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Multi-step Adomian decomposition method for solving a delayed Chikungunya virus system

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Abstract

This paper is an extension of work [M. Chamekh, M. A. Latrach, F. Jday, J. Umm Al-Qura Univ. Appll. Sci., **9** (2023), 123–131] in which we proposed a multi-step semi-analytical solution for a chikungunya virus system without delay. Accordingly, this work targets the same problem but with delay. We first used the Lyapunov function method to present the theoretical stability analysis of the Chikungunya virus model with latency and with delay. Afterwards, we discussed the use of the Adomian decomposition method with a technique of multi-step to obtain a solution to a delayed Chikungunya problem in particular, and we aimed in general at some problems of dynamical systems with delay. To validate the accuracy and efficiency of this method, the numerical results were discussed by comparing them to the Runge-Kutta method.

Keywords: Multi-step method, Adomian decomposition method, Chikungunya virus, delay differential equations.

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1. Introduction

Delay differential equations (DDE) present an essential model for many applications in economics, biology, chemical kinetics, engineering sciences, and in the study of the evolution of infectious disease transmission in the field of medical sciences [1, 3, 15, 18, 22]. As the analytical treatments which use mathematical tools to solve these DDE can be difficult, the numerical analysis holds a capital place in the mathematical treatment, as well as in the physical sciences as in the field of biology. It offers very rich possibilities for scientific research. Moreover, recent developments in scientific computing means have opened up new perspectives. Although the proposed numerical methods are considered effective and give good estimates, the numerical tools require certain programming skills. For this, many researchers have developed so-called semi-analytical methods which have been frequently used in recent years to solve PDEs such as the variational iteration method, the differential transformation method, the perturbation method of homotopy and the Adomian decomposition method (ADM) [6, 13, 16, 19, 24, 29]. As example in [4], we find an interesting method based on ADM to follow nonlinear solutions with historical

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functions. This method does not require much theoretical effort. Although this method showed its good accuracy, the resolution domain was a bit restricted. The initial goal of researchers of these methods is to provide the simplicity and the practicability in solving some difficult differential problems with the minimum of mathematical tools, and the power to find an approximate explicit analytical solution has been decisive. Most of the solutions they propose are restricted to specific domains (generally not exceeding the interval $[0, 1]$), which do not allow to conclude, for example, the shape of the solution for a phenomenon epidemiological for a wider scope of time. To overcome these drawbacks, many researchers (as example[2]) have proposed some modifications to these methods to increase their elasticity and their precision as well as to broaden the field of study.

In general, the mathematical models studied in biology which intervene in many important physical phenomena can lead to dynamic systems. Moreover, the delay of dynamical systems that have appeared in many biological problems has led to the emergence of a powerful method of analysis recently used by biologists. By way of example, we can cite the study of the Chikungunya virus (CHIKV) by taking into account the intracellular delays distributed [8, 12] and the study of the nutrient-plankton interaction by taking into account the time necessary for phytoplankton to mature after the release of toxins [21]. Motivated by these phenomena of epidemiology, we propose to study the spread of CHIKV in humans taking into account their past state. To achieve this goal, we start with a stability analysis, where we used Lyapunov's function method as the first step. In the next step, we mainly propose a numerical processing based on ADM equipped with a multi-step strategy (MADM) to find a semi-analytical solution for the CHIKV model with latency and with delay. This method was used in [7] to solve systems of differential equations but without adaptive multi-step. The method is very useful and reliable for certain systems of ordinary differential equations. We, therefore, propose to show that this multi-step adaptive method can be applied to more complex dynamic systems and in particular to the CHIKV model with latency and with delay. Finally, we propose practical numerical examples that can be used to discuss the performance of MADM by comparing its results with those of the Runge-Kutta method.

2. CHIKV model with latency and with delay

Chikungunya virus is a viral disease transmitted between people through a person-to-mosquito-to-person cycle, the mosquitoes that transmit this disease are of the genus Aedes or yellow fever. In scientific literature, several mathematical models have been presented that describe the evolution of disease transmission in a population or that describe the population growth of Chikungunya virus within-host at each stage. Sometimes models take into account the time delay factor to avoid the delays decision-making on how to intervene, or it may be that knowledge of the actual evolution of the disease requires the consideration of a historic part of the condition of the infected. Given this, and since it is necessary to consider a discrete delay regarding the emergence of symptoms in asymptomatic or pauci-symptomatic patients, we study the following CHIKV dynamic model with two discrete-time delays and with CHIKV-to-cell transmission [8]:

$$\begin{cases} \dot{S}(t) = \mu - aS(t) - \alpha S(t)V(t), \\ \dot{L}(t) = (1-\rho)e^{-\delta_1\tau_1}\alpha S(t-\tau_1)V(t-\tau_1) - (h+\nu)L(t), \\ \dot{I}(t) = \rho e^{-\delta_2\tau_2}\alpha S(t-\tau_2)V(t-\tau_2) + \nu L(t) - pI(t), \\ \dot{V}(t) = mI(t) - rV(t) - qB(t)V(t), \\ \dot{B}(t) = \eta + cB(t)V(t) - dB(t), \end{cases} \tag{2.1}$$

where S, L, I, V, and B are positive represent the concentrations of uninfected monocytes, latently infected monocytes, actively infected monocytes, CHIKV particles, and concentrations of B cells, respectively. Since it is necessary, as indicated in the aim of the study, to consider the delays regarding the emergence of symptoms in asymptomatic or pauci-symptomatic patients, the time delay τ_1 denotes the time between contact of CHIKV particles with uninfected host cells and latent infection, and $e^{-\delta_1\tau_1}$ is the probability of latently infected host cells surviving to the age of τ_1, where δ_1 is constant. Time delay τ_2 denotes the time

between infection of host cells and the production of active CHIKV particles, and $e^{-\delta_2\tau_2}$ is the probability of actively infected host cells surviving to the age of τ_2, where δ_2 is constant. The rest of the parameters appearing in CHIKV dynamics model (2.1) are interpreted as follows [8]:

- μ is the creation rate of uninfected susceptible host cells who die at rate αS.
- αSV is the attack rate of uninfected susceptible host cells by CHIKV particles, where b is the contact rate between susceptible host cells and free CHIKV particles.
- ν is the transmission rate from a latent to active infected host cell.
- $0 < \rho < 1$ is the part of infected cells assumed to be actively infected cells and $1-\rho$ is a part assumed to be latently infected cells.
- m is the reproduction rate of CHIKV particles.
- qBV is the rate at which CHIKV particles are attacked by B cells.
- η and cBV are the rates of creation and proliferation of B cells, respectively.
- hL, pI, rV, and dB are, respectively, the death rates of latently infected host cells, actively infected host cells, free CHIKV particles, and B cells.

In the next, we consider the initial conditions

$$\begin{aligned} &S(\phi)=\vartheta_1(\phi), \quad L(\phi)=\vartheta_2(\phi), \quad I(\phi)=\vartheta_3(\phi), \qquad V(\phi)=\vartheta_4(\phi), \quad B(\phi)=\vartheta_5(\phi) \\ &\vartheta_i(\phi)\geqslant 0, \qquad \phi\in[-k,0], \qquad \vartheta_i\in C([-k,0],\mathbb{R}_+), \qquad i=1,2,3,4,5, \end{aligned} \tag{2.2}$$

where $k=\max\{\tau_1,\tau_2\}$ and C is the Banach space of continuous functions mapping the interval $[-k,0]$ into $\mathbb{R}_+$.

2.1. Basic properties

Lemma 2.1. *The solutions of system (2.1) with the initial condition (2.2) are non-negative and ultimately bounded.*

Proof. We have

$$\begin{aligned} &\dot{S}\,|_{S=0}=\mu>0,\\ &\dot{V}\,|_{V=0}=mI\geqslant 0, \forall\, I\geqslant 0,\\ &\dot{B}\,|_{B=0}=\eta>0,\\ &L(t)=\vartheta_2(0)e^{-(h+\nu)t}+(1-\rho)\alpha e^{-\delta_1\tau_1}\int_0^t e^{-(h+\nu)(t-\theta)}S(\theta-\tau_1)V(\theta-\tau_1)d\theta\geqslant 0,\\ &I(t)=\vartheta_3(0)e^{-pt}+\rho\alpha e^{-\delta_2\tau_2}\int_0^t e^{-p(t-\theta)}S(\theta-\tau_2)V(\theta-\tau_2)d\theta+\nu\int_0^t e^{-p(t-\theta)}L(\theta)d\theta\geqslant 0, \text{ for } t\in[0,k]. \end{aligned}$$

Then, $\mathbb{R}^5_+$ is positively invariant for system (2.1). We let $H_1^d(t)=(1-\rho)e^{-\delta_1\tau_1}S(t-\tau_1)+\rho e^{-\delta_2\tau_2}S(t-\tau_2)+L(t)+I(t)$ and $H_2^d(t)=V(t)+\frac{q}{c}B(t)$, then

$$\begin{aligned} \dot{H}_1^d(t)&=(1-\rho)e^{-\delta_1\tau_1}(\mu-aS(t-\tau_1)-\alpha S(t-\tau_1)V(t-\tau_1))\\ &\quad+\rho e^{-\delta_2\tau_2}(\mu-aS(t-\tau_2)-\alpha S(t-\tau_2)V(t-\tau_2))\\ &\quad+(1-\rho)e^{-\delta_1\tau_1}\alpha S(t-\tau_1)V(t-\tau_1)-(h+\nu)L(t)+\rho e^{-\delta_2\tau_2}\alpha S(t-\tau_2)V(t-\tau_2)+\nu L(t)-pI(t)\\ &=(1-\rho)e^{-\delta_1\tau_1}(\mu-aS(t-\tau_1))+\rho e^{-\delta_2\tau_2}(\mu-aS(t-\tau_2))-hL(t)-pI(t)\\ &\leqslant \mu-\sigma_1((1-\rho)e^{-\delta_1\tau_1}S(t-\tau_1)+\rho e^{-\delta_2\tau_2}S(t-\tau_2)+L(t)+I(t))=\mu-\sigma_1H_1^d(t), \end{aligned}$$

where, $\sigma_1=\min\{a,h,p\}$. Moreover, we have

$$\dot{H}_2^d(t)=mI(t)-rV(t)+\frac{q}{c}\eta-\frac{qd}{c}B(t)\leqslant mM_1+\frac{q}{c}\eta-\sigma_2\left(V(t)+\frac{q}{c}B(t)\right)=mM_1+\frac{q}{c}\eta-\sigma_2H_2^d(t),$$

where, $\sigma_2 = \min\{r, d\}$. Thus, $S(t)$, $L(t)$, $I(t)$, $V(t)$, and $B(t)$ are ultimately bounded. □

2.2. Steady states

Let $\gamma = \dfrac{(1-\rho)\nu e^{-\delta_1\tau_1} + \rho(h+\nu)e^{-\delta_2\tau_2}}{(h+\nu)}$, $S_0 = \frac{\mu}{a}$, and $B_0 = \frac{\eta}{d}$, then the basic reproduction number is given by

$$\mathscr{R}_0^d = \frac{\gamma m\alpha\mu d}{ap(rd+q\eta)} = \frac{\gamma m\alpha S_0}{p(r+qB_0)}.$$

Note that for $\tau_1 = \tau_2 = 0$, we obtain $\mathscr{R}_0^d = \mathscr{R}_0$ which is defined in [5] for the for chikungunya virus system without delay.

Lemma 2.2. *If $\mathscr{R}_0^d \leqslant 1$, then system (2.1) has only one disease-free equilibrium E_0^d, and if $\mathscr{R}_0^d > 1$, then the system has two equilibria: a disease-free equilibrium E_0^d and an endemic equilibrium E_1^d.*

Proof. Let (S, L, I, V, B) be any steady state satisfying

$$\begin{aligned}
0 &= \mu - aS - \alpha SV,\\
0 &= (1-\rho)e^{-\delta_1\tau_1}\alpha SV - (h+\nu)L,\\
0 &= \rho e^{-\delta_2\tau_2}\alpha SV + \nu L - pI,\\
0 &= mI - rV - qBV,\\
0 &= \eta + cBV - dB.
\end{aligned} \tag{2.3}$$

Solving equations (2.3) there exists a CHIKV-free steady state $E_0^d = (S_0, 0, 0, 0, B_0)$. Now, let $E_1^d = (S, L, I, V, B)$ be any steady state, then from equations (2.3) we have

$$\begin{aligned}
0 &= \mu - \frac{p(h+\nu)(a+\alpha V)}{\alpha\big(\rho(h+\nu)e^{-\delta_2\tau_2} + (1-\rho)\nu e^{-\delta_1\tau_1}\big)}\left(\frac{r}{m} + \frac{q\eta}{m(d-cV)}\right),\\
S &= \frac{(h+\nu)L}{(1-\rho)\alpha V e^{-\delta_1\tau_1}} = \frac{p(h+\nu)}{\alpha\big(\rho(h+\nu)e^{-\delta_2\tau_2} + (1-\rho)\nu e^{-\delta_1\tau_1}\big)}\left(\frac{r}{m} + \frac{q\eta}{m(d-cV)}\right),\\
L &= \frac{(1-\rho)pe^{-\delta_1\tau_1}}{\rho(h+\nu)e^{-\delta_2\tau_2} + (1-\rho)\nu e^{-\delta_1\tau_1}} I = \frac{(1-\rho)pe^{-\delta_1\tau_1}}{\rho(h+\nu)e^{-\delta_2\tau_2} + (1-\rho)\nu e^{-\delta_1\tau_1}}\left(\frac{rV}{m} + \frac{q\eta V}{m(d-cV)}\right),\\
I &= \frac{V(r+qB)}{m} = \frac{rV}{m} + \frac{q\eta V}{m(d-cV)},\\
B &= \frac{\eta}{d-cV}.
\end{aligned} \tag{2.4}$$

Define the function

$$g_d(V) = \mu - \frac{p(h+\nu)(a+\alpha V)}{\alpha\big(\rho(h+\nu)e^{-\delta_2\tau_2} + (1-\rho)\nu e^{-\delta_1\tau_1}\big)}\left(\frac{r}{m} + \frac{q\eta}{m(d-cV)}\right).$$

Then we obtain

$$\begin{aligned}
\lim_{V\to 0^+} g_d(V) &= \lim_{V\to 0^+}\left(\mu - \frac{p(h+\nu)(a+\alpha V)}{\alpha\big(\rho(h+\nu)e^{-\delta_2\tau_2} + (1-\rho)\nu e^{-\delta_1\tau_1}\big)}\left(\frac{r}{m} + \frac{q\eta}{m(d-cV)}\right)\right)\\
&= \mu - \frac{pa(h+\nu)}{\alpha\big(\rho(h+\nu)e^{-\delta_2\tau_2} + (1-\rho)\nu e^{-\delta_1\tau_1}\big)}\left(\frac{r}{m} + \frac{q\eta}{md}\right).
\end{aligned}$$

Since $a = \frac{\mu}{S_0}$, $B_0 = \frac{\eta}{d}$, and $\mathscr{R}_0^d > 1$, we obtain

$$\lim_{V\to 0^+} g_d(V) = \mu - \frac{pa(h+\nu)\,(r+qB_0)}{m\alpha\big(\rho(h+\nu)e^{-\delta_2\tau_2}+(1-\rho)\nu e^{-\delta_1\tau_1}\big)} = \mu\left(1-\frac{1}{\mathscr{R}_0^d}\right) = \frac{\mu}{\mathscr{R}_0^d}\left(\mathscr{R}_0^d-1\right) > 0.$$

Now, we have

$$\lim_{V\to\left(\frac{d}{c}\right)^-} g_d(V) = \lim_{y\to+\infty}\left(\mu - \frac{p(h+\nu)\left(a+\frac{\alpha d}{c}\right)}{\alpha\big(\rho(h+\nu)e^{-\delta_2\tau_2}+(1-\rho)\nu e^{-\delta_1\tau_1}\big)}\left(\frac{r}{m}+\frac{q\eta}{m}y\right)\right) = -\infty.$$

Furthermore, we have

$$\begin{aligned} g_d'(V) = &-\alpha\frac{p(h+\nu)}{\alpha\big(\rho(h+\nu)e^{-\delta_2\tau_2}+(1-\rho)\nu e^{-\delta_1\tau_1}\big)}\left(\frac{r}{m}+\frac{q\eta}{m(d-cV)}\right)\\ &-(a+\alpha V)\frac{p(h+\nu)}{\alpha\big(\rho(h+\nu)e^{-\delta_2\tau_2}+(1-\rho)\nu e^{-\delta_1\tau_1}\big)}\frac{cq\eta}{m(d-cV)^2}. \end{aligned}$$

Therefore $g_d'(V) < 0$ for all $V \in \left(0, \frac{d}{c}\right)$. Finally, we conclude that there exists a unique $V_1 \in (0, \frac{d}{c})$ such that $g_d(V_1) = 0$. If $\mathscr{R}_0^d > 1$, then the system (2.1) has infected steady state $E_1^d = (S_1, L_1, I_1, V_1, B_1)$, where

$$\begin{aligned} S_1 &= \frac{p(h+\nu)}{\alpha\big(\rho(h+\nu)e^{-\delta_2\tau_2}+(1-\rho)\nu e^{-\delta_1\tau_1}\big)}\left(\frac{r}{m}+\frac{q\eta}{m(d-cV_1)}\right),\\ L_1 &= \frac{(1-\rho)pe^{-\delta_1\tau_1}}{\rho(h+\nu)e^{-\delta_2\tau_2}+(1-\rho)\nu e^{-\delta_1\tau_1}}\left(\frac{rV_1}{m}+\frac{q\eta V_1}{m(d-cV_1)}\right),\\ I_1 &= \frac{rV_1}{m}+\frac{q\eta V_1}{m(d-cV_1)},\\ B_1 &= \frac{\eta}{d-cV_1}. \end{aligned}$$

□

2.3. Global properties

Let the positive definite function $G(z) = z - 1 - \ln z$.

Theorem 2.3. *If $\mathscr{R}_0^d \leqslant 1$, then the disease-free equilibrium E_0^d is G.A.S.*

Proof. Define the Lyapunov function F_0^d given by:

$$\begin{aligned} F_0^d(S,L,I,V,B) = &\gamma S_0 G\left(\frac{S}{S_0}\right)+\frac{\nu}{h+\nu}L+I+\frac{\gamma\alpha S_0}{r+qB_0}V+\frac{\gamma q\alpha S_0}{c(r+qB_0)}B_0 G\left(\frac{B}{B_0}\right)\\ &+\frac{(1-\rho)\nu\alpha e^{-\delta_1\tau_1}}{h+\nu}\int_0^{\tau_1} S(t-\theta)V(t-\theta)d\theta+\rho\alpha e^{-\delta_2\tau_2}\int_0^{\tau_2} S(t-\theta)V(t-\theta)d\theta. \end{aligned}$$

Calculating $\dot{F}_0^d$ along system (2.1) we obtain

$$\begin{aligned} \dot{F}_0^d = &\gamma\frac{(S-S_0)}{S}(\mu-aS-\alpha SV)+\frac{\nu}{h+\nu}\big((1-\rho)e^{-\delta_1\tau_1}\alpha S(t-\tau_1)V(t-\tau_1)-(h+\nu)L\big)\\ &+(\rho e^{-\delta_2\tau_2}\alpha S(t-\tau_2)V(t-\tau_2)+\nu L-pI)+\frac{\gamma\alpha S_0}{r+qB_0}(mI-rV-qBV) \end{aligned}$$

$$
\begin{aligned}
&+\frac{\gamma q\alpha S_0}{c(r+qB_0)}\frac{(B-B_0)}{B}(\eta+cBV-dB)+\frac{(1-\rho)\nu\alpha e^{-\delta_1\tau_1}}{h+\nu}(SV-S(t-\tau_1)V(t-\tau_1))\\
&+\rho\alpha e^{-\delta_2\tau_2}(SV-S(t-\tau_2)V(t-\tau_2))\\
&=\gamma\frac{(S-S_0)}{S}(\mu-aS-\alpha SV)+\frac{\nu}{h+\nu}((1-\rho)e^{-\delta_1\tau_1}\alpha SV-(h+\nu)L)\\
&+(\rho e^{-\delta_2\tau_2}\alpha SV+\nu L-pI)+\frac{\gamma\alpha S_0}{r+qB_0}(mI-rV-qBV)+\frac{\gamma q\alpha S_0}{c(r+qB_0)}\frac{(B-B_0)}{B}(\eta+cBV-dB)\\
&=-\gamma a\frac{(S-S_0)^2}{S}-\frac{\gamma qd\alpha S_0}{c(r+qB_0)}\frac{(B-B_0)^2}{B}+p(\mathscr{R}_0^d-1)I.
\end{aligned}
$$

If $\mathscr{R}\leqslant 1$ we get $\dot{F}_0^d\leqslant 0$ for all $S,L,I,V,B>0$. Let $D_0=\{(S,L,I,V,B):\dot{F}_0^d=0\}$, $D_0=\{E_0\}$. E_0^d is G.A.S.. □

Theorem 2.4. *If $\mathscr{R}_0^d>1$, then the endemic equilibrium E_1^d is G.A.S..*

Proof. Define the Lyapunov function F_1^d given by

$$
\begin{aligned}
F_1^d(S,L,I,V,B)=&\gamma S_1 G\left(\frac{S}{S_1}\right)+\frac{\nu}{h+\nu}L_1 G\left(\frac{L}{L_1}\right)+I_1 G\left(\frac{I}{I_1}\right)+\frac{\gamma\alpha S_1V_1}{mI_1}V_1 G\left(\frac{V}{V_1}\right)\\
&+\frac{\gamma q\alpha S_1V_1}{cmI_1}B_1 G\left(\frac{B}{B_1}\right)+\frac{(1-\rho)\nu}{h+\nu}e^{-\delta_1\tau_1}\alpha S_1V_1\int_0^{\tau_1}G\left(\frac{S(t-\theta)V(t-\theta)}{S_1V_1}\right)d\theta\\
&+\rho e^{-\delta_2\tau_2}\alpha S_1V_1\int_0^{\tau_2}G\left(\frac{S(t-\theta)V(t-\theta)}{S_1V_1}\right)d\theta.
\end{aligned}
$$

Using the fact that $\mu=aS_1+\alpha S_1V_1$, $L_1=\dfrac{(1-\rho)}{(h+\nu)}e^{-\delta_1\tau_1}\alpha S_1V_1$, $mI_1=rV_1+qB_1V_1$, $\eta+cB_1V_1=dB_1$,

$$
pI_1=\gamma\alpha S_1V_1=\frac{(1-\rho)\nu}{h+\nu}e^{-\delta_1\tau_1}\alpha S_1V_1+\rho e^{-\delta_2\tau_2}\alpha S_1V_1,
$$

the derivative of F_1^d with respect to the time t is given by

$$
\begin{aligned}
\dot{F}_1^d=&\gamma\left(1-\frac{S_1}{S}\right)(\mu-aS-\alpha SV)+\frac{\nu}{h+\nu}\left(1-\frac{L_1}{L}\right)((1-\rho)\alpha SV-(h+\nu)L)\\
&+\left(1-\frac{I_1}{I}\right)(\rho\alpha SV+\nu L-pI)+\frac{\gamma\alpha S_1V_1}{mI_1}\left(1-\frac{V_1}{V}\right)(mI-rV-qBV)\\
&+\frac{\gamma q\alpha S_1V_1}{cmI_1}\left(1-\frac{B_1}{B}\right)(\eta+cBV-dB)\\
&+\frac{(1-\rho)\nu}{h+\nu}e^{-\delta_1\tau_1}\alpha S_1V_1\left(\frac{SV}{S_1V_1}-\frac{S(t-\tau_1)V(t-\tau_1)}{S_1V_1}+\ln\left(\frac{S(t-\tau_1)V(t-\tau_1)}{S_1V_1}\right)\right)\\
&+\rho e^{-\delta_2\tau_2}\alpha S_1V_1\left(\frac{SV}{S_1V_1}-\frac{S(t-\tau_2)V(t-\tau_2)}{S_1V_1}+\ln\left(\frac{S(t-\tau_2)V(t-\tau_2)}{S_1V_1}\right)\right)\\
=&-\gamma a\frac{(S-S_1)^2}{S}-\frac{\gamma q\eta}{mcB_1I_1}\alpha S_1V_1\frac{(B-B_1)^2}{B}-\gamma\alpha S_1V_1\,G\left(\frac{S_1}{S}\right)\\
&-\frac{(1-\rho)\nu}{h+\nu}e^{-\delta_1\tau_1}\alpha S_1V_1\,G\left(\frac{S(t-\tau_1)V(t-\tau_1)L_1}{S_1V_1L}\right)\\
&-\rho e^{-\delta_2\tau_2}\alpha S_1V_1\,G\left(\frac{S(t-\tau_2)V(t-\tau_2)I_1}{S_1V_1I}\right)-\frac{(1-\rho)\nu}{h+\nu}e^{-\delta_1\tau_1}\alpha S_1V_1\,G\left(\frac{LI_1}{L_1I}\right)\\
&-\gamma\alpha S_1V_1\,G\left(\frac{I(t-\tau_1)V_1}{I_1V}\right).
\end{aligned}
$$

Therefore $\dot{F}_1^d \leqslant 0$ for all $S, L, I, V, B > 0$. Let $D = \{(S, L, I, V, B) \in \mathbb{R}_+^5;\ \dot{F}_1^d = 0\}$ and N be the largest invariant subset of D. The trajectory of model (2.1) tends to N. It can be verified that $\dot{F}_1^d = 0$ implies $S = S_1$, $I = I_1$, $V = V_1$, $B = B_1$, and

$$\frac{S(t-\tau_1)V(t-\tau_1)L_1}{S_1V_1L} = \frac{S(t-\tau_1)I(t-\tau_1)L_1}{S_1I_1L} = \frac{S(t-\tau_2)V(t-\tau_2)I_1}{S_1V_1I} = \frac{S(t-\tau_2)I(t-\tau_2)}{S_1I}.$$

Applying the above relations to the second equation of system (2.1), we get $L(t) = L_1$ and hence $N = \{E_1^d\}$. This shows that all the sufficient conditions given in Corollary 5.2 of Kuang [20] are satisfied under the condition $\mathscr{R}_0^d > 1$. This implies that E_1^d is G.A.S. when $\mathscr{R}_0^d > 1$. □

3. Numerical method

We will consider the nonlinear differential compact system with proportional delays

$$\dot{u}(t) = c + \varphi\big(t, u(t)\big) + \psi\big(t, u_1(t-\tau_1), \dots, u_n(t-\tau_n)\big), \quad t \in [0, T], \tag{3.1}$$

where $c \in \mathbb{R}^n$, φ and ψ are the linear and nonlinear parts, $\dot{u}$ is the time derivative of the vector function $u = (u_1, u_2, \dots, u_n)$, and $\tau_k \in [0, t[$, $k \in \{1, 2, \dots, n\}$ are a discrete and regular time delays.
Equation (3.1) gives

$$u(t) = ct + \int_0^t \varphi\big(s, u(s)\big)\,ds + \int_0^t \psi\big(s, u_1(s-\tau_1), \dots, u_n(s-\tau_n)\big)\,ds. \tag{3.2}$$

The principle of ADM for DDEs consist to find a solution in the form [4],

$$u(t) = \sum_{i=0}^{\infty} u_i(t). \tag{3.3}$$

Substituting (3.3) in (3.2), we obtain:

$$\begin{cases} u_0 = u(0), \\ u_{i+1} = ct + \int_0^t \varphi\big(s, u_i(s)\big)\,ds + \int_0^t A_i\big(u(s-\tau_k)\big)\,ds, \ 1 \leqslant k \leqslant n. \end{cases} \tag{3.4}$$

With A_i is the Adomian polynomial which generates the nonlinearity at order i, its expression is given by

$$A_i\big(u(s-\tau_k)\big) = \frac{1}{i!}\frac{d^i}{d\lambda^i}\left[\psi\Big(\sum_{j=0}^{i}\lambda^j u_{1,j}(s-\tau_1), \dots, \sum_{j=0}^{i}\lambda^j u_{n,j}(s-\tau_n)\Big)\right]_{\lambda=0}. \tag{3.5}$$

3.1. Application of MADM on CHIKV model with delay

In [5] the authors used the differential transform method (DTM) with multi-step for this CHIKV system without delay. But we have noticed that taking into account of delay by DTM is more complicated than the ADM, this may explain our choice of ADM. This method involves applying the recursive formula

(3.4) for $u = (S, L, I, V, B)$ in the CHIKV dynamics model given by (2.1), we obtain the following system:

$$\begin{cases} S_0 = S(0), \;\; L_0 = L(0), \;\; I_0 = I(0), \;\; V_0 = V(0), \;\; B_0 = B(0), \\ S_1 = \int_0^t (\mu - aS_0)ds - \alpha \int_0^t S_0 V_0 ds, \\ B_1 = \int_0^t (\eta - dB_0)ds + c \int_0^t B_0 V_0 ds, \\ S_{k+1} = -a \int_0^t S_k ds - \alpha \int_0^t A_k(S, V)ds, \;\; k \geqslant 2, \\ L_{k+1} = -(h+\nu) \int_0^t L_k ds + (1-\rho)e^{-\delta_1 \tau_1} \alpha \int_0^t A_k(S(s-\tau_1), V(s-\tau_1))ds, \;\; k \geqslant 1, \\ I_{k+1} = \int_0^t (\nu L_k - pI_k)ds + \rho e^{-\delta_2 \tau_2} \alpha \int_0^t A_k(S(s-\tau_2), V(s-\tau_2))ds, \;\; k \geqslant 1, \\ V_{k+1} = \int_0^t (mI_k - rV_k)ds - q \int_0^t A_k(B, V)ds, \;\; k \geqslant 1, \\ B_{k+1} = -d \int_0^t B_k ds + c \int_0^t A_k(B, V)ds, \;\; k \geqslant 2. \end{cases} \tag{3.6}$$

The resolution of inhomogeneous differential equations by ADM can lead to the noise phenomenon, these terms attenuate or can affect the speed of convergence. The strategy for overcoming this noise phenomenon is choosing the right computational startup. For this, it is convenient to choose the initial terms of the solution suitably. Here, the computational situation in which the convergence will be good for this system under study depends on our choosing the terms S_1 and B_1 appropriately (see the system (3.6)). We report that semi-analytical methods can lead to the phenomenon of noise terms, a topic addressed for example by Wazwaz [27, 28].

To apply the Adomian decomposition method, we consider the following expression

$$x(t) = \sum_{i=0}^{\infty} x_i(t), \quad y(t) = \sum_{i=0}^{\infty} y_i(t),$$

where each variable of (x, y) is one of the variables S, L, I, V, or B. In ours problem, $\psi(x, y) = xy$ introduces each form of nonlinearity that exists, then

$$\psi(x, y) = \sum_{i=0}^{\infty} A_i(x, y)(t),$$

with A_i are Adomian polynomials that generate the nonlinearity as in (3.4). We have (see (3.5))

$$A_i(x, y) = \frac{1}{i!} \frac{d^i}{d\lambda^i} \left[\psi\left(\sum_{j=0}^{i} \lambda^j x_j(t), \sum_{j=0}^{i} \lambda^j y_j(t)\right) \right]_{\lambda=0}.$$

We simply obtain $A_0 = x_0 y_0$, and $A_i = \sum_{k=0}^{i} x_k y_{i-k}$ for $i \geqslant 1$.

3.2. Splitting step

The idea of the multi-step method is simple, its effectiveness based on the good precision of the Adomian method when the time variable is close to zero. To calculate the solution $u = (S, L, I, V, B)$ on the interval $I = [0, T]$ with $T > 0$, we start with the first step, where we calculate the solution by ADM on the interval $[0, t_1]$, $t_k = i\Delta t$, $i \in \mathbb{N}$ and with the initial condition $U(0) = (S_0, L_0, I_0, V_0, B_0)$. At the k^{th} step, we seek the solution U_k on the interval $I_k = [t_k, t_{k+1}]$ with the initial condition $U_k(0) = U_{k-1}(t_k)$ thus in succession until covering the interval $I = [0, T]$.

4. Numerical examples

In order to validate the MADM, we have performed some numerical tests on the spread of the Chikungunya virus epidemic. All calculations were done with a time discretization $\Delta t = 0.1$ and a stopping condition requiring error $\varepsilon = 10^{-6}$. We have used Matlab in all calculations and its performer DDE32 function that represents the Runge-Kutta (RK) as a reference method to compare it with MADM.

We consider the following values of the parameters: $\mu = 6$, $a = 0.1$, $\alpha = 2$, $\rho = 0.5$, $h = 0.1$, $\nu = 1$, $p = 0.1$, $m = 0.2$, $r = 0.1$, $q = 1$, $\eta = 5$, $c = 0.1$, $d = 1$, $\delta_1 = 2$, and $\delta_2 = 1$. With the initial conditions $S(0) = 2$, $L(0) = 0.2$, $I(0) = 0.4$, $V(0) = 0.4$, and $B(0) = 1$. We started with a first test with delay $(1, 0.5)$ (i.e., $\tau_1 = 1$ and $\tau_2 = 0.5$), where we aim to evaluate the MADM solution with the RK method. With these parameters, we obtain a rapid convergence of the B-cells B and latently infected cells L. Thus, for the uninfected cells variable, we notice that S pass through a peak before taking the rhythm towards the point of stability (see Figure 1). These parameters used induce a value of the basic reproduction number $\mathscr{R}_0^d = 17.1662$, we notice that we have represented the unknowns of the problem that are close to each other to the stability point in a same figure. Figure 2 shows the comparison of the Adomian

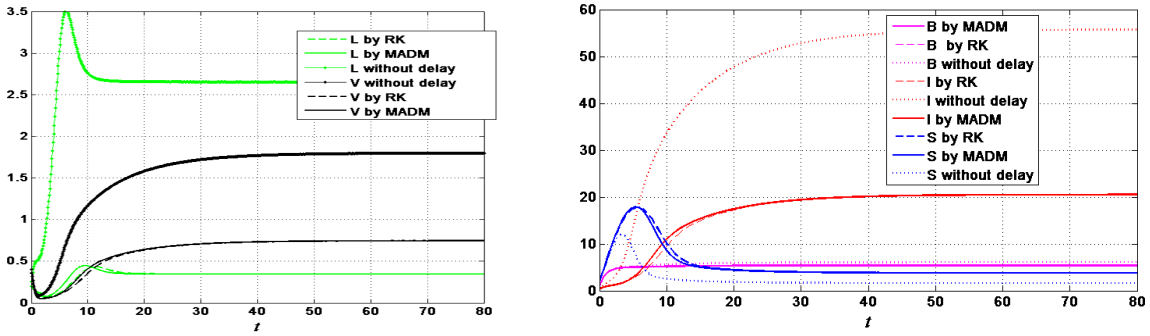

Figure 1: MADM solution (S, L, I, V, B) compared to DDE23 solution with delay $(1, 0.5)$ and the solution without delay.

decomposition method with/without the multi-step technique. The ADM without the multi-step cannot be effective. Both methods can give comparable results but only on an interval that does not exceed $[0, 1]$ and perhaps the delay is excluded. With MADM we keep the major advantage of ADM which is simplicity and ease of implementation. Figure 1 shows that the solution is close to that calculated by the

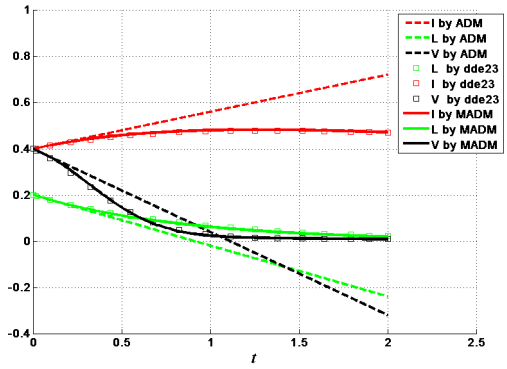

Figure 2: ADM solution (L, I, V) compared to dde23 and MADM solutions.

RK method. In this situation with the delay $(1, 0.5)$, we detect an absolute error at most 10^{-2}. But the error rate at the equilibrium point is acceptable as shown in Table 1. If we assume that the stability point of V is $V_1 = 0.36943$ (value calculated by DDE32), so with this value we can estimate a precise value of stability point in accordance with the study carried out in the Section 2. Then, for $\mathscr{R}_0^d = 17.1662 > 1$, the stability point of the solution is $E_1^d = (S_1, L_1, I_1, V_1, B_1)$; this is an endemic equilibrium and we obtain the results presented in Table 1.

Figure 3 illustrates the change of solutions as a function of the chosen delays τ_1 and τ_2. We note that the magnification of the delay factor τ_1 reduces the concentrations of latently infected host cells L more than magnification of the delay factor τ_2, and vice versa for the concentrations of the active free CHIKV particles V. Overall, the magnification of the delay factors τ_1 and/or τ_2 decrease the concentrations of latently infected host cells L, actively infected host cells I and CHIKV particles V, and increase the concentrations of the uninfected host cells S. Therefore, the concentrations of B cells that responsible for

Table 1: Summary of absolute errors of stability point.

Variable	S_1	L_1	I_1	V_1	B_1
MADM	7.1510	0.0059	9.7769	0.3695	5.1920
DDE23	7.15153	0.00595	9.77680	0.36943	5.19183
Exact	7.14851	0.00595	9.77474	0.36943	5.19180
Error	2.4×10^{-3}	5×10^{-5}	2.2×10^{-3}	7×10^{-5}	2×10^{-4}

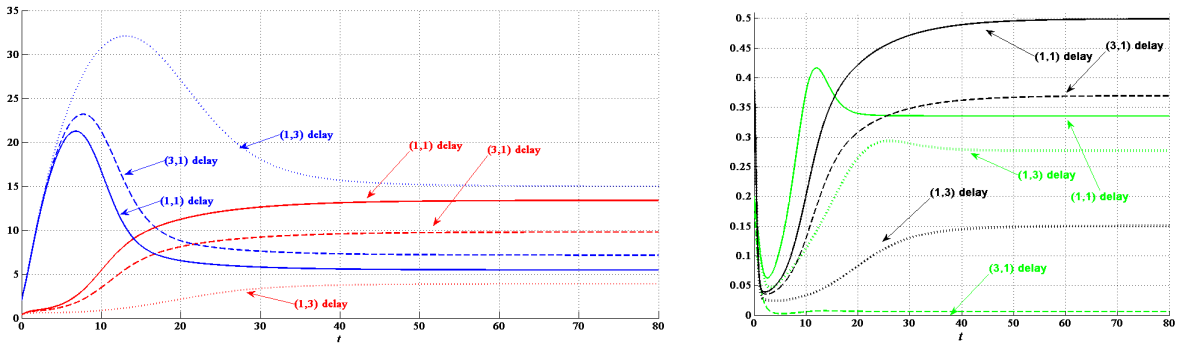

Figure 3: The effect of the different delays on the solution (S in bleu, L in green, I in red, V in Black).

immunity against foreign antigens decreases as we magnify the delay factors. Thus, taking into account a larger historic part of the condition of the infected (large delay factors) enhances success in controlling the epidemic.

We now consider the following new values of the CHIKV dynamic model parameters: $\mu = 6$, $a = 1$, $\alpha = 0.1$, $\rho = 0.5$, $h = 0.1$, $\nu = 1$, $p = 0.1$, $m = 0.2$, $r = 0.1$, $q = 1$, $\eta = 0.5$, $c = 0.1$, $d = 0.1$, $\delta_1 = 2$, and $\delta_2 = 1$; these parameters induce a value of the basic reproduction number $\mathscr{R}_0^d = 0.0858$. The stability study seen in Section 2 shows that the solution (S, L, I, V, B) of the system converges towards the solution $E_0^d = (S_0, 0, 0, 0, B_0)$; there is a disease-free equilibrium state. Numerically, we find the limit to be $(5.9999, 6 \times 10^{-8},\ 21 \times 10^{-4}, 8 \times 10^{-5}, 4.9926)$, see Figure 4. We notice that with the decreasing of the B-cells (by decreasing η and increasing d) and with $\mathscr{R}_0^d$ close to zero, the convergence of S (uninfected monocytes) is speedy towards the solution $S_0 = \mu/a = 6$. On the other hand, the convergence of B towards $B_0 = \eta/d = 5$ is slower than the convergence of S. For the rest of the unknowns (L, I and V), we can see the influence of the both delays on them which consists in accelerating their convergence towards 0.

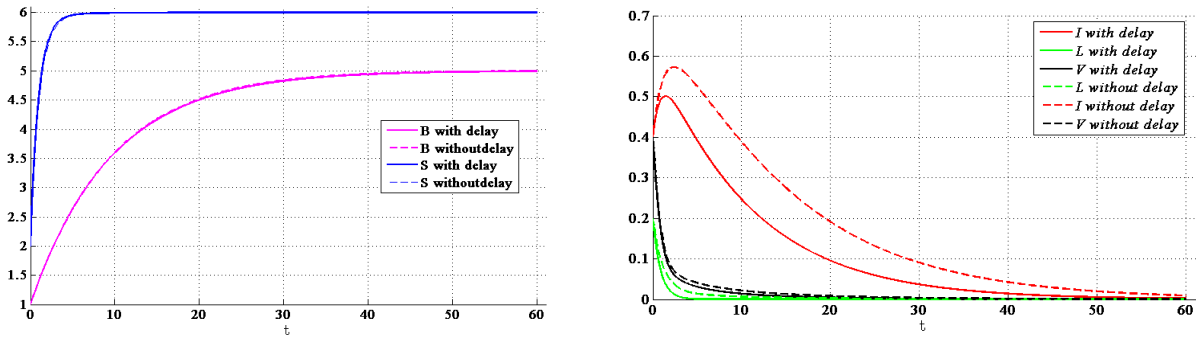

Figure 4: The behavior of solution with and without delay in a disease-free equilibrium state.

5. Discussion

The Adomian decomposition method is simple and practical. Its great advantage is that one can obtain in a few iterations an approximate or even closed analytical solution. But its success is, sometimes, only in a very restricted field of study which generally does not exceed the interval $[0, 1]$. This is why the combination of ADM and a multi-step technique is necessary for a dynamic system to make further progress in the computation of the solution on a large interval $[0, T]$, $T > 1$. This conclusion is visible in Figure 2 which shows that the solutions obtained by ADM cannot be used to approximate the solution of a dynamical system outside the interval $[0, 1]$. Moreover, it can be concluded from these plots that the graphs of MADM and dde23 solutions are similar in this calculation. The layouts of these solutions appear to be identical with a maximum absolute error bound of 2×10^{-3} in the L and I solutions which are the variables most concerned by the delays, while we found a maximum absolute error bound of 10^{-5} in the V solution.

6. Conclusions

The ADM can work well with the Chikungunya delay differential system. It is distinguished by its flexibility and its accessibility to a specific solution. However, due to the limitations of the study area, we used MADM which consists of managing ADM with a multi-step technique to achieve a solution in the widest possible study area. This method is efficient and can be applied to dynamical systems that exhibit more nonlinearity or extreme delays because this method is free from theoretical complexity. For this, we are thinking of extending the application of this method to a more generalized Chikungunya system.

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