming that when $R_0 < 1$, there exists an eigenvalue $\lambda^*$, and $Re(\lambda^*) \geq 0$, then we divide both sides of (26) by:

$$\left(\lambda^* - \frac{\beta_A\Lambda}{\mu} + \mu + \delta + \gamma\right)(\lambda^* + \gamma + \mu + d)(\lambda^* + \mu_e) \tag{28}$$

Taking the absolute value, we obtain:

$$1 = \left|\frac{\beta_e\xi\Lambda}{(\mu\lambda^*+\beta_A\Lambda+\mu^2+\mu\delta+\mu\gamma)(\lambda^*+\mu_e)} + \frac{\beta\Lambda\delta}{(\mu\lambda^*+\beta_A\Lambda+\mu^2+\mu\delta+\mu\gamma)(\lambda^*+\gamma+\mu+d)} + \frac{\beta_e\Lambda\xi\delta}{(\mu\lambda^*-\beta_A\Lambda+\mu^2+\mu\delta+\mu\gamma)(\lambda^*+\gamma+\mu+d)(\lambda^*+\mu_e)}\right| \tag{29}$$

Let $\lambda^* = a + bi, a \geq 0$, then: $|\lambda + \mu_e|\, a + \mu_e \geq \mu_e,\ |\lambda + \gamma + \mu + d| \geq a + \gamma + \mu + d \geq \gamma + \mu + d\ |\mu\lambda + \beta_A\Lambda + \mu^2 + \mu\delta + \mu\gamma| \geq |\mu a + \beta_A\Lambda + \mu^2 + \mu\delta + \mu\gamma| \geq |\beta_A\Lambda + \mu^2 + \mu\delta + \mu\gamma|$.

Thus,

$$1 = \left|\frac{\beta_e\xi\Lambda}{(\mu\lambda^*+\beta_A\Lambda+\mu^2+\mu\delta+\mu\gamma)(\lambda^*+\mu_e)} + \frac{\beta\Lambda\delta}{(\mu\lambda^*+\beta_A\Lambda+\mu^2+\mu\delta+\mu\gamma)(\lambda^*+\gamma+\mu+d)} + \frac{\beta_e\Lambda\xi\delta}{(\mu\lambda^*-\beta_A\Lambda+\mu^2+\mu\delta+\mu\gamma)(\lambda^*+\gamma+\mu+d)(\lambda^*+\mu_e)}\right| \leq \left|\frac{\beta_e\Lambda\xi}{(\beta_A\Lambda+\mu^2+\mu\delta+\mu\gamma)\mu e} + \frac{\beta\Lambda\delta}{(\beta_A\Lambda+\mu^2+\mu\delta+\mu\gamma)(\gamma+\mu+d)} + \frac{\beta_e\Lambda\xi\delta}{(\beta_A\Lambda+\mu^2+\mu\delta+\mu\gamma)(\gamma+\mu+d)\mu_e}\right| = \frac{\beta\delta\mu_e+\xi\beta_e(\gamma+\mu+d+\delta)}{\left(\frac{\beta_A\Lambda}{\mu}+\mu+\delta+\gamma\right)(\gamma+\mu+d)\mu_e} \cdot \frac{\Lambda}{\mu} \tag{30}$$

Moreover, based on the result for $\mathrm{R}_0$, it can be reformulated as follows:

$$\frac{\beta\delta\mu_e+\xi\beta_e(\gamma+\mu+d+\delta)}{(\mu+\delta+\gamma)(\gamma+\mu+d)\mu_e} \cdot \frac{\Lambda}{\mu} = R_0 - \frac{\beta_A\mu_e(\gamma+\mu+d)}{(\mu+\delta+\gamma)(\gamma+\mu+d)\mu_e} \cdot \frac{\Lambda}{\mu} \tag{31}$$

Consequently, we deduce that $1 \leq R_0 - \frac{\beta_A\mu_e(\gamma+\mu+d)}{(\mu+\delta+\gamma)(\gamma+\mu+d)\mu_e} \cdot \frac{\Lambda}{\mu}$ is in direct contradiction with $R_0 < 1$, thereby demonstrating that the initial assumption is invalid.

Therefore, when $R_0 < 1$, all the eigenvalues of $J(E_0)$ exhibit negative real parts, implying that the disease-free equilibrium point of model (2) is locally asymptotically stable.

**Lemma 1[11]**: Let $B$ be bounded and continuously differentiable. Then there exist two sequences $\{s_n\}$ and $\{t_n\}$ such that $s_n \to \infty, t_n \to \infty, B(s_n) \to B_\infty, B'(s_n) \to 0, B(t_n) \to B^\infty$ and $B'(t_n) \to 0$ as $n \to \infty$.

**Theorem 3**: When $R_0$ <1, the disease-free equilibrium point $E_0 = \left(\frac{\Lambda}{\mu}, 0,0,0\right)$is globally asymptotically stable.

**Proof**: For the model (10):

$$\begin{cases} \dfrac{dA(t)}{dt} = \beta_A A(t)S(t) + \beta I(t)S(t) + \beta_e W(t)S(t) - \mu A(t) - \delta A(t) - \gamma A(t) \\ \dfrac{dI(t)}{dt} = \delta A(t) - \gamma I(t) - \mu I(t) - dI(t) \\ \dfrac{dW(t)}{dt} = \xi A(t) + \xi I(t) - \mu_e W(t) \end{cases} \tag{10}$$

Let the sequence $\{t_n\}$, substituting this into the model yields:

$$\begin{cases} \dfrac{dA(t_n)}{dt} = \beta_A A(t_n)S(t_n) + \beta I(t_n)S(t_n) + \beta_e W(t_n)S(t_n) - \mu A(t_n) - \delta A(t_n) - \gamma A(t_n) \\ \dfrac{dI(t_n)}{dt} = \delta A(t_n) - \gamma I(t_n) - \mu I(t_n) - dI(t_n) \\ \dfrac{dW(t_n)}{dt} = \xi A(t_n) + \xi I(t_n) - \mu_e W(t_n) \end{cases} \tag{32}$$

From Lemma 1, it can be concluded that when $t_n \to \infty$, $A(t_n) \to A^\infty$, $I(t_n) \to I^\infty$ and $I'(t_n) \to 0$. Therefore, it can be derived from the second equation of the system of equations (32) that:

$$\frac{dI(t_n)}{t_n} \le \delta A^\infty - (\gamma + \mu + d)I^\infty, 0 \le \delta A^\infty - (\gamma + \mu + d)I^\infty \tag{33}$$

$$I^\infty \le \frac{\delta}{\gamma+\mu+d} A^\infty \tag{34}$$

Similarly, it can be inferred from the third equation of the system of equations (4) that $W^\infty \le \frac{\xi}{\mu_e} + \frac{\xi\delta}{\mu_e(\gamma+\mu+d)} A^\infty$, For the first equation of the system of equations (4) : $\frac{dA(t_n)}{dt} \le \beta_A A^\infty S^\infty + \beta I^\infty S^\infty + \beta_e W^\infty S^\infty - (\mu + \delta + \gamma)A^\infty$, it can be readily deduced that : $S^\infty \le \frac{\Lambda}{\mu}$, By substituting the values, it can be derived that : $0 \le \left[\beta_A \frac{\Lambda}{\mu} + \beta \frac{\Lambda}{\mu} \frac{\delta}{\gamma+\mu+d} + \beta_e \frac{\Lambda}{\mu}\left(\frac{\xi}{\mu_e} + \frac{\xi\delta}{\mu_e(\gamma+\mu+d)}\right) - (\mu + \delta + \gamma)\right] A^\infty$, it can be concluded that : $A^\infty \le R_0 A^\infty$, Additionally, given that $R_0 < 1$, it can be inferred that $A^\infty = 0$, which subsequently leads to the conclusion that $I^\infty = 0$ and $W^\infty = 0$. From this, it can be deduced that $A \to 0, I \to 0, W \to 0$.

Let us further define a sequence $\{s_n\}$. Substituting this sequence into the first equation of Model (2) results in the following expression:

$$\frac{dS(s_n)}{ds_n} = \Lambda - \beta_A A(s_n)S(s_n) - \beta I(s_n)S(s_n) - \beta_e W(s_n)S(s_n) - \mu S(s_n) \tag{35}$$

From Lemma 1, it can be concluded that when $s_n \to \infty$, $\frac{dS(s_n)}{ds_n} \to 0$, $A(t_n) \to A^\infty = 0$ $I(t_n) \to I^\infty = 0$ and $W(t_n) \to W^\infty = 0$. Therefore, $0 = \Lambda - \mu S_\infty$, $S_\infty = \frac{\Lambda}{\mu}$, then $S_\infty = S^\infty = \frac{\Lambda}{\mu}$. Thus, the disease-free equilibrium point $E_0$ is globally attractive. Furthermore, according to Theorem 2, the disease-free equilibrium point $E_0$ is locally asymptotically stable, thus indicating that $E_0$ is globally asymptotically stable.

## 6 UNIFORM PERSISTENCE OF THE MODEL

**Theorem 4**: When $R_0 > 1$, the model (2) demonstrated uniform persistence behavior. That is, there exists $\varepsilon > 0$ such that :

$$\lim_{t\to\infty} inf\big(A(t), I(t), W(t)\big) \ge (\varepsilon, \varepsilon, \varepsilon)$$

**Proof:** Dfine $x = \{S(t), A(t), I(t), W(t) | S + A + I + W \le \frac{\xi\Lambda}{\mu\mu_e} + \frac{\Lambda}{\mu}\}$, where $x_0 = \{(S, A, I, W) \in x | S, A, I, W > 0\}$, let $\partial x_0 = x \setminus x_0$. From model (2), we obtain :

$$A(t) \ge A(t_0)e^{-(\mu+\delta+\gamma)(t-t_0)} \ge 0 \tag{36}$$

$$I(t) \ge I(t_0)e^{-(\mu+d+\gamma)(t-t_0)} \ge 0 \tag{37}$$

$$W(t) \ge W(t_0)e^{-\mu_e(t-t_0)} \ge 0 \tag{38}$$

Thus, $x$ and $x_0$ are positively invariant sets, and $\partial x_0$ is relative closed set of $x$ let:

$$M_\partial = \{S(0), A(0), I(0), W(0) | S(t), A(t), I(t), W(t) \in \partial x_0\}, \forall t \ge 0$$

It is demonstrated that $M_\partial = \{S(0), 0,0,0 | S(t) \ge 0\}$ holds true, clearly, $\{S(0), 0,0,0 | S(t) \ge 0\} \subseteq M_\partial$ and to prove $M_\partial \subseteq \{S(0), 0,0,0 | S(t) \ge 0\}$. Let $\big(S(0), A(0), I(0), W(0)\big) \in M_\partial$. It is essential to establish that for all $\forall t \ge 0$, $A(t) =$

$0, I(t) = 0, W(t) = 0$. This argument employs proof by contradiction, assuming otherwise, there exists a $\exists t_0 \geq 0$ such that:

*(i)* $A(t_0) > 0$ *(ii)* $I(t_0) > 0$ *(iii)* $W(t_0) > 0$

For (ii), solving for (37) yields: For all $t > t_0$, there exists $I(t) > 0$, substituting into model (2) yields $A(t) > 0, W(t) > 0$, thus $\big(S(t), A(t), I(t), W(t)\big) \notin \partial x_0$, This contradicts the assumption. A contradiction can be derived in a similar manner for (iii). For (i), when $t > t_0$, it can be inferred that $I(t) = I(t_0)e^{(\gamma+\mu+d)} + \int_{t_0}^{t} k\delta A(t)e^{(\mu+\delta+\gamma)}\, d_t$. Clearly, when $A(t) > 0$, we have $I(t) > 0$. Similarly, it follows that $W(t) > 0$.

In summary, $\big(S(t), A(t), I(t), W(t)\big) \notin \partial x_0$ contradicts the hypothesis. Thus, it is proven $M_\partial = \{S(0), 0,0,0 | S(t) \geq 0\}$. The disease-free equilibrium point $E_0$ of model (2) is globally asymptotically stable, and there is only one equilibrium point $E_0$ in $M_\partial$.

We will next demonstrate that $E_0$ exhibits weak exclusion with respect to the set $x_0$, which requires showing that $\lim\limits_{t\to\infty} \sup d\big(\Phi(t), E_0\big) > 0$. It suffices to prove that $W^s_{(E_0)} \cap x_0 = \emptyset$. Using a proof by contradiction, we assume that this conclusion is not valid. Therefore, there exists a positive solution $\big(S(t), A(t), I(t), W(t)\big)$ for model (2), such that $\lim\limits_{t\to\infty} \big(S(t), A(t), I(t), W(t)\big) = (S^0, 0,0,0)$.

Define $M = F - V$, given that $R_0 > 1$, it follows that $S(M) > 0$, For sufficiently small $\varepsilon > 0$, there exists $S(M - M_\varepsilon) > 0$. In this context,

$$M_\varepsilon = \begin{pmatrix} \beta_A\varepsilon & \beta\varepsilon & \beta_e\varepsilon \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{39}$$

There exists $T > 0$ such that $\forall t > T$, there holds $S^0 - \varepsilon < S(t) < S^0 + \varepsilon$, leading to the derivation of the differential inequality:

$$\begin{cases} \frac{dA}{dt} \geq \beta_A(S^\circ - \varepsilon)A + \beta(S^\circ - \varepsilon)I + \beta_e(S^\circ - \varepsilon)W - (\mu + \delta + \gamma)A \\ \frac{dI}{dt} = \delta A - (\gamma + \mu + d)I \\ \frac{dW}{dt} = \xi A + \xi I - \mu_e W \end{cases} \tag{40}$$

Consider the auxiliary system:

$$\begin{cases} \frac{dA}{dt} = \beta_A(S^\circ - \varepsilon)A + \beta(S^\circ - \varepsilon)I + \beta_e(S^\circ - \varepsilon)W - (\mu + \delta + \gamma)A \\ \frac{dI}{dt} = \delta A - (\gamma + \mu + d)I \\ \frac{dW}{dt} = \xi A + \xi I - \mu_e W \end{cases} \tag{41}$$

Because $S(M - M_\varepsilon) > 0$, as $t \to \infty$, it follows that $A(t) \to \infty, I(t) \to \infty, W(t) \to \infty$. This contradicts the assumption that $A(t) \to 0, I(t) \to 0, W(t) \to 0$ when $t \to \infty$[12], thus, it is proven: $W^s_{(E_0)} \cap x_0 = \emptyset$.

In summary, it can be obtained that when $R_0 > 1$, the model (2) concerning $(x_0, \partial x_0)$ persists consistently.

## 7 NUMERICAL SIMULATION

Assuming:

$\Lambda = 1.4, \beta_A = 0.0001, \beta = 0.0001, \beta_e = 0.003, \mu = 0.01, \gamma = 0.05, \mu_e = 0.01, d = 0.006, \xi = 0.001, \delta = 0.02$, the calculation yields $R_0 = 0.912, R_0 < 1$. According to Theorem 4, the model (2) has a globally asymptotically stable disease-free equilibrium point, and its numerical simulation is shown in Figure 2.

Assuming:

$\Lambda = 1.4, \beta_A = 0.0001, \beta = 0.0001, \beta_e = 0.01, \mu = 0.007, \gamma = 0.05, \mu_e = 0.01, d = 0.006, \xi = 0.001, \delta = 0.02$, the calculation yields $R_0 = 3.764,\ R_0 > 1$. According to Theorem 5, the model (2) is uniformly persistent, and its numerical simulation is shown in Figure 3.

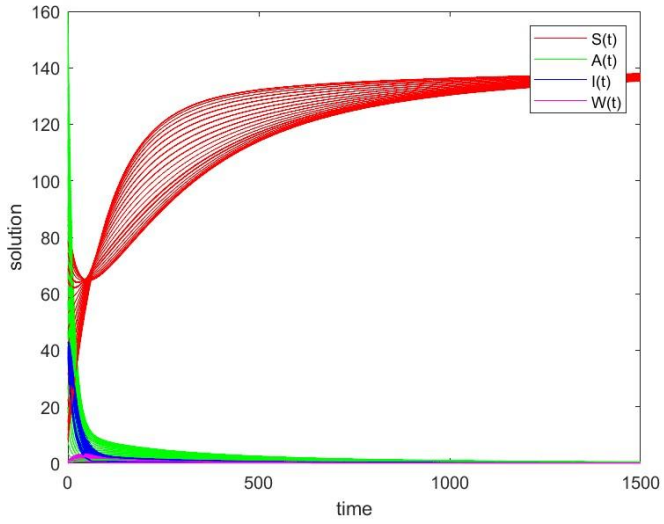


**Figure 2** $R_0 < 1$

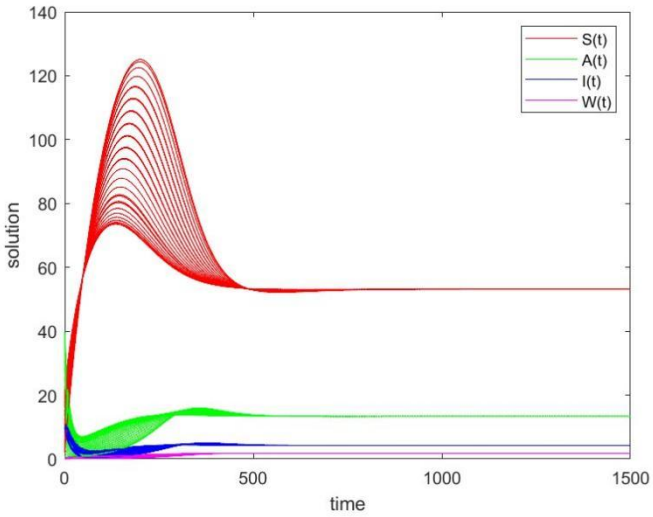


**Figure 3** $R_0 > 1$

## 8 CONCLUSION

This study establishes a comprehensive dynamical model for the transmission of H1N1 influenza virus, considering asymptomatic infection mechanisms and environmental transmission pathways.(SAIWR model). In-depth analysis of the dual roles of asymptomatic carriers and environmental virus transmission mechanisms on the dynamics of epidemic evolution. Firstly, the study rigorously demonstrates the non-negativity and boundedness of the model, thereby ensuring a robust mathematical foundation for epidemiological research. Subsequently, utilizing the next-generation matrix method, this study derives an analytical expression for the basic reproduction number $R_0$, thereby establishing threshold criteria for disease transmission dynamics.The theoretical results are validated through numerical simulations, providing a quantitative assessment tool and a theoretical basis for formulating strategies for epidemic prevention and control.Subsequent studies will prioritize the incorporation of more authentic parameters, the formulation of models such as age structure, and the investigation of the impact of vaccination and antiviral treatment on enhancing the predictive accuracy and public health applicability of these models.

## COMPETING INTERESTS

The authors have no relevant financial or non-financial interests to disclose.

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