GLOBAL RESULTS FOR AN HIV/AIDS MODEL WITH MULTIPLE SUSCEPTIBLE CLASSES AND NONLINEAR INCIDENCE*

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Abstract In this paper, an HIV/AIDS epidemic model is proposed in which there are two susceptible classes. Two types of general nonlinear incidence functions are employed to depict the scenarios of infection among cautious and incautious individuals. Qualitative analyses are performed, in terms of the basic reproduction number $R_0$, to gain the global dynamics of the model: the disease-free equilibrium is of global asymptotic stability when $R_0 \leq 1$; a unique endemic equilibrium exists and globally asymptotically stable $R_0 > 1$. The introduction of cautious susceptible and the resulting multiple transmission functions has positive effect on HIV/AIDS prevalence. Numerical simulations are carried out to illustrate and extend the obtained analytical results.

Keywords HIV/AIDS, cautious susceptible, general nonlinear incidence, basic reproduction number.

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1. Introduction

The human immunodeficiency virus (HIV) infection, leading to the acquired immunodeficiency syndrome (AIDS) [27], continues to be a major public health issue across the globe. In 2017, there were approximately 36.9 million people worldwide living with HIV/AIDS, out of which an estimated 1.8 million individuals became newly infected [22].

Infected individuals advance through several stages of HIV life cycle before developing full-blown AIDS [12]. Without treatment, the majority of HIV-positive people will develop signs of HIV-related illness and even AIDS within 5-10 years. HIV is a sexually transmitted disease in most cases, and it is transmitted by performing unprotected sex with someone who is HIV positive. HIV education programs may prevent new infections from taking place by giving the public information about HIV - what HIV/AIDS are, how they are transmitted, and how people can avoid infection. The mass media is a very effective way to convey this information. Therefore, creation of public awareness about HIV prevention, treatment, care and support can potentially influence individual’s behavior.

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The transmission dynamic of HIV/AIDS has been of extreme interest to researchers, including both mathematicians and biologists. Mathematical models have been used extensively in helping improve our understanding of the major contributing factors to the pandemic. The classical mathematical model for infectious diseases is the compartmental model, first proposed by Kermack and McKendric in the year of 1927, in which, individuals are divided into multiple compartments dependent on their epidemiological status [10]. Since then, a large amount of epidemiological models have popped up to find out the mechanisms of HIV transmission and to determine the effective measures in preventing and controlling the spread of HIV/AIDS. de Arazoza and Lounes studied an epidemic model with contact tracing and fitted it with the data for the Cuban HIV/AIDS outbreak [2]. Naresh formulated an HIV model with varying population size and considered both horizontal and vertical transmission [17]. The optimal strategy for controlling HIV/AIDS was studied by Yusuf and Benyah targeting South Africa [30]. An HIV/AIDS model with different latent stages and treatment was developed and investigated [8]. Silva and Torres obtained the global results of an HIV/AIDS model with constant recruitment rate, mass action incidence, and varying population size [19].

Bilinear and standard incidence functions have been widely used in previous work [16, 24]. Several nonlinear incidence functions have been proposed corresponding to different scenarios. A saturated incidence $S_f(I)$ was introduced by Capasso and Serio to study the cholera in Bari in 1973 [1]. Another two types of nonlinear incidence function $\beta I^p S^q$ and $\beta I^p S/(1 + \alpha I^q)$ were proposed by Liu et al. [15], and ever since they have been employed in various epidemic models (see [5, 6, 18] and the references therein). The nonlinear incidence form of $\beta(I + \nu I^p)S$ was proposed by van den Driessche and Watmough [25]. An HIV/AIDS epidemic model with general nonlinear incidence rate and treatment was formulated and studied in [9]. A more general form of nonlinear incidence $f(S, I, N)$ was considered in [11], and many other forms of nonlinear incidence were proposed and discussed in [4, 13, 21, 24, 28, 29].

Motivated by previous work, we consider an HIV/AIDS epidemic model with two general nonlinear incidences and two susceptible classes: the cautious susceptible class and the incautious susceptible class. The paper is organized as follows. In section 2, the compartmental model is formulated; In section 3, preliminary results are summarized and the basic reproduction number is derived; In section 4, the global stability of the disease-free equilibrium is analyzed; In section 5, the existence and uniqueness of endemic equilibrium are identified, and its global stability is proved; In section 6, numerical simulations are performed. The paper ends up with a conclusion.

2. Model formulation

The total population is divided into five classes depending on epidemiological status of individuals: incautious susceptible $S_u(t)$, cautious susceptible $S_w(t)$, asymptomatic infective $E(t)$, symptomatic infective $I(t)$ before the onset of AIDS, and AIDS patients $A(t)$. The AIDS patients are usually hospitalized or sexually inactive, who are then assumed not to engage in HIV transmission activities, and do not contribute to HIV infection accordingly. The dynamic flow chart describing HIV infection between compartments is shown in Figure 1.

The following system of nonlinear ordinary differential equations is formulated from Figure 1.
Global results for an HIV/AIDS model

\[
\begin{align*}
\frac{dS_u(t)}{dt} &= \Lambda_u - S_u(t)g_u(E(t), I(t)) - dS_u(t), \\
\frac{dS_w(t)}{dt} &= \Lambda_w - S_w(t)g_w(E(t)) - dS_w(t), \\
\frac{dE(t)}{dt} &= S_u(t)g_u(E(t), I(t)) + S_w(t)g_w(E(t)) - \alpha E(t) - dE(t), \\
\frac{dI(t)}{dt} &= \alpha E(t) - \delta I(t) - dI(t), \\
\frac{dA(t)}{dt} &= \delta I(t) - rA(t) - dA(t),
\end{align*}
\]

where \( \Lambda_u \) and \( \Lambda_w \) are the recruitment rates of the susceptible classes \( S_u(t) \) and \( S_w(t) \), respectively; \( d \) is the natural death rate; \( \alpha \) is the transfer rate from asymptomatic stage to symptomatic stage of HIV infection; \( \delta \) is the transfer rate from symptomatic stage to the full-blown AIDS; \( r \) is the AIDS-related death rate; \( g_u(E(t), I(t)) \) is the general nonlinear force of infection toward incautious susceptible, which can be infected by both asymptomatic infective and symptomatic infective through sexual contact; \( g_w(E(t)) \) is the general nonlinear force of infection toward cautious susceptible, which only can be infected by asymptomatic infective. The initial conditions are listed below:

\[
S_u(0) > 0, S_w(0) > 0, E(0), I(0), A(0) \geq 0, E(0) + I(0) > 0. \tag{2.2}
\]

Motivated by previous research \([7, 9, 13, 20, 23, 24, 28]\), assume \( g_u(E, I) \) and \( g_w(E) \) satisfies the following properties:

(H1) \( g_u(E, I) \) is a real locally Lipschitz function in \([0, +\infty) \times [0, +\infty)\); \( g_w(E) \) is a real locally Lipschitz function on \([0, +\infty)\);
(H2) $g_u(0, 0) = 0$, $g_u(E, I) > 0$ for $E > 0$, $I > 0$; $g_w(0) = 0$, $g_w(E) > 0$ for $E > 0$;

(H3) $\frac{\partial g_u(E, I)}{\partial E} > 0$ and $\frac{\partial g_u(E, I)}{\partial I} > 0$ for $E$, $I \geq 0$; $g'_w(E) > 0$ for all $E \geq 0$;

(H4) $\frac{\partial^2 g_u(E, I)}{\partial E^2} \leq 0$, $\frac{\partial^2 g_u(E, I)}{\partial I^2} \leq 0$, and $\frac{\partial^2 g_u(E, I)}{\partial E \partial I} = 0$;

(H5) $g_u(E, I)/E$ is continuous and monotonously non-increasing with respect to $E$, for $E, I > 0$; Similarly, $g_u(E, I)/I$ is continuous and monotonously non-increasing with respect to $I$, for $E, I > 0$; $g_w(E)/E$ is continuous and monotonously non-increasing with respect to $E$, for $E > 0$.

3. Preliminary results

Let $N(t) = S_u(t) + S_w(t) + E(t) + I(t) + A(t)$. From system (2.1), we have the equation for total population $N(t)$:

$$\frac{dN(t)}{dt} = \Lambda_u + \Lambda_w - dN - rA,$$

yielding

$$\frac{dN(t)}{dt} \leq (\Lambda_u + \Lambda_w) - dN.$$

Therefore, the biologically feasible domain for system (2.1) is

$$\Omega = \{(S_u, S_w, E, I, A) \in \mathbb{R}_+^5 | 0 \leq S_u + S_w + E + I + A \leq (\Lambda_u + \Lambda_w)/d\}. \quad (3.1)$$

Note that system (2.1) always has a disease-free equilibrium $E_0 = (S^0_u, S^0_w, 0, 0, 0)$ with $S^0_u = \Lambda_u/d$ and $S^0_w = \Lambda_w/d$.

Follow the next generation method to calculate the basic reproduction number for deterministic compartmental models [3, 26]. Denote $x = (E, I, A, S_u, S_w)^T$. Using the same notations as [26], we rewrite system (2.1) as

$$\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x), \quad (3.2)$$

where

$$\mathcal{F}(x) = (S_u g_u(E, I) + S_w g_w(E), 0, 0, 0)^T, \quad (3.3)$$

and

$$\mathcal{V}(x) = \begin{pmatrix}
\alpha E + dE \\
(\delta + d)I - \alpha E \\
(\gamma + d)A - \delta I \\
S_u g_u(E, I) + dS_u - \Lambda_u \\
S_w g_w(E) + dS_w - \Lambda_w
\end{pmatrix}. \quad (3.4)$$
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Denote
\[
\partial_1 g_u(0,0) = \left. \frac{\partial g_u(E,I)}{\partial E} \right|_{E=0,I=0},
\partial_2 g_u(0,0) = \left. \frac{\partial g_u(E,I)}{\partial I} \right|_{E=0,I=0},
g'_w(0) = \left. \frac{dg_w(E)}{\partial E} \right|_{E=0}.
\]

Set \(E = I = A = 0\), and then \(x_0 = (0, 0, 0, S_u^0, S_w^0)^T\). Taking the Fréchet derivatives of \(F(x)\) and \(V(x)\) and evaluating them at \(x_0\), we find
\[
F = \begin{pmatrix}
S_u \partial_1 g_u(0,0) + S_w g'_w(0) & S_u \partial_2 g_u(0,0) \\
0 & 0 & 0 \\
0 & 0 & 0
\end{pmatrix},
\]
and
\[
V = \begin{pmatrix}
\alpha + d & 0 & 0 \\
-\alpha & \delta + d & 0 \\
0 & -d & \gamma + d
\end{pmatrix},
\]
where \(F\) is non-negative and \(V\) is non-singular. Additionally,
\[
FV^{-1} = \frac{1}{(\alpha + d)(\delta + d)(\gamma + d)} \begin{pmatrix}
M_1 & M_2 & 0 \\
0 & 0 & 0 \\
0 & 0 & 0
\end{pmatrix},
\]
where
\[
M_1 = (S_u \partial_1 g_u(0,0) + S_w g'_w(0))((\delta + d)(\gamma + d) + S_u \partial_2 g_u(0,0)\alpha(\gamma + d))
\]
and
\[
M_2 = S_u \partial_2 g_u(0,0)((\gamma + d)(\alpha + d)).
\]
The basic reproduction number of system (2.1) is given as follows.
\[
R_0 = \rho(FV^{-1}) = \frac{(S_u^0 \partial_1 g_u(0,0) + S_w^0 g'_w(0))((\delta + d) + S_u^0 \partial_2 g_u(0,0)\alpha)}{(\alpha + d)(\delta + d)}. \tag{3.5}
\]

4. Global stability of disease-free equilibrium

Considering the variable \(A(t)\) in (2.1c) decouples from the first four equations of (2.1), we target the following subsystem for the rest of paper:
\[
\frac{dS_u(t)}{dt} = \Lambda_u - S_u(t)g_u(E(t), I(t)) - dS_u(t), \tag{4.1a}
\frac{dS_w(t)}{dt} = \Lambda_w - S_w(t)g_w(E(t)) - dS_w(t) \tag{4.1b}
\]
\[
\frac{dE(t)}{dt} = S_u(t)g_u(E(t), I(t)) + S_w(t)g_w(E(t)) - \alpha E(t) - dE(t), \quad (4.1c)
\]
\[
\frac{dI(t)}{dt} = \alpha E(t) - \delta I(t) - dI(t). \quad (4.1d)
\]

System (4.1) always has a disease-free equilibrium \( E_0 = (S^0_u, S^0_w, 0, 0) \), where \( S^0_u = \Lambda_u/d \) and \( S^0_w = \Lambda_w/d \). The Jacobian matrix of system (4.1) at \( E_0 \) is
\[
J(E_0) = \begin{pmatrix}
-d & 0 & -S^0_u \partial_1 g_u(0, 0) & -S^0_u \partial_2 g_w(0, 0) \\
0 & -d & -S^0_w g'_w(0) & 0 \\
0 & 0 & S^0_u \partial_1 g_u(0, 0) + S^0_w g'_w(0) - \alpha - d & S^0_u \partial_2 g_w(0, 0) \\
0 & 0 & \alpha & -\delta - d
\end{pmatrix}.
\]

The characteristic equation of \( J(E_0) \) is
\[
\det(\lambda I - J(E_0)) = 0. \quad (4.2)
\]

Obviously, equation (4.2) has four roots: a negative double root \(-d\); and other two roots satisfy the following equation
\[
\lambda^2 + B_1 \lambda + B_2 = 0, \quad (4.3)
\]
where
\[
B_1 = -[S^0_u \partial_1 g_u(0, 0) + S^0_w g'_w(0) - \alpha - d - \delta - d],
\]
and
\[
B_2 = -[S^0_u \partial_1 g_u(0, 0) + S^0_w g'_w(0) - \alpha - d - \alpha S^0_u \partial_1 g_u(0, 0)]
= (\delta + d)(\alpha + d)(1 - R_0).
\]

When \( R_0 < 1 \), we have
\[
S^0_u \partial_1 g_u(0, 0) + S^0_w g'_w(0) < \alpha + d.
\]

It infers that \( B_1 > 0 \) and \( B_2 > 0 \), which means that all the eigenvalues of matrix \( J(E_0) \) have negative real parts. Therefore, the disease-free equilibrium \( E_0 \) is locally asymptotically stable.

Next, we prove the global stability of \( E_0 \). It is obvious from Eqs. (4.1a) and (4.1b) that
\[
S_u(t) \leq S^0_u, \quad S_w(t) \leq S^0_w.
\]

Let
\[
h_1(E, I) = g_u(E, I) - (\partial_1 g_u(0, 0)E + \partial_2 g_u(0, 0)I),
\]
and
\[
h_2(E) = g_w(E) - g'_w(0)E.
\]

For \( E, I > 0 \), from assumptions (H1)~(H5), we have
\[
\frac{\partial h_1(E, I)}{\partial E} = \frac{\partial g_u(E, I)}{\partial E} - \partial_1 g_u(0, 0) < 0,
\]
\[
\frac{\partial h_1(E, I)}{\partial I} = \frac{\partial g_u(E, I)}{\partial I} - \partial_2 g_u(0, 0) < 0, \quad (4.4)
\]
and
\[ \begin{align*}
\frac{\partial^2 h_1(E, I)}{\partial E^2} &= \frac{\partial^2 g_u(E, I)}{\partial E^2} < 0, \\
\frac{\partial^2 h_1(E, I)}{\partial I^2} &= \frac{\partial^2 g_u(E, I)}{\partial I^2} < 0, \\
\frac{\partial^2 h_1(E, I)}{\partial E \partial I} &= \frac{\partial^2 g_u(E, I)}{\partial E \partial I} = 0.
\end{align*} \tag{4.5} \]
Together with \( h_1(0, 0) = 0 \), we have \( h_1(E, I) < 0 \) for all \( E, I > 0 \).

Similarly, for \( E > 0 \), we have
\[ \begin{align*}
\frac{\partial h_2(E)}{\partial E} &= \frac{\partial g_w(E)}{\partial E} - g_w'(0) < 0, \\
\frac{\partial^2 h_2(E)}{\partial E^2} &= \frac{\partial^2 g_w(E)}{\partial E^2} < 0. \tag{4.6}
\end{align*} \]
Together with \( h_2(0) = 0 \), we have \( h_2(E) < 0 \) for all \( E > 0 \).

Therefore, we have
\[ \begin{align*}
\frac{dE(t)}{dt} &\leq S^0_u \left[ \partial_1 g_u(0, 0) E + \partial_2 g_u(0, 0) I \right] + S^0_w g_w'(0) E - \alpha E - dE, \\
\frac{dI(t)}{dt} &\leq \alpha E - \delta I - dI. \tag{4.7}
\end{align*} \]

The following auxiliary system is then constructed:
\[ \begin{align*}
\frac{dE(t)}{dt} &= [S^0_u \partial_1 g_u(0, 0) + S^0_w g_w'(0) - \alpha - d] E + S^0_u \partial_2 g_u(0, 0) I, \\
\frac{dI(t)}{dt} &= \alpha E - (\delta + d) I. \tag{4.8}
\end{align*} \]
The coefficient matrix of system (4.8) is:
\[ M_C = \begin{pmatrix} S^0_u \partial_1 g_u(0, 0) + S^0_w g_w'(0) - \alpha - d & S^0_u \partial_2 g_u(0, 0) \\ \alpha & -\delta - d \end{pmatrix}. \tag{4.9} \]
Discussions above indicate that all the eigenvalues of matrix \( M_C \) have negative real parts. Therefore,
\[ \lim_{t \to +\infty} E(t) = 0, \quad \lim_{t \to +\infty} I(t) = 0, \tag{4.10} \]
and
\[ \lim_{t \to +\infty} S_u(t) = S^0_u, \quad \lim_{t \to +\infty} S_w(t) = S^0_w. \tag{4.11} \]

We then have the following results.

**Theorem 4.1.** The disease-free equilibrium \( E_0 \) of system (4.1) is globally asymptotically stable when \( R_0 \leq 1 \), and unstable when \( R_0 > 1 \).
5. Existence and global stability of endemic equilibrium

The existence and uniqueness of an endemic equilibrium is investigated in this section. Let the right hand side of system (4.1) be zero.

\[ \Lambda_u - S_u g_u(E, I) - dS_u = 0, \]
\[ \Lambda_w - S_w g_w(E) - dS_w = 0, \]
\[ S_u g_u(E, I) + S_w g_w(E) - \alpha E - dE = 0, \]
\[ \alpha E - \delta I - dI = 0. \]

From (5.1), (5.2) and (5.4), we have

\[ S_u = \frac{\Lambda_u}{g_u(E, I) + d}, \quad S_w = \frac{\Lambda_w}{g_w(E) + d}, \quad E = \frac{(\delta + d)I}{\alpha}. \]

Substituting (5.5) into (5.3) gives

\[ \frac{\Lambda_u g_u(E, I)}{g_u(E, I) + d} + \frac{\Lambda_w g_w(E)}{g_w(E) + d} = \frac{(\alpha + d)(\delta + d)I}{\alpha}. \]

Rewrite (5.6) as the following equation

\[ \frac{1}{d} \left( \Lambda_u + \Lambda_w - \frac{\alpha + d}{\alpha} (\delta + d) I \right) = \frac{\Lambda_u}{g_u(E, I) + d} + \frac{\Lambda_w}{g_w(E) + d}. \]

Let

\[ f_1(I) = \frac{1}{d} \left( \Lambda_u + \Lambda_w - \frac{\alpha + d}{\alpha} (\delta + d) I \right), \]
\[ f_2(E, I) = \frac{\Lambda_u}{g_u(E, I) + d} + \frac{\Lambda_w}{g_w(E) + d}. \]

Then (5.7) presents

\[ f_1(I) = f_2(E, I). \]

Moreover,

\[ f_1(0) = \frac{1}{d} (\Lambda_u + \Lambda_w), \]
\[ f_2(E, 0) = \frac{\Lambda_u}{g_u(E, 0) + d} + \frac{\Lambda_w}{g_w(E) + d}. \]

If \( E > 0 \), we have \( g_u(E, 0) > 0 \), namely, \( f_2(E, 0) < f_1(0) \); If \( E = 0 \), we have \( g_w(0) = 0 \), namely, \( f_2(E, 0) = f_1(0) \). When \( f_1(I) = 0 \), we have

\[ \hat{I} = \frac{(\Lambda_u + \Lambda_w)\alpha}{(\alpha + d)(\delta + d)}, \]

and \( f_2(E, \hat{I}) > 0 \). Then \( f_2(E, \hat{I}) > f_1(\hat{I}) \). Next, the derivatives of \( f_1 \) and \( f_2 \) with respect to \( I \) are calculated.

\[ \left. \frac{\partial f_2(E, I)}{\partial I} \right|_{I=0} = -\frac{(\Lambda_u \partial_1 g_u(E, 0) + \Lambda_u g_u'(E))(\delta + d) + \Lambda_u \partial_2 g_u(E, 0) \alpha}{\alpha d^2} < 0, \]
\[ \left. \frac{\partial f_1(I)}{\partial I} \right|_{I=0} = -\frac{(\alpha + d)(\delta + d)}{\alpha d} < 0. \]
Then at $E = 0$, we have
\[
\left. \frac{\partial f_2(E, I)}{\partial I} \right|_{I=0} - \left. \frac{\partial f_1(I)}{\partial I} \right|_{I=0} = \frac{(\alpha + d)(\delta + d)}{ad} (1 - R_0). \tag{5.15}
\]
When $R_0 \leq 1$,
\[
\left. \frac{\partial f_2(E, I)}{\partial I} \right|_{I=0} - \left. \frac{\partial f_1(I)}{\partial I} \right|_{I=0} \geq 0; \tag{5.16}
\]
when $R_0 > 1$,
\[
\left. \frac{\partial f_2(E, I)}{\partial I} \right|_{I=0} - \left. \frac{\partial f_1(I)}{\partial I} \right|_{I=0} < 0. \tag{5.17}
\]

![Sketch map of $y = f_1(I)$ and $y = f_2(E, I)$ for $E \geq 0$.](image)

Fig. 2 indicates that system (4.1) has a unique endemic equilibrium $E_* = (S^*_u, S^*_w, E^*, I^*)$ when $R_0 > 1$. The components of $E_*$ satisfy
\[
\begin{align*}
\Lambda_u - S^*_u g^*_u - dS^*_u &= 0, \\
\Lambda_w - S^*_w g^*_w - dS^*_w &= 0, \\
S^*_u g^*_u + S^*_w g^*_w - \alpha E^* - dE^* &= 0, \\
\alpha E^* - \delta I^* - dI^* &= 0,
\end{align*} \tag{5.18}
\]
where $g^*_u = g_u(E^*, I^*)$ and $g^*_w = g_w(E^*)$.

Next, the second Lyapunov criterion is employed to prove the global stability of the endemic equilibrium $E_*$. Define
\[
V(S_u, S_w, E, I) = \left( S_u - S^*_u - S^*_u \ln \left( \frac{S_u}{S^*_u} \right) \right) + \left( S_w - S^*_w - S^*_w \ln \left( \frac{S_w}{S^*_w} \right) \right) + \frac{S^*_u g^*_u}{(\delta + d)I^*} \left( I - I^* - I^* \ln \left( \frac{I}{I^*} \right) \right) + \left( E - E^* - E^* \ln \left( \frac{E}{E^*} \right) \right). \tag{5.19}
\]
It is easy to verify that $V \geq 0$, where $V = 0$ if and only if $(S_u, S_w, E, I) = (S^*_u, S^*_w, E^*, I^*)$. 
Differentiating $V(S_u, S_w, E, I)$ along the solution of system (4.1) yields

$$\frac{dV}{dt}(S_u, S_w, E, I)\bigg|_{(4.1)} = P_1 + P_2 + P_3 + P_4,$$

where $P_i (i = 1, \cdots, 4)$ are calculated as follows.

$$P_1 = \left(1 - \frac{S_u^*}{S_u}\right) S'_u = \left(1 - \frac{S_u^*}{S_u}\right) (\Lambda_u - S_u g_u - dS_u)$$

$$= \left(1 - \frac{S_u^*}{S_u}\right) (S_u^* g_u + S_u g_u^* - S_u g_u - dS_u)$$

$$= dS_u^* \left(2 - \frac{S_u^*}{S_u} - \frac{S_u}{S_u^*} + S_u^* g_u^* \left(1 - \frac{S_u}{S_u^*} + \frac{S_u g_u}{S_u^* g_u^*} + \frac{g_u}{S_u^*}ight),

P_2 = \left(1 - \frac{S_w^*}{S_w}\right) S'_w = \left(1 - \frac{S_w^*}{S_w}\right) \Lambda_w - S_w g_w - dS_w$$

$$= \left(1 - \frac{S_w^*}{S_w}\right) (S_w^* g_w^* + dS_w^* - S_w g_w - dS_w)$$

$$= dS_w^* \left(2 - \frac{S_w^*}{S_w} - \frac{S_w}{S_w^*} + S_u^* g_w^* \left(1 - \frac{S_w}{S_w^*} - \frac{S_w g_w}{S_w^* g_w^*}+ \frac{g_w}{S_w^*}ight),

P_3 = \left(1 - \frac{E^*}{E}\right) E' = \left(1 - \frac{E^*}{E}\right) \left(S_u g_u + S_w g_w - \alpha E - dE\right)$$

$$= \left(1 - \frac{E^*}{E}\right) \left(S_u g_u + S_w g_w - \frac{(S_u^* g_u^* + S_w^* g_w^*) E}{E^*}\right)$$

$$= S_u^* g_u^* \left(1 - \frac{E}{E^*} - \frac{S_u g_u}{S_u^* g_u^*} + \frac{S_u g_u}{S_u^* g_u^*}\right) + S_w^* g_w^* \left(1 - \frac{E}{E^*} - \frac{S_w g_w}{S_w^* g_w^*} + \frac{S_w g_w}{S_w^* g_w^*}\right),$$

and

$$P_4 = \frac{S_u^* g_u^*}{(\delta + d) I} \left(1 - \frac{I^*}{I}\right) I' = \frac{S_u^* g_u^*}{(\delta + d) I} \left(1 - \frac{I^*}{I}\right) (\alpha E - \delta I - dI)$$

$$= \frac{S_u^* g_u^*}{(\delta + d) I} \left(1 - \frac{I^*}{I}\right) \left(\frac{\delta + d}{I} - (\delta + d) I\right)$$

$$= S_u^* g_u^* \left(\frac{E}{E^*} - \frac{E^* I}{E^* I} - \frac{I}{I^*} + 1\right).$$
We then have

\[ P_1 + P_2 + P_3 + P_4 = dS_u^* \left( 2 - \frac{S_u^*}{S_u^* - S_u} \right) + dS_w^* \left( 2 - \frac{S_w^*}{S_w^* - S_w} \right) \]

\[ + S_u^* g_u \left( 3 - \frac{S_u^*}{S_u^* - S_u} - I^* - \frac{S_u g_u E^*}{S_u^* g_u E^*} + \frac{g_u}{g_u^*} \right) \]

\[ + S_w^* g_w \left( 2 - \frac{S_w^*}{S_w^* - E^*} - \frac{S_w g_w E^*}{S_w^* g_w E^*} + \frac{g_w}{g_w^*} \right). \]

Since

\[ \frac{g_u}{g_u^*} - \frac{I}{I^*} = \left( \frac{g_u}{g_u^*} - 1 \right) \left( 1 - \frac{g_u I^*}{g_u^* I} \right) + 1 - \frac{g_u I^*}{g_u^* I}, \]

and

\[ \frac{g_w}{g_w^*} - \frac{E}{E^*} = \left( \frac{g_w}{g_w^*} - 1 \right) \left( 1 - \frac{g_w E^*}{g_w^* E} \right) + 1 - \frac{g_w E^*}{g_w^* E}, \]

then

\[ P_1 + P_2 + P_3 + P_4 = dS_u^* \left( 2 - \frac{S_u^*}{S_u^* - S_u} \right) + dS_w^* \left( 2 - \frac{S_w^*}{S_w^* - S_w} \right) \]

\[ + S_u^* g_u \left( 4 - \frac{S_u^*}{S_u^* - S_u} - \frac{S_u g_u E^*}{S_u^* g_u E^*} - \frac{g_u E^*}{g_u^* E^*} \right) \]

\[ + S_w^* g_w \left( 3 - \frac{S_w^*}{S_w^* - S_w} - \frac{S_w g_w E^*}{S_w^* g_w E^*} - \frac{g_w E^*}{g_w^* E^*} \right) \]

\[ + S_u^* g_u \left( \frac{g_u}{g_u^*} - 1 \right) \left( 1 - \frac{g_u I^*}{g_u^* I} \right) \]

\[ + S_w^* g_w \left( \frac{g_w}{g_w^*} - 1 \right) \left( 1 - \frac{g_w E^*}{g_w^* E} \right). \]

Therefore,

\[ P_1 + P_2 + P_3 + P_4 \leq S_u^* g_u \left( \frac{g_u}{g_u^*} - 1 \right) \left( 1 - \frac{g_u I^*}{g_u^* I} \right) + S_w^* g_w \left( \frac{g_w}{g_w^*} - 1 \right) \left( 1 - \frac{g_w E^*}{g_w^* E} \right). \]

Using the monotonous conditions in (H1)~(H5), we have

\[ \left( \frac{g_u}{g_u^*} - 1 \right) \left( 1 - \frac{g_u I^*}{g_u^* I} \right) = \frac{I}{g_u g_u} \left( g_u - g_u^* \right) \left( \frac{g_u}{I} - \frac{g_u^*}{I^*} \right) \leq 0 \]

and

\[ \left( \frac{g_w}{g_w^*} - 1 \right) \left( 1 - \frac{g_w E^*}{g_w^* E^*} \right) = \frac{E}{g_w g_w} \left( g_w - g_w^* \right) \left( \frac{g_w}{E} - \frac{g_w^*}{E^*} \right) \leq 0. \]

Then

\[ \frac{dV(t)}{dt} = P_1 + P_2 + P_3 + P_4 \leq 0. \]

And \( dV(t)/dt = 0 \) if and only if \( (S_u, S_w, E, I) = (S_u^*, S_w^*, E^*, I^*) \). According to the second Lyapunov criterion [14], the endemic equilibrium of system (4.1) is globally asymptotically stable. The results are summarized in following theorem.

**Theorem 5.1.** System (4.1) has a unique endemic equilibrium \( E^* \), and it is globally asymptotically stable when \( R_0 > 1 \).
6. Numerical considerations

The following specific forms of \( g_u(E, I) \) and \( g_w(E) \) are chosen to illustrate the obtained theoretical results for model (2.1):

\[
g_u(E, I) = \frac{\beta_1 E}{1 + \alpha_1 E} + \frac{\beta_2 I}{1 + \alpha_2 I}, \quad g_w(E) = \frac{\beta_3 E}{1 + \alpha_3 E},
\]

which satisfy conditions (H1)∼(H5).

The parameter values are assigned as follows: \( \Lambda_u = 7, \Lambda_w = 5, d = 0.0196, \delta = 0.4, \alpha = 0.2, r = 0.15, \alpha_1 = 0.02, \alpha_2 = 0.03, \alpha_3 = 0.01 \). The initial condition \((S_u(0), S_w(0), E(0), I(0), A(0)) = (200; 150; 80; 40; 20)\). Fig. 3(a) presents the global asymptotic stability of the disease-free equilibrium when \( R_0 < 1 \) with \( \beta_1 = 0.0003, \beta_2 = 0.0002, \beta_3 = 0.00025 \); Fig. 3(b) presents the global asymptotic stability of the endemic equilibrium when \( R_0 > 1 \) with \( \beta_1 = 0.003, \beta_2 = 0.002, \beta_3 = 0.004 \).

Next, the effects of incorporating two susceptible classes and two incidence functions on disease transmission are investigated by considering the basic reproduction number \( R_0 \). If we do not distinguish classes \( S_u \) and \( S_w \), i.e., both of them getting infected through the same infection force \( g_u(E, I) \), (2.1) turns into the following system:

\[
\begin{align*}
\frac{dS_u(t)}{dt} &= \Lambda_u - S_u g_u(E, I) - dS_u, \quad (6.2a) \\
\frac{dS_w(t)}{dt} &= \Lambda_w - S_w g_u(E, I) - dS_w, \quad (6.2b) \\
\frac{dE(t)}{dt} &= S_u g_u(E, I) + S_w g_u(E, I) - \alpha E - dE, \quad (6.2c) \\
\frac{dI(t)}{dt} &= \alpha E - \delta I - dI, \quad (6.2d) \\
\frac{dA(t)}{dt} &= \delta I - r A - dA. \quad (6.2e)
\end{align*}
\]
Denote $S = S_u + S_w$ and $\Lambda = \Lambda_u + \Lambda_w$. By adding (6.2a) and (6.2b), we have
\[
\frac{dS(t)}{dt} = \Lambda - Sg_u(E, I) - dS, \\
\frac{dE(t)}{dt} = Sg_u(E, I) - \alpha E - dE.
\] (6.3)

System (6.3) is the classic model equations for $S(t)$ and $E(t)$.

The basic reproduction number for model (6.2) is calculated following the same method in section 3:

\[
\tilde{R}_0 = \frac{(S_u^0\partial_1 g_u(0, 0) + S_w^0\partial_1 g_u(0, 0))(\delta + d) + (S_u^0\partial_2 g_u(0, 0) + S_w^0\partial_2 g_u(0, 0))\alpha}{(\alpha + d)(\delta + d)}.
\]

It is checked that
\[R_0 \leq \tilde{R}_0.\] (6.4)

This implies that system (2.1) has a smaller basic reproduction number because of the introduction of cautious susceptible $S_w$, which does not interact with symptomatic infective. Moreover, Fig. 4 shows the consistent result that the cautious individuals $S_w(t)$ and the resulting infection force $g_w(E)$ have positive impact on HIV transmission.

**Figure 4.** Effect of the introduction of cautious class and multiple transmission functions on HIV/AIDS prevalence when $R_0 > 1$.

### 7. Conclusion

In this paper, an HIV/AIDS epidemic model is proposed, incorporating the cautious and incautious susceptible classes. The incautious susceptible can be infected by both the asymptomatic and the symptomatic infectives. However, the cautious susceptible can only get infected by asymptomatic infectives. This results in two types of general nonlinear incidence function.

The basic reproduction number $R_0$ is derived by the next generation method, which is crucial to the global dynamics of the model system. The system only has a globally asymptotically stable disease-free equilibrium when $R_0 \leq 1$, and it implies that the disease eventually dies out; the system has a unique globally asymptotically
stable endemic equilibrium when $R_0 > 1$, and it means that the disease becomes endemic in the long run.

The introduction of cautious susceptible may reduce the basic reproduction number to some extent through avoiding excessive contact with symptomatic infectives (presence of two nonlinear infection force functions). This in turn mitigates the HIV epidemic and give hints for the role that the mass media may play in disease prevention and control by raising public awareness.

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