JOURNAL OF INFORMATION SCIENCE AND ENGINEERING 36, 1261-1277 (2020)
DOI: 10.6688/JISE.202011_36(6).0009

Modeling and Analysis of Scabies Transmission Disease

N. H. ALSHAMRANI[1,2], A. M. ELAIW[1,3], H. A. BATARFI[1]
AND A. D. HOBINY[1]
[1]*Department of Mathematics*
King Abdulaziz University
Jeddah, 21589 Saudi Arabia
E-mail: {nalshmrane; aelaiwksu.edu.sa; hbatarfi; ahobany}@kau.edu.sa
[2]*Department of Mathematics*
University of Jeddah
Jeddah, 21589 Saudi Arabia
E-mail: nhalshamrani@uj.edu.sa
[3]*Department of Mathematics*
Al-Azhar University
Assiut, 71524 Egypt

In this work we present two mathematical models for the infection dynamics of scabies. The dynamics is described by four-dimensional system of ordinary differential equations that expresses the transmissions between susceptible and infectious/infective individuals. In the second model, we include the importance of adult scabiei mite in the real interaction with hosts. Nonnegativity and boundedness of solutions of the models are conducted. A threshold parameter is calculated for each model which ensures the existence of all corresponding equilibria. Using candidate Lyapunov functions, it is shown that whenever the threshold parameter is less than or equal unity, the models have an associated disease-free equilibrium that is globally asymptotically stable. In addition, when the threshold exceeds unity the models have a globally asymptotically stable endemic equilibrium. Finally, using some parameter values related to the scabies infection dynamics, numerical simulation results are demonstrated to clarify the main theoretical results.

Keywords: scabies transmissions, infectious diseases, local stability, global stability, infection rate, Lyapunov function

1. INTRODUCTION

Scabies is one of the infectious diseases caused by an ectoparasitic mite known as Sarcoptes scabiei and belonged to a kind of skin infestations. Worldwide, around three hundred million cases diagnosed with scabies annually and this fact makes scabies globally a significant public health matter [1-3]. In fact, scabies is commonly transmitted in poor communities living in crowded conditions. As an example, prevalence in northern Australia is as high as 49% compared to the percentage of infested individuals in Fiji prevalence and Solomon Islands which is 28% and 43% [4, 5]. Based on the Saudi Ministry of Health, in the first six months of 2018, more than 1700 infected individuals were diagnosed with scabies in Mecca which is located in the western region of Saudi Arabia [6].

Received January 31, 2020; revised February 26, 2020; accepted March 3, 2020.
Communicated by Mahmoud Abdel-Aty.

The major mode of scabies transmission in individuals is direct contact through skin-to-skin. Another way of transmission is due to sharing articles or the usage of personal stuff that are infected with mites such as bed sheet, clothing, towels, *etc*. In addition, sexual contact is a direct path to spread the infestation [4, 7-9]. Actually, there is a prolong period up to ten weeks between the first infestation and the time when the infected individual begins to develop symptoms. During the asymptomatic period, infected individuals with scabies can spread the infestation throughout the population [4, 9].

There are three general life stages for scabies mite progression inside the hosts: egg, young mite and adult [3, 4]. In the beginning, the pregnant mites start tunneling as soon as they transferred, and lay two to three eggs each day [4, 10]. The incubation time for eggs before being hatched is about two days [11, 13]. Then, larvae with three pairs of legs reaches the skin surface and starts borrowing for approximately five days [11, 13]. After that, there is two developmental stages for larvae passing through before the nymph (young mites) stage that are Protonymphs and Tritonymphs [11, 12]. In the meantime, the mites keep roaming about the body for the nymph stage [14]. The last stage is for young mites being either male or female adult in approximately five days [11, 12]. These adult mites takes around two weeks searching for a partner. After sexual contact the pregnant female mites start to lay eggs completing the cycle of infestation. In fact, male mites die immediately after mating. Comparing to the male, female mites can survive up to one-two months [3]. After approximately 30 days of initial infestation the second generation of adult mites appears and start the cycle of infestation from the beginning [4, 14].

In the last decades, studying a population dynamics of infectious diseases transmissions has attracted much efforts and considerations from researchers. Many sophisticated mathematical models have been developed to investigate the transmission dynamics of infectious diseases through entire populations such as dengue [15], malaria [16], yellow fever [17], Zika [18] and cholera [19]. The great advantage of mathematical models and their analysis is to understand the disease transmissions and to play a crucial role in controlling the infection within entire populations. In fact, mathematical modeling has a pivotal ability in expressing the most important experimental observations and features of real phenomenons. It seems like an adaptive procedure which develops new models using collected information from experimental trials. The affordability in the clinical trial needs and medication costs reflect a significant importance of modeling.

From an epidemic point of view, some researchers have paid their attention to investigate scabies infection dynamics [2, 7, 20]. However, these models have neglected the aspects of Sarcoptes scabiei life cycle as well as the real interaction between mites and susceptible individuals. In a very recent work, Lydeamore *et al*. [4] have formulated a mathematical model which described the scabies infection dynamics and considered the mite's life cycle and its interact to hosts. Nevertheless, the basic and global properties of their model have not been studied.

The aim of the present paper is to formulate two mathematical models that characterize the scabies infection dynamics among susceptible and infective individuals in a population. In the second model, we include the adult mite state into the dynamics. We investigate the basic dynamical behavior of the constructed models such as the nonnegativity and boundedness. We derive a threshold parameter that fully determined the existence and global stability of the model's equilibria. We carry out numerical simulations that substantiate the theoretical results.

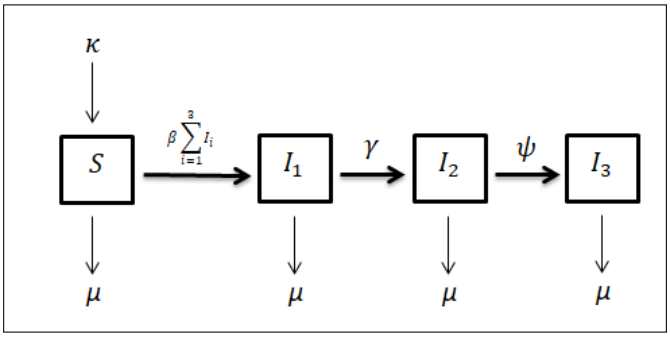

Fig. 1. Scheme of the basic model. Parameter β refers to the infection rate constant. In addition, parameters κ, γ, ψ represent the birth rate constant of susceptible individuals, the transmission rate constant of developing symptoms, and the transmission rate constant for egg hatching, respectively. Furthermore, all individuals have a natural death rate constant μ which is independent of disease status.

2. SCABIES TRANSMISSION MODEL

In this section, we aim to formulate and analyze the transmission of scabies disease through its target population. In fact, the targeting population can be divided into four categories: susceptible individuals (S), infectious individuals that having living mites and eggs during the asymptomatic period after infection (I_1), infectious individuals that having living mites and eggs after developing symptoms (I_2), and the infected individuals who have only young mites (*i.e.* eggs have been hatched) (I_3). Fig. 1 represents the transition scheme of scabies transmission through its individuals in a population.

Consequently, our mathematical model which is characterizing the scabies dynamics in a population is given by the following system of differential equations:

$$\dot{S}(t) = \kappa - \mu S(t) - \beta S(t) \sum_{i=1}^{3} I_i(t), \tag{1}$$

$$\dot{I}_1(t) = \beta S(t) \sum_{i=1}^{3} I_i(t) - (\gamma + \mu) I_1(t), \tag{2}$$

$$\dot{I}_2(t) = \gamma I_1(t) - (\psi + \mu) I_2(t), \tag{3}$$

$$\dot{I}_3(t) = \psi I_2(t) - \mu I_3(t), \tag{4}$$

where $\beta S \sum_{i=1}^{3} I_i$ is the infection rate. All variables and parameters are described as above.

2.1 Basic Properties

In this subsection, we investigate the non-negativity and boundedness of the solutions. In addition we calculate the equilibrium points. We consider the following lemma:
Lemma 1. Let $\Omega_1 > 0$ and define

$$\Gamma_1 = \left\{ (S, I_1, I_2, I_3) \in \mathbb{R}^4_{\geq 0} : 0 \leq S + I_1 + I_2 + I_3 \leq \Omega_1 \right\}.$$

Then, the compact set Γ_1 is positively invariant for system (1)-(4).

Proof: We have

$$\dot{S}\,|_{S=0}=\kappa>0, \quad \dot{I}_1\,|_{I_1=0}=\beta S(I_2+I_3)\geq 0 \quad \text{for all } S,I_2,I_3\geq 0,$$
$$\dot{I}_2\,|_{I_2=0}=\gamma I_1\geq 0 \quad \text{for all } I_1\geq 0, \quad \dot{I}_3\,|_{I_3=0}=\psi I_2\geq 0 \quad \text{for all } I_2\geq 0.$$

Hence, the orthant $\mathbb{R}^4_{\geq 0}$ is positively invariant for system (1)-(4).

Next we prove that the solutions of system (1)-(4) are bounded. Let $\Phi_1(t)=S(t)+\sum_{i=1}^{3} I_i(t)$, then

$$\dot{\Phi}_1(t)=\kappa-\mu S(t)-\mu\sum_{i=1}^{3} I_i(t)=\kappa-\mu\left(S(t)+\sum_{i=1}^{3} I_i(t)\right)=\kappa-\mu\Phi_1(t).$$

It follows that, $0\leq\Phi_1(t)\leq\Omega_1$ if $\Phi_1(0)\leq\Omega_1$ for $t\geq 0$, where $\Omega_1=\frac{\kappa}{\mu}$. This implies that, $0\leq S(t)+\sum_{i=1}^{3} I_i(t)\leq\Omega_1$ if $0\leq S(0)+\sum_{i=1}^{3} I_i(0)\leq\Omega_1$. Hence, Γ_1 is positively invariant with respect to model (1)-(4). □

We define a threshold parameter $R_0=\frac{\beta S_0}{\mu}$ where $S_0=\kappa/\mu$. The parameter R_0 determines whether the disease will progress or not and defines as the number of secondary scabies cases produced by one infectious individual during his/her entire infectious period. system (1)-(4) admits two equilibrium points that are disease-free equilibrium $Q_0=(S_0,0,0,0)$ and endemic equilibrium $\bar{Q}=(\bar{S},\bar{I}_1,\bar{I}_2,\bar{I}_3)$ where

$$\bar{S}=\frac{S_0}{R_0}, \qquad \bar{I}_1=\frac{\mu^2}{\beta(\gamma+\mu)}(R_0-1),$$
$$\bar{I}_2=\frac{\gamma\mu^2}{\beta(\gamma+\mu)(\psi+\mu)}(R_0-1), \quad \bar{I}_3=\frac{\gamma\psi\mu}{\beta(\gamma+\mu)(\psi+\mu)}(R_0-1).$$

2.2 Local Stability Analysis of the Equilibria

In order to investigate the local stability of the two equilibria, we linearize system (1)-(4) which results in the following Jacobian matrix:

$$J(S,I_1,I_2,I_3)=\begin{pmatrix} -\mu-\beta\sum_{i=1}^{3} I_i & -\beta S & -\beta S & -\beta S \\ \beta\sum_{i=1}^{3} I_i & \beta S-(\gamma+\mu) & \beta S & \beta S \\ 0 & \gamma & -(\psi+\mu) & 0 \\ 0 & 0 & \psi & -\mu \end{pmatrix}. \tag{5}$$

Accordingly, we have the following results:

Theorem 1: For system (1)-(4) the disease-free equilibrium Q_0 is locally asymptotically stable when $R_0<1$, and unstable when $R_0>1$.

Proof: The Jacobian matrix given in (5) at the disease-free equilibrium Q_0, $J(Q_0)$ provided us with four eigenvalues

$$\lambda_1=-\mu, \quad \lambda_2=-(\gamma+\mu), \quad \lambda_3=-(\psi+\mu), \quad \lambda_4=\frac{\kappa\beta-\mu^2}{\mu}=\mu(R_0-1).$$

Clearly, all eigenvalues of the characteristic equation at Q_0 are negative when $R_0 < 1$. Thus, the disease-free equilibrium Q_0 is locally asymptotically stable when $R_0 < 1$, and unstable when $R_0 > 1$. □

Theorem 2: For system (1)-(4) the endemic equilibrium $\bar{Q}$ is locally asymptotically stable when $R_0 > 1$.

Proof: The Jacobian matrix given in (5) at the endemic equilibrium $\bar{Q}$, $J(\bar{Q})$ provided us with four eigenvalues

$$\lambda_1 = -\mu, \quad \lambda_2 = -(\gamma+\mu), \quad \lambda_3 = -(\psi+\mu), \quad \lambda_4 = \frac{\mu^2 - \kappa\beta}{\mu} = \mu\,(1-R_0)\,.$$

Clearly, all eigenvalues of the characteristic equation at $\bar{Q}$ are negative when $R_0 > 1$. Thus, the endemic equilibrium $\bar{Q}$ is locally asymptotically stable when $R_0 > 1$. □

2.3 Global Stability Analysis of the Equilibria

In this subsection we investigate the global asymptotic stability of the equilibria of model (1)-(4) using Lyapunov method which has been used in epidemiological models [21-24], and virological models [25-36]. We will use the notation $(S, I_1, I_2, I_3) = (S(t), I_1(t), I_2(t), I_3(t))$. Let us define the function $F : (0,\infty) \to [0,\infty)$ as $F(\eta) = \eta - 1 - \ln\eta$. It is clear that $F(\eta) \geq 0$ for any $\eta > 0$ and $F(\eta) = 0$ if and only if $\eta = 1$. The stability of the infection-free equilibrium Q_0 will be given in the following result.

Theorem 3: For system (1)-(4) suppose that $R_0 \leq 1$, then the disease-free equilibrium Q_0 is globally asymptotically stable in Γ_1.

Proof: We construct a Lyapunov function candidate as:

$$L_0(S, I_1, I_2, I_3) = S_0 F\left(\frac{S}{S_0}\right) + I_1 + I_2 + I_3. \tag{6}$$

It is seen that, $L_0(S, I_1, I_2, I_3) > 0$ for all $S, I_1, I_2, I_3 > 0$ and $L_0(S_0, 0, 0, 0) = 0$. We calculate $\frac{dL_0}{dt}$ along the solutions of model (1)-(4) as:

$$\begin{aligned}\frac{dL_0}{dt} &= \left(1 - \frac{S_0}{S}\right)\left(\kappa - \mu S - \beta S \sum_{i=1}^{3} I_i\right) + \beta S \sum_{i=1}^{3} I_i - (\gamma+\mu) I_1 \\ &\quad + \gamma I_1 - (\psi+\mu) I_2 + \psi I_2 - \mu I_3 \\ &= -\mu \frac{(S-S_0)^2}{S} + \mu \sum_{i=1}^{3} I_i\,(R_0 - 1)\,. \end{aligned} \tag{7}$$

Therefore, if $R_0 \leq 1$, then $\frac{dL_0}{dt} \leq 0$ for all $S, I_1, I_2, I_3 > 0$ with equality holding when $S = S_0$ and $I_1 = I_2 = I_3 = 0$. We note that solutions of system (1)-(4) are limited to Υ_0', the largest invariant subset of $\Upsilon_0 = \left\{(S, I_1, I_2, I_3) : \frac{dL_0}{dt} = 0\right\}$ [37]. Using LaSalle's invariance principle, we conclude that $\Upsilon_0' = \{Q_0\}$ and the equilibrium Q_0 is globally asymptotically stable. □

Theorem 4: For system (1)-(4) assume that $R_0 > 1$, then the endemic equilibrium point $\bar{Q}$ is globally asymptotically stable in $\overset{\circ}{\Gamma}_1$.

Proof: To prove that the system is globally asymptotically stable at $\bar{Q}$, we investigate this system by introducing the following Lyapunov function candidate:

$$L_1(S,I_1,I_2,I_3) = \bar{S}F\left(\frac{S}{\bar{S}}\right) + \left(\sum_{i=1}^{3}\bar{I}_i\right)F\left(\sum_{i=1}^{3}I_i/\sum_{i=1}^{3}\bar{I}_i\right),$$

where $L_1(S,I_1,I_2,I_3) > 0$ for all $S, I_1, I_2, I_3 > 0$ and $L_1(\bar{S},\bar{I}_1,\bar{I}_2,\bar{I}_3) = 0$. Now, we need to show that the equilibrium point is globally attractive. The derivative of L_1 with respect to time along the trajectories of system (1)-(4) is given as follows:

$$\begin{aligned}\frac{dL_1}{dt} &= \left(1-\frac{\bar{S}}{S}\right)\left(\kappa-\mu S-\beta S\sum_{i=1}^{3}I_i\right)+\left(1-\left(\sum_{i=1}^{3}\bar{I}_i/\sum_{i=1}^{3}I_i\right)\right)\left(\beta S\sum_{i=1}^{3}I_i-\mu\sum_{i=1}^{3}I_i\right)\\ &= \left(1-\frac{\bar{S}}{S}\right)(\kappa-\mu S)+\beta\bar{S}\sum_{i=1}^{3}I_i-\mu\sum_{i=1}^{3}I_i-\beta S\sum_{i=1}^{3}\bar{I}_i+\mu\sum_{i=1}^{3}\bar{I}_i. \qquad (8)\end{aligned}$$

Using the equilibrium point conditions of $\bar{Q}$

$$\kappa = \mu\bar{S}+\beta\bar{S}\sum_{i=1}^{3}\bar{I}_i, \quad \beta\bar{S}\sum_{i=1}^{3}\bar{I}_i = \mu\sum_{i=1}^{3}\bar{I}_i,$$

we obtain

$$\frac{dL_1}{dt} = -\mu\frac{(S-\bar{S})^2}{S}+\beta\bar{S}\sum_{i=1}^{3}\bar{I}_i\left(2-\frac{\bar{S}}{S}-\frac{S}{\bar{S}}\right) = -\left(\mu+\beta\sum_{i=1}^{3}\bar{I}_i\right)\frac{(S-\bar{S})^2}{S}.$$

Clearly, $\frac{dL_1}{dt} \le 0$ for all $S, I_i > 0$, $i = 1,2,3$ with equality holding when $S = \bar{S}$. Let $\Upsilon_1 = \left\{(S,I_1,I_2,I_3) : \frac{dL_1}{dt} = 0\right\}$ and Υ_1' is the largest invariant subset of Υ_1. We note that, the solutions of system (1)-(4) are confined to Υ_1' [37]. The set Υ_1' is invariant and contains elements which satisfy $S(t) = \bar{S}$. Then, $\dot{S}(t) = 0$ and from Eqs. (1) and (2), we have

$$0 = \dot{S}(t) = \kappa-\mu\bar{S}-(\gamma+\mu)I_1(t),$$

which gives that $I_1(t) = \bar{I}_1$ for all t. Similarly, one can easily verified that Υ_1' contains elements satisfying $I_2(t) = \bar{I}_2$ and $I_3(t) = \bar{I}_3$ for all t. Therefore, from the local stability result constructed earlier and global attractive property proven here, we have shown that the solution trajectories will approach $\bar{Q}$ asymptotically and $\bar{Q}$ is globally asymptotically stable using LaSalle's invariance principle [37]. □

3. SCABIES TRANSMISSION WITH ADULT MITES

The model presented in the previous section does not consider the adult mites state and the effect of this on the infectiousness and scabies transmissions. This omission leads us to modify model (1)-(4) by incorporating the adult mites state (M). In fact, the infection rate which describes the probability of contact and infectiousness transmissions between adult mites and susceptible individuals is represented by the term ($\phi\beta MS$), where $\phi \in$

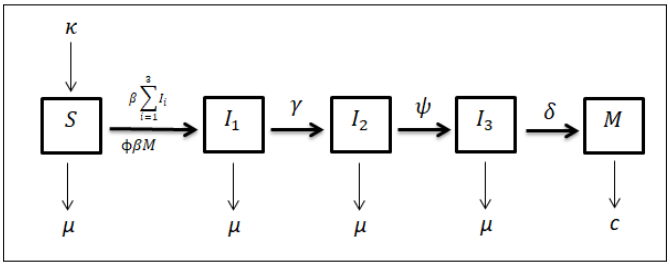

Fig. 2. Scheme of model (9)-(13).

$[0,1]$ is the susceptibility to infection due to mites. Fig. 2 illustrates the structure of this dynamics.

Our proposed model is given by five-dimensional ODEs in the form:

$$\dot{S}(t) = \kappa - \mu S(t) - \beta S(t)\sum_{i=1}^{3} I_i(t) - \phi\beta M(t)S(t), \tag{9}$$

$$\dot{I}_1(t) = \beta S(t)\sum_{i=1}^{3} I_i(t) + \phi\beta M(t)S(t) - (\gamma+\mu)I_1(t), \tag{10}$$

$$\dot{I}_2(t) = \gamma I_1(t) - (\psi+\mu)I_2(t), \tag{11}$$

$$\dot{I}_3(t) = \psi I_2(t) - (\delta+\mu)I_3(t), \tag{12}$$

$$\dot{M}(t) = \delta I_3(t) - cM(t), \tag{13}$$

where δ is the transmission rate constant for mites mature and become adults. Moreover, c represents the natural death rate constant of the adult mites which is independent of disease status. Moreover, this model includes all biological relevant features for the remaining variables and parameters.

3.1 Properties of Solutions

In this subsection, we study some properties of solutions of the model such as the non-negativity and boundedness. Moreover, we calculate the equilibrium points. Let $\mathbb{R}^5_{\geq 0} = \{(x_1,x_2,x_3,x_4,x_5) : x_i \geq 0,\ i = 1,...,5\}$ and consider the following lemma:

Lemma 2. Let $\Omega_2 > 0$ and define

$$\Gamma_2 = \left\{(S,I_1,I_2,I_3,M) \in \mathbb{R}^5_{\geq 0} : 0 \leq S+I_1+I_2+I_3+M \leq \Omega_2\right\}.$$

Then, the compact set Γ_2 is positively invariant for system (9)-(13).

Proof: The proof is similar to the proof of Lemma 1. □

Let us define the threshold parameter for system (9)-(13) as:

$$R_0^M = \frac{\beta S_0\{c(\gamma+\mu)(\delta+\mu) + c\psi(\gamma+\delta+\mu) + \phi\gamma\delta\psi\}}{c(\gamma+\mu)(\delta+\mu)(\psi+\mu)}.$$

System (9)-(13) admits two equilibria as follows:

(i) Disease-free equilibrium point $Q_0^M = (S_0,0,0,0,0)$, where $S_0 = \dfrac{\kappa}{\mu}$.

(ii) Endemic equilibrium point $\bar{Q}^M = (\bar{S}, \bar{I}_1, \bar{I}_2, \bar{I}_3, \bar{M})$, where

$$\bar{S} = \frac{S_0}{R_0^M}, \qquad \bar{I}_1 = \frac{c\mu(\delta+\mu)(\psi+\mu)}{\Upsilon}\left(R_0^M - 1\right),$$
$$\bar{I}_2 = \frac{c\mu\gamma(\delta+\mu)}{\Upsilon}\left(R_0^M - 1\right), \quad \bar{I}_3 = \frac{c\mu\gamma\psi}{\Upsilon}\left(R_0^M - 1\right), \quad \bar{M} = \frac{\mu\gamma\delta\psi}{\Upsilon}\left(R_0^M - 1\right).$$

3.2 Global Stability Analysis of the Equilibria

In this subsection, we study the global stability of system (9)-(13) by using Lyapunov method. We will use the notation $(S, I_1, I_2, I_3, M) = (S(t), I_1(t), I_2(t), I_3(t), M(t))$.

Theorem 5: For system (9)-(13) suppose that $R_0^M \leq 1$, then the disease-free equilibrium Q_0^M is globally asymptotically stable in Γ_2.

Proof: A Lyapunov function candidate is given by:

$$V_0(S, I_1, I_2, I_3, M) = S_0 F\left(\frac{S}{S_0}\right) + I_1 + \frac{\beta S_0 (c\delta + c\mu + c\psi + \phi\delta\psi)}{c(\delta+\mu)(\psi+\mu)} I_2 + \frac{\beta S_0 (c + \phi\delta)}{c(\delta+\mu)} I_3 + \frac{\phi\beta S_0}{c} M.$$

It is seen that, $V_0(S, I_1, I_2, I_3, M) > 0$ for all $S, I_1, I_2, I_3, M > 0$, and $V_0(S_0, 0, 0, 0, 0) = 0$. Calculating $\frac{dV_0}{dt}$ and collecting terms we get

$$\frac{dV_0}{dt} = -\mu\frac{(S - S_0)^2}{S} + (\gamma + \mu)\left(R_0^M - 1\right) I_1.$$

Therefore, if $R_0^M \leq 1$, then $\frac{dV_0}{dt} \leq 0$ for all $S, I_1, I_2, I_3, M > 0$ with equality holding when $S = S_0$ and $I_1 = 0$. The solutions of system (9)-(13) reach $\Lambda_0^{'}$, the largest invariant subset of $\Lambda_0 = \left\{(S, I_1, I_2, I_3, M) : \frac{dV_0}{dt} = 0\right\}$. The set $\Lambda_0^{'}$ is invariant and for any element belongs to Λ_0 satisfies $S(t) = S_0$ and $I_1(t) = 0$. According to the LaSalle's invariance principle $\lim_{t\to\infty} S(t) = S_0$ and $\lim_{t\to\infty} I_1(t) = 0$. Then, $\dot{S}(t) = 0$ and $\dot{I}_1(t) = 0$. From Eqs. (11)-(13), we have

$$\dot{I}_2(t) = -(\psi + \mu) I_2(t), \tag{14}$$
$$\dot{I}_3(t) = \psi I_2(t) - (\delta + \mu) I_3(t), \tag{15}$$
$$\dot{M}(t) = \delta I_3(t) - cM(t). \tag{16}$$

Let us define a Lyapunov function as $\tilde{V}_0 = I_2(t) + I_3(t) + M(t)$. Therefore, the time derivative of $\tilde{V}_0$ along the solutions of (14)-(16) can be calculated as follows:

$$\frac{d\tilde{V}_0}{dt} = -\mu I_2(t) - \mu I_3(t) - cM(t) \leq 0.$$

Clearly $\frac{d\tilde{V}_0}{dt} = 0$ if and only if $I_2(t) = I_3(t) = M(t) = 0$ for all t. Let $\Lambda_0^{''} = \{(S, I_1, I_2, I_3, M) \in \Lambda_0^{'} : \frac{d\tilde{V}_0}{dt} = 0\} = \left\{(S, I_1, I_2, I_3, M) \in \Lambda_0^{'} : S = S_0, I_1 = I_2 = I_3 = M = 0\right\} = \left\{Q_0^M\right\}$. Hence, all solutions trajectories approach Q_0^M and this means that Q_0^M is globally asymptotically stable [37]. □

Theorem 6: For system (9)-(13) assume that $R_0^M > 1$, then the endemic equilibrium point $\bar{Q}^M$ is globally asymptotically stable in $\overset{\circ}{\Gamma}_2$.

Proof: We consider a candidate Lyapunov function as:

$$V_1(S,I_1,I_2,I_3,M) = \bar{S}F\left(\frac{S}{\bar{S}}\right) + \bar{I}_1F\left(\frac{I_1}{\bar{I}_1}\right) + \frac{\beta\bar{S}(\bar{I}_2+\bar{I}_3+\phi\bar{M})}{(\psi+\mu)\bar{I}_2}\bar{I}_2F\left(\frac{I_2}{\bar{I}_2}\right) + \frac{\beta\bar{S}(\bar{I}_3+\phi\bar{M})}{(\delta+\mu)\bar{I}_3}\bar{I}_3F\left(\frac{I_3}{\bar{I}_3}\right) + \frac{\phi\beta\bar{S}\bar{M}}{\delta\bar{I}_3}\bar{M}F\left(\frac{M}{\bar{M}}\right).$$

Clearly, $V_1(S,I_1,I_2,I_3,M) > 0$ for all $S,I_1,I_2,I_3,M > 0$ and $V_1(\bar{S},\bar{I}_1,\bar{I}_2,\bar{I}_3,\bar{M}) = 0$. The derivative $\frac{dV_1}{dt}$ is calculated as:

$$\begin{aligned}\frac{dV_1}{dt} &= \left(1-\frac{\bar{S}}{S}\right)(\kappa-\mu S-\beta S(I_1+I_2+I_3+\phi M)) + \left(1-\frac{\bar{I}_1}{I_1}\right)(\beta S(I_1+I_2+I_3+\phi M)-(\gamma+\mu)I_1)\\ &+\frac{\beta\bar{S}(\bar{I}_2+\bar{I}_3+\phi\bar{M})}{(\psi+\mu)\bar{I}_2}\left(1-\frac{\bar{I}_2}{I_2}\right)(\gamma I_1-(\psi+\mu)I_2) + \frac{\beta\bar{S}(\bar{I}_3+\phi\bar{M})}{(\delta+\mu)\bar{I}_3}\left(1-\frac{\bar{I}_3}{I_3}\right)(\psi I_2-(\delta+\mu)I_3)\\ &+\frac{\phi\beta\bar{S}\bar{M}}{\delta\bar{I}_3}\left(1-\frac{\bar{M}}{M}\right)(\delta I_3-cM). \qquad (17)\end{aligned}$$

Collecting terms of Eq. (17) and using the equilibrium point conditions of $\bar{Q}^M$:

$$\kappa-\mu\bar{S} = \beta\bar{S}(\bar{I}_1+\bar{I}_2+\bar{I}_3+\phi\bar{M}) = (\gamma+\mu)\bar{I}_1,$$
$$\gamma\bar{I}_1 = (\psi+\mu)\bar{I}_2,\ \psi\bar{I}_2 = (\delta+\mu)\bar{I}_3,\ \delta\bar{I}_3 = c\bar{M},$$

we obtain

$$\begin{aligned}\frac{dV_1}{dt} &= -(\mu+\beta\bar{I}_1)\frac{(S-\bar{S})^2}{S} + \beta\bar{S}\bar{I}_2\left(3-\frac{\bar{S}}{S}-\frac{S\bar{I}_1I_2}{\bar{S}I_1\bar{I}_2}-\frac{I_1\bar{I}_2}{\bar{I}_1I_2}\right)\\ &+\beta\bar{S}\bar{I}_3\left(4-\frac{\bar{S}}{S}-\frac{S\bar{I}_1I_3}{\bar{S}I_1\bar{I}_3}-\frac{I_1\bar{I}_2}{\bar{I}_1I_2}-\frac{I_2\bar{I}_3}{\bar{I}_2I_3}\right)\\ &+\phi\beta\bar{S}\bar{M}\left(5-\frac{\bar{S}}{S}-\frac{SM\bar{I}_1}{\bar{S}\bar{M}I_1}-\frac{I_1\bar{I}_2}{\bar{I}_1I_2}-\frac{I_2\bar{I}_3}{\bar{I}_2I_3}-\frac{\bar{M}I_3}{M\bar{I}_3}\right). \qquad (18)\end{aligned}$$

Since the arithmetical mean is greater than or equal to the geometrical mean, then, $\frac{dV_1}{dt} \le 0$ for all $S,I_i,M > 0$, $i = 1,2,3$ with equality holding when $S = \bar{S}$, $I_1 = \bar{I}_1$, $I_2 = \bar{I}_2$, $I_3 = \bar{I}_3$ and $M = \bar{M}$. Let $\Lambda_1 = \left\{(S,I_1,I_2,I_3,M) : \frac{dV_1}{dt} = 0\right\}$. It is easy to verify that $\Lambda_1' = \{\bar{Q}^M\}$ is the largest invariant subset of Λ_1 and each solution of system (9)-(13) are eventually approaches Λ_1 [37]. Noting that $R_0^M > 1$, then $\bar{Q}^M$ is globally asymptotically stable employing LaSalle's invariance principle. □

4. NUMERICAL SIMULATIONS AND DISCUSSIONS

In this section, we present computer simulation results for models (1)-(4) and (9)-(13) to illustrate numerically our theoretical results given in Sections 2 and 3. All computations are carried out by MATLAB.

4.1 Numerical Studies of Model (1)-(4)

For model (1)-(4) we consider three different initial conditions as:
IC1 : $(S(0),I_1(0),I_2(0),I_3(0)) = (300,10,10,10)$,
IC2 : $(S(0),I_1(0),I_2(0),I_3(0)) = (200,30,20,20)$,
IC3 : $(S(0),I_1(0),I_2(0),I_3(0)) = (100,60,30,30)$.

In addition, we fix some parameters of the model as $\kappa = 200$, $\gamma = 0.4$, $\psi = 0.4$ and $\mu = 0.5$. Besides, we choose two different values of β as given bellow which leads to the following cases:

(i) when $\beta = 0.001$ the threshold parameter is calculated as $R_0 = 0.8 < 1$. Fig. 3 illustrate that when $R_0 \leq 1$ the susceptible individuals $S(t)$ tend to its equilibrium value, while $I_1(t)$, $I_2(t)$ and $I_3(t)$ approach zero as time increases for initial conditions **IC1-IC3**. This means that the disease dies out. Then, the unique disease-free equilibrium $Q_0 = (400,0,0,0)$ exists and it is globally asymptotically stable and this result is compatible with Lemma 2 and Theorem 3.

(ii) when $\beta = 0.002$ the threshold parameter is given as $R_0 = 1.6 > 1$. Fig. 3 demonstrate that when $R_0 > 1$ the solutions starting from the initial conditions **IC1-IC3** tend to the endemic equilibrium $\bar{Q} = (250,83,37,30)$. It means that $\bar{Q}$ exists and it is globally asymptotically stable and this support the result of Lemma 2 and Theorem 4. In this case the disease becomes endemic.

In fact, the value of R_0 depends on the effect of the infection transmission parameter β. As β increases the threshold parameter increases as well and the stability behavior is changed. Moreover, we calculate $\beta^{critical}$ as:

$$\frac{\beta^{critical}\kappa}{\mu^2} = 1 \Longrightarrow \beta^{critical} = \frac{\mu^2}{\kappa}.$$

Using the values of parameters given above we have $\beta^{critical} = 0.00125$. Therefore, if $\beta \leq 0.00125$, then the system has only one equilibrium Q_0. Moreover, if $\beta > 0.00125$, then the system has two equilibria Q_0 and $\bar{Q}$.

4.2 Numerical Studies for Model (9)-(13)

In this subsection we only study the effect of parameter ϕ on the stability behavior of the solutions.

We choose the parameters $\kappa = 200$, $\gamma = 0.4$, $\psi = 0.4$, $\mu = 0.5$, $\delta = 0.4$, $c = 0.3$, $\beta = 0.0013$ and consider the following initial condition:

IC4 : $(S(0),I_1(0),I_2(0),I_3(0),M(0)) = (380,10,5,2.5,2.5)$.

Under this chosen values we consider the entire range of possible values for the infection rate due to mites ϕ, and its influence on R_0^M. Let $\phi^{critical}$ be calculated as:

$$R_0^M = \frac{\beta S_0\left\{c(\gamma+\mu)(\delta+\mu)+c\psi(\gamma+\delta+\mu)+\phi^{critical}\gamma\delta\psi\right\}}{c(\gamma+\mu)(\delta+\mu)(\psi+\mu)} = 1,$$

under the constraint $0 \leq \phi^{critical} \leq 1$. In fact, the value of $\phi^{critical}$ is calculated to be $\phi^{critical} = 0.337134$. From Fig. 4 we have noticed that, if $0 \leq \phi \leq \phi^{critical}$, then $R_0^M \leq 1$ and the solution trajectories tend to the equilibrium Q_0^M. Besides, if $\phi^{critical} < \phi \leq 1$, then $R_0^M > 1$ and the solution trajectories tend to the equilibrium $\bar{Q}^M$. This means that

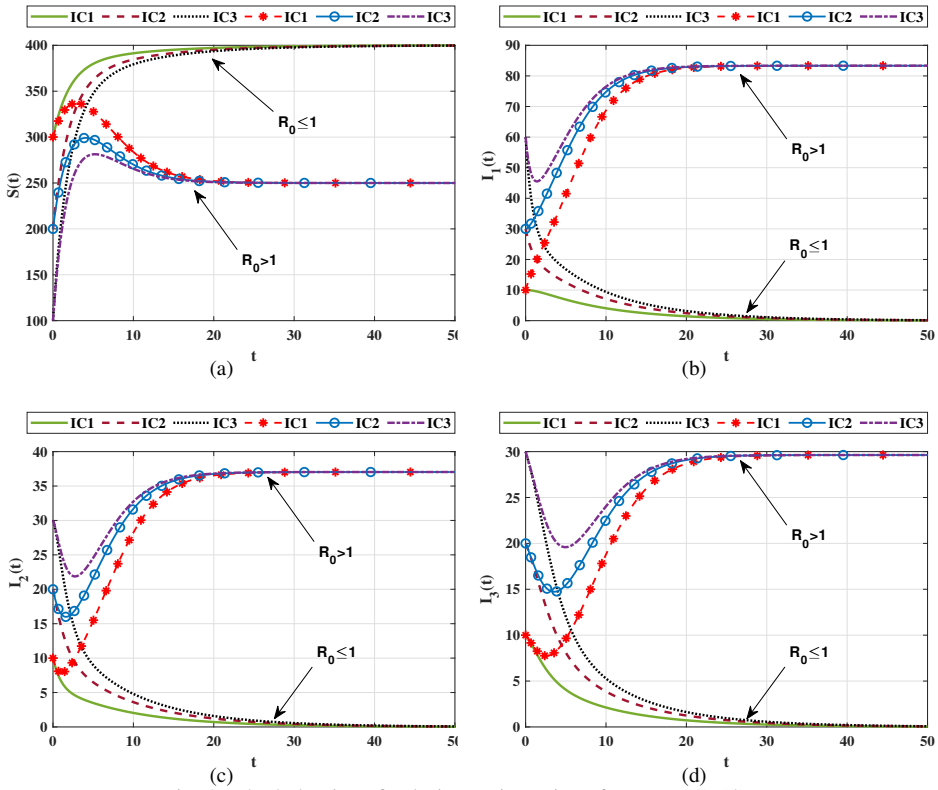

Fig. 3. The behavior of solution trajectories of system (1)-(4).

the stability behavior of all equilibria can be changed under the effect of ϕ. It is observed from Fig. 4 that, for small amount of ϕ the infectious individuals, infected individuals and number of adult mites are decreased while the susceptible individuals increase which means that the population is more immune. This gives a significant meaning that as long as the susceptible individuals starts treating sooner, the possible risk of reinfection becomes less.

5. CONCLUSION AND DISCUSSION

In this paper, we have studied mathematical models for the infection dynamics of scabies. We have considered four-dimensional system of ordinary differential equations that described the transmissions between susceptible and infectious/infective individuals. In the second model, we have included the importance of adult scabiei mite in the real interaction to hosts. We have shown that the solutions of the system are nonnegative and bounded, which ensures the well-posedness of the proposed model. For each model we have derived a threshold parameters R_0 which completely determine the existence and stability of the disease-free equilibrium Q_0 and endemic equilibrium $\bar{Q}$. The global

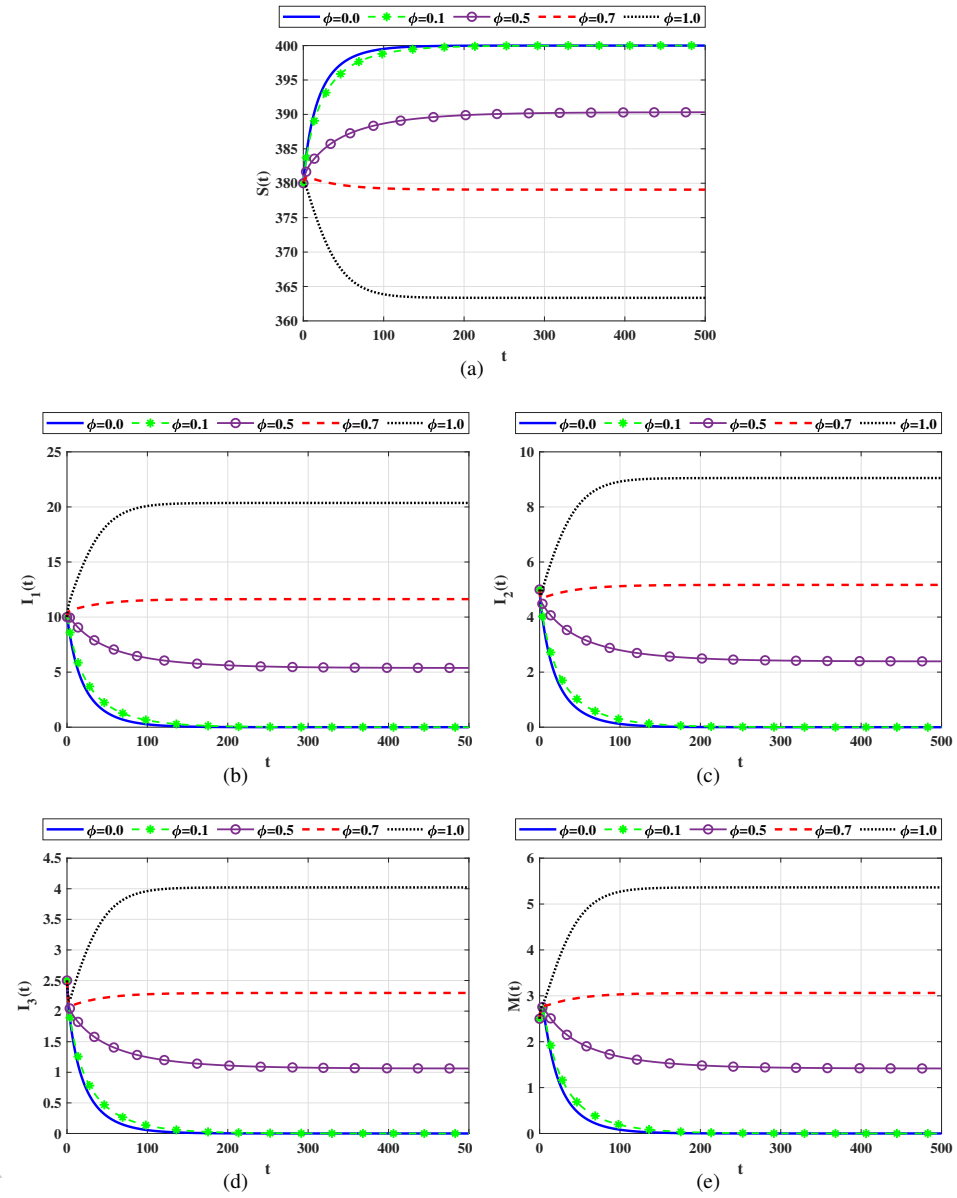

Fig. 4. The influence of the parameter ϕ on the evolution of solution trajectories of system (9)-(13).

asymptotic stability of the two equilibria has been investigated by constructing Lyapunov functions and applying LaSalle's invariance principle. We have proven that if $R_0 \leq 1$, then Q_0 is globally asymptotically stable and if $R_0 > 1$, then $\bar{Q}$ is globally asymptotically stable. To illustrate our theoretical results, we have presented conducted some numerical simulations.

We note that the exact analytical solutions of our proposed models are not known, therefore approximate solutions can only be found. Therefore, the corresponding discrete-time models of the continuous-time models (1)-(4) and (9)-(13) needs to be studied. Non-standard finite difference method is one of the discretization methods which has been widely used to discretize the continuous time models in epidemiology [38] and virology [39-41]. Further, it is commonly observed that in many biological processes, time delay is inevitable [42-47]. Therefore our models can be extended to incorporate time delay. These extensions require more investigations, therefore we leave it for future works.

REFERENCES

1. S. J. Gilmore, "Control strategies for endemic childhood scabies," *PLoS ONE*, Vol. 6, 2011, No. e15990.
2. K. Karthikean, "Treatment of scabies: newer perspectives," *Postgraduate Medical Journal*, Vol. 81, 2005, pp. 7-11.
3. M. A. Elmaraghy and A. M. El Meghawry, "Inflammatory allergic immune response in scabies pyoderma," *Journal of American Science*, Vol. 7, 2011, pp. 128-138.
4. M. J. Lydeamore, P. T. Campbell, D. G. Regan, S. Y. C. Tong, R. M. Andrews, A. C. Steer, L. Romani, J. M. Kaldor, J. McVernon, and J. M. McCaw, "A biological model of scabies infection dynamics, and treatment informs mass drug administration strategies to increase the likelihood of elimination," *Mathematical Biosciences*, Vol. 309, 2019, pp. 163-173.
5. T. Kearns, D. Clucas, C. Connors, B. J. Currie, J. R. Carapetis, and R. M. Andrews, "Clinic attendances during the first 12 months of life for Aboriginal children in five remote communities of northern Australia," *PLoS ONE*, Vol. 8, 2013, pp. 1-5.
6. A. E. Ahmed, H. Jradi, D. A. AlBuraikan, B. I. ALMuqbil, M. A. Albaijan, A. M. Al-Shehri, and H. AL-Jahdali, "Rate, and factors for scabies recurrence in children in Saudi Arabia: a retrospective study," *BMC Pediatrics*, Vol. 19, 2019, No. 187.
7. T. Kinyanjui, J. Middleton, S. Guttel, J. Cassell, J. Ross, and T. House, "Scabies in residential care homes: Modelling, inference, and interventions for well-connected population sub-units," *PLOS Computational Biology*, Vol. 14, 2018, No. e1006046.
8. K. Mellanby, "Transmission of scabies," *British Medical Journal*, Vol. 2, 1941, pp. 405-406.
9. D. Engelman and A. C. Steer, "Control strategies for scabies," *Tropical Medicine, and Infectious Disease*, Vol. 3, 2018, No. 98.
10. University of Sydney, "Scabies," http://medent.usyd.edu.au/fact/scabies.html.
11. L. G. Arlian and D. L. Vyszenski-Moher, "Life cycle of sarcoptes scabiei var. canis," *The Journal of Parasitology*, Vol. 74, 1988, pp. 427-430.
12. B. J. Currie and J. S. McCarthy, "Permethrin and ivermectin for scabies," *The New England Journal of Medicine*, Vol. 362, 2010, pp. 717-725.

13. S. F. Walton, D. C. Holt, B. J. Currie, and D. J. Kemp, *Scabies: New Future for a Neglected Disease*, Vol. 57, 2004, pp. 309-376.
14. K. Mellanby, "The development of symptoms, parasitic infection, and immunity in human scabies," *Parasitology*, Vol. 35, 1944, pp. 197-206.
15. L. Esteva and C. Vargas, "A model for dengue disease with variable human population," *Journal of Mathematical Biology*, Vol. 1999, 1999, pp. 220-240.
16. N. Chitnis, J. M. Cushing, and J. M. Hyman, "Bifurcation analysis of a mathematical model for malaria transmission," *SIAM Journal of Applied Mathematics*, Vol. 67, 2006, pp. 24-45.
17. S. M. Raimundo, M. Amaku, and E. Massad, "Equilibrium analysis of a yellow fever dynamical model with vaccination," *Computational and Mathematical Methods in Medicine*, Vol. 2015, 2015, No. 482091.
18. E. Dantas, M. Tosin, and A. Cunha Jr, "Calibration of a SEIR-SEI epidemic model to describe the Zika virus outbreak in Brazil," *Applied Mathematics, and Computation*, Vol. 338, 2018, pp. 249-259.
19. J. P. Tian and J. Wang, "Global stability for cholera epidemic models," *Mathematical Biosciences*, Vol. 232, 2011, pp. 31-41.
20. C. P. Bhunu, S. Mushayabasa, and T. G. Monera, "Assessing the impact of vaccination on controlling the spread of human scabies," *ISRN Computational Biology*," Vol. 2013, 2013, No. 362973.
21. C. V. de León, "On the global stability of infectious disease models with relapse," *Abstraction & Application*, Vol. 9, 2013, pp. 50-61.
22. E. Beretta and V. Capasso, "On the general structure of epidemic systems. Global asymptotic stability," *Computers & Mathematics with Applications, Part A*, Vol. 12, 1986, pp. 677-694.
23. C. Vargas-De-Leon, "On the global stability of SIS, SIR, and SIRS epidemic models with standard incidence," *Chaos, Solitons & Fractals*, Vol. 44, 2011, pp. 1106-1110.
24. A. Korobeinikov and G. C. Wake, "Lyapunov functions, and global stability for SIR, SIRS, and SIS epidemiological models," *Applied Mathematics Letters*, Vol. 15, 2002, pp. 955-960.
25. A. Korobeinikov, "Global properties of basic virus dynamics models," *Bulletin of Mathematical Biology*, Vol. 66, 2004, pp. 879-883.
26. A. M. Elaiw and N. H. AlShamrani, "Stability of an adaptive immunity pathogen dynamics model with latency and multiple delays," *Mathematical Methods in the Applied Science*, Vol. 36, 2018, pp. 125-142.
27. A. M. Elaiw, E. Kh. Elnahary, and A. A. Raezah, "Effect of cellular reservoirs, and delays on the global dynamics of HIV," *Advances in Difference Equations*, Vol. 2018, 2018, No. 85.
28. A. M. Elaiw, A. A. Raezah, and S. A. Azoz, "Stability of delayed HIV dynamics models with two latent reservoirs and immune impairment," *Advances in Difference Equations*, Vol. 2018, 2018, No. 414.
29. A. M. Elaiw and N. H. AlShamrani, "Stability of a general delay-distributed virus dynamics model with multi-staged infected progression, and immune response," *Mathematical Methods in the Applied Sciences*, Vol. 40, 2017, pp. 699-719.

30. A. M. Elaiw and A. A. Raezah, "Stability of general virus dynamics models with both cellular, and viral infections and delays," *Mathematical Methods in the Applied Sciences*, Vol. 40, 2017, pp. 5863-5880.
31. A. M. Elaiw and N. A. Almuallem, "Global dynamics of delay-distributed HIV infection models with differential drug efficacy in cocirculating target cells," *Mathematical Methods in the Applied Sciences*, Vol. 39, 2016, pp. 4-31.
32. A. M. Elaiw and N. H. AlShamrani, "Global properties of nonlinear humoral immunity viral infection models," *International Journal of Biomathematics*, Vol. 8, 2015, Article ID 1550058.
33. A. D. Hobiny, A. M. Elaiw, and A. Almatrafi, "Stability of delayed pathogen dynamics models with latency, and two routes of infection," *Advances in Difference Equations*, Vol. 2018, 2018, No. 276.
34. A. M. Elaiw and A. D. AlAgha, "Global dynamics of reaction-diffusion oncolytic M1 virotherapy with immune response," *Applied Mathematics, and Computation*, Vol. 367, 2020, No. 124758.
35. A. M. Elaiw and A. D. AlAgha, "Global dynamics of a general diffusive HBV infection model with capsids, and adaptive immune response," *Advances in Difference Equations*, Vol. 2019, 2019, No. 519.
36. A. M. Elaiw and N. H. AlShamrani, "Global stability of humoral immunity virus dynamics models with nonlinear infection rate, and removal," *Nonlinear Analysis: Real World Applications*, Vol. 26, 2015, pp. 161-190.
37. J. K. Hale and S. V. Lunel, *Introduction to Functional Differential Equations*, Springer-Verlag, NY, 1993.
38. D. Ding, W. Qin, and X. Ding, "Lyapunov functions, and global stability for a discretized multi-group SIR epidemic model," *Discrete, and Continuous Dynamical Systems – Series B*, Vol. 20, 2015, pp. 1971-1981.
39. A. M. Elaiw and M. A. Alshaikh, "Stability of a discrete-time general delayed viral model with antibody, and cell-mediated immune responses," *Advances in Difference Equations*, Vol. 2020, 2020, p. 54.
40. A. M. Elaiw and M. A. Alshaikh, "Stability analysis of a general discrete-time pathogen infection model with humoral immunity," *Journal of Difference Equations, and Applications*, Vol. 25, 2019, pp. 1149-1172.
41. A. M. Elaiw and M. A. Alshaikh, "Stability of discrete-time HIV dynamics models with three categories of infected CD4, T-cells," *Advances in Difference Equations*, Vol. 2019, 2019, No. 407.
42. N. M. Dixit, M. Markowitz, D. D. Ho, and A. S. Perelson, "Estimates of intracellular delay, and average drug efficacy from viral load data of HIV-infected individuals under antiretroviral therapy," *Antiviral Therapy*, Vol. 9, 2004, pp. 237-246.
43. A. M. Elaiw and E. Kh. Elnahary, "Analysis of general humoral immunity HIV dynamics model with HAART, and distributed delays," *Mathematics*, Vol. 7, 2019, No. 157.
44. A. M. Elaiw, A. Almatrafi, A. D. Hobiny, and K. Hattaf, "Global properties of a general latent pathogen dynamics model with delayed pathogenic, and cellular infections," *Discrete Dynamics in Nature and Society*, Vol. 2019, 2019, No. 9585497.

45. A. M. Elaiw, S. F. Alshehaiween, and A. D. Hobiny, "Global properties of delay-distributed HIV dynamics model including impairment of B-cell functions," *Mathematics*, Vol. 7, 2019, No. 837.
46. A. M. Elaiw and A. D. AlAgha, "Analysis of a delayed, and diffusive oncolytic M1 virotherapy model with immune response," *Nonlinear Analysis: Real World Applications*, Vol. 55, 2020, No. 103116.
47. A. M. Elaiw and N. H. AlShamrani, "Stability of a general adaptive immunity virus dynamics model with multi-stages of infected cells, and two routes of infection," *Mathematical Methods in the Applied Sciences*, 2019, No. 5923.

Noura H. AlShamrani received her MS of Science in Mathematics from King AbdulAziz University, Saudi Arabia 2014-2015. Currently, she is a Ph.D. student in Applied Mathematics (Biomathematics) at KAU. She works as a Lecturer at the Department of Mathematics, Faculty of Science, University of Jeddah, Saudi Arabia. In 2017, she received award of best graduate student from the Deanship of Scientific Research at King Abdulaziz University. She is passionate about scientific research in mathematical modelling in biology, epidemiology and ecology.

Ahmed M. Elaiw graduated in Mathematics at Sohag University in 1992. He received his Ph.D. degree in stabilization of sampled-data nonlinear systems form Budapest University of Technology and Economics in 2005. Since 2005, he has been with Al-Azhar University, Assiut, Egypt. Currently he is a Professor at Department of Mathematics, Faculty of Science, King Abdulaziz University, Saudi Arabia. His research interests include nonlinear control systems, power systems, optimization, mathematical biology and fluid dynamics.

Hanan A. Batarfi is a Saudi academic and researcher based in the Mathematics Department (Women's Section) at King Abdulaziz University, Jeddah. Having achieved Bachelor's and Master's degrees from KAU, she completed a Ph.D. in Applied Mathematics at UMIST, Manchester, United Kingdom. Following her studies in the UK, Dr. Hanan returned to KAU where in addition to teaching both undergraduate and postgraduate courses, she was also involved in their development introducing new initiatives utilising digital technology and supporting independent learning styles. Dr. Hanan has also been involved in the support of female Saudi Ph.D. students as part of a Joint Supervision program with universities in the UK. Immensely proud of her Saudi heritage, Dr. Hanan has been a pioneer in organising guided tours of Jeddah's Historical Area for visitors to her university and others. In addition to her professional life, Dr. Hanan plays a major role within her family and also enjoys cooking, travel and art. She can be considered as the foremost Middle Eastern authority on Sticky Toffee Pudding.

Aatef D. Hobiny obtained his Ph.D. degree in Mathematics from Heriot-Watt University in 2013. He is an Associate Professor in the Department of Mathematics at King Abdulaziz University. He is a member of NAAM "nonlinear analysis and applied mathematics" research group.