Nonlinear Analysis: Modelling and Control
ISSN: 1392-5113
ISSN: 2335-8963
nonlinear@mii.vu.lt
Vilniaus Universitetas
Lituania

Transmission dynamic and backward bifurcation of Middle Eastern respiratory syndrome coronavirus

Fatima, Bibi; Zaman, Gul; Jarad, Fahd
Transmission dynamic and backward bifurcation of Middle Eastern respiratory syndrome coronavirus
Nonlinear Analysis: Modelling and Control, vol. 27, núm. 1, 2022
Vilniaus Universitetas, Lituania
Disponible en: https://www.redalyc.org/articulo.oa?id=694173132004

Transmission dynamic and backward bifurcation of Middle Eastern respiratory syndrome coronavirus

Bibi Fatima fatma.uom@gmail.com
Department of Mathematics, University of Malakand Chakdara, Dir Lower, Khyber Pakhtunkhwa, Pakistán
Gul Zaman gzaman@uom.edu.pk
Department of Mathematics, Çankaya University, Etimesgut, Turquía
Fahd Jarad fahd@cankaya.edu.tr
Department of Medical Research, China Medical University Hospita, China

Nonlinear Analysis: Modelling and Control, vol. 27, núm. 1, 2022

Vilniaus Universitetas, Lituania

Recepción: 14 Agosto 2020
Publicación: 01 Enero 2022

Redalyc: https://www.redalyc.org/articulo.oa?id=694173132004

Abstract: Middle East respiratory syndrome coronavirus (MERS-CoV) remains an emerging disease threat with regular human cases on the Arabian Peninsula driven by recurring camels to human transmission events. In this paper, we present a new deterministic model for the transmission dynamics of (MERS-CoV). In order to do this, we develop a model formulation and analyze the stability of the proposed model. The stability conditions are obtained in term of R_0 , we find those conditions for which the model become stable. We discuss basic reproductive number R_0 along with sensitivity analysis to show the impact of every epidemic parameter. We show that the proposed model exhibits the phenomena of backward bifurcation. Finally, we show the numerical simulation of our proposed model for supporting our analytical work. The aim of this work is to show via mathematical model the transmission of MERS-CoV between humans and camels, which are suspected to be the primary source of infection.

Keywords: epidemic model, reproductive number, stability analysis, backward bifurcation, numerical simulation.

1 Introduction

A new coronavirus was identified in Saudi Arabia in September 2012 known as Middle Eastern respiratory syndrome coronavirus (MERS-CoV) [5, 11]. MERS-CoV is associ- ated with an animal source in the Middle East. Besides human, MERS-CoV has been found in camel in several countries [1]. Since its emergence in 2012, the Middle East respiratory coronavirus (MERS-CoV) has caused spill over from the dromedary camel population into the human population. This virus also spread from an infected person's respiratory secretion such as through coughing. MERS-CoV has spread from ill people to others through closed contacts such as caring for or living with an infected person [3]. Since April 2012 till date, there have been a total of 536 cases with 145 deaths, a case fa- tality rate of 27 percent with the majority being reported in the Middle East (Saudi Arabia, Jordan, and Qatar) [2]. For the forecast of dynamics of infectious diseases, see [8, 10, 18]. One of the largest outbreaks of MERS-CoV has been described by Assire et al. [4] with the description that the virus is transmissible from human to human.

Zumla et al. [19] pointed out in a review article that the reason for the camel to human transmission could be the indirect exposure, e.g., it was possible that the patient's exposure to MERS-CoV was consumption of unpasteurized camel milk, which is very common practice in Saudi Arabia.

Poletto et al. [15] believed that peoples movement and maxing during Hajj and Umrah were mainly responsible for MERS-CoV transmission. Besides, camel racing, closing and the opening again of camel market along with climatic factors could have an impact on the transmission of MERS-CoV from camels to humans and then among humans.

In this paper, we take the human and camel population. We construct a compartmental model for the transmission dynamics of MERS-CoV. The model is consisting of human population, that is: susceptible human S_h , exposed or latent human E_h , symptomatic and infectious human I_h , infectious but asymptomatic class human A_h , hospitalized class H_h , and recovery class human R_h . Camel population, which consists of susceptible camel S_c , asymptomatic infected camel X_c , and symptomatic infected camel Y_c . We analyze the stability of the proposed model. The stability conditions are obtained in terms of basic reproductive number. To find the transmission potential of diseases, we investigate a formula for the basic reproduction number of camel to human population and from human to human population by using next-generation matrix method. For local stability of the proposed model (1), we use Routh–Hurwitz criteria. When the basic reproductive number is less than one, the disease-free equilibrium of the model is locally asymptotically stable, therefore, the disease dies out after some period of time. While when the basic reproduction number is greater than one, the disease will prevail and persist in the population. We also investigate the model for global stability by using Lyapunov function theory. Sensitivity analysis was carried out on the model parameters to analyze their impact on disease transmission. The backward bifurcation in a disease model has an important qualitative implications. We find backward bifurcation and endemic equilibria. Numerical simulation of the proposed model (1) was carried out, and the results are displayed.

This article is arranged as follows. Section 2 represents the mathematical construction of epidemic model. In Section 3, we show the positivity and boundedness of the proposed model. In Section 4, we analyzed the stability of the proposed model. Equilibria and basic reproductive number of model (1) are presented in Sections of 4.1 and 4.2. Section 5 deal with backward bifurcation and endemic equilibria. In Section 6, we present the global asymptotic stability of endemic equilibrium by using Lyapunov function theory. Numerical simulation results of the proposed model are presented in Section 7.

2 Model formulation

In this section, we develop a compartmental epidemic model of Middle Eastern respira- tory syndrome coronavirus (MERS-CoV). According

to biological characteristics of the MERS-CoV, we divide the total population into human and camel populations. $S_h(t)$ is susceptible human, $E_h(t)$ is the exposed human, $I_h(t)$ is symptomatic and infectious hu- man, infectious but asymptotic class human $A_h(t)$, hospitalized human $H_h(t)$, recovery class $R_h(t)$, $S_c(t)$ is susceptible camel, $X_c(t)$ is asymptomatic infected camel, and $Y_c(t)$ is symptomatic infected camel. Keeping the characteristic of Middle Eastern respiratory syndrome coronavirus (MERS-CoV) along with the above characterization leads to the following system of ordinary differential equations:

$$\begin{aligned}
\frac{\mathrm{d}S_h}{\mathrm{d}t} &= b_h - \frac{\beta_1 X_c + \beta_2 Y_c + \beta_3 I_h + \beta_4 q H_h}{N_h} S_h - \mu_0 S_h, \\
\frac{\mathrm{d}E_h}{\mathrm{d}t} &= \frac{\beta_1 X_c + \beta_2 Y_c + \beta_3 I_h + \beta_4 q H_h}{N_h} S_h - (\gamma + \mu_0) E_h, \\
\frac{\mathrm{d}I_h}{\mathrm{d}t} &= \rho\gamma E_h - (\phi_a + \phi_1) I_h - (\mu_0 + \mu_1) I_h, \\
\frac{\mathrm{d}A_h}{\mathrm{d}t} &= \gamma(1-\rho) E_h - (\mu_0 + \mu_2) A_h, \\
\frac{\mathrm{d}H_h}{\mathrm{d}t} &= \phi_a I_h - \phi_\phi H_h - \mu_0 H_h, \\
\frac{\mathrm{d}R_h}{\mathrm{d}t} &= \phi_1 I_h + \phi_\phi H_h - \mu_0 R_h, \\
\frac{\mathrm{d}Sc}{\mathrm{d}t} &= b_c - \frac{\beta_5 X_c + \beta_6 Y_c}{N_c} S_c - k_1 S_c, \\
\frac{\mathrm{d}X_c}{\mathrm{d}t} &= \frac{\beta_5 X_c + \beta_6 Y_c}{N_c} S_c - (k_2 + \gamma_c) X_c, \\
\frac{\mathrm{d}Y_c}{\mathrm{d}t} &= \gamma_c X_c - (k_3 + \alpha_c) Y_c
\end{aligned} \tag{1}$$

with initial size of population

$$\begin{gathered}
S_h(0) > 0, \qquad E_h(0) \geqslant 0, \qquad I_h(0) \geqslant 0, \qquad A_h(0) \geqslant 0, \qquad H_h(0) \geqslant 0, \\
R_h(0) \geqslant 0, \qquad S_c(0) \geqslant 0, \qquad X_c(0) \geqslant 0, \qquad Y_c(0) \geqslant 0.
\end{gathered}$$

Here b_h is the birth rate of human population, β_1, β_2, β_3, β_4, β_5, β_6 show the transmission rate per unit time, q show the approximate transmission rate of hospitalized patient. The rate at which individuals leave the exposed class by becoming infectious is γ. ρ is the proportion of progression from exposed class $E_h(t)$ to symptomatic infectious class $I_h(t)$, 1 ρ is that of progression to asymptotic class $A_h(t)$, ϕa is the average rate at which symptomatic individuals hospitalize, and ϕ1 is the recovery rate without being hospitalized. ϕ_ϕ is the recovery rate of the hospitalized

patient. μ_0 is the natural death rate, μ_1, μ_2 are death rate due to MERS-CoV.

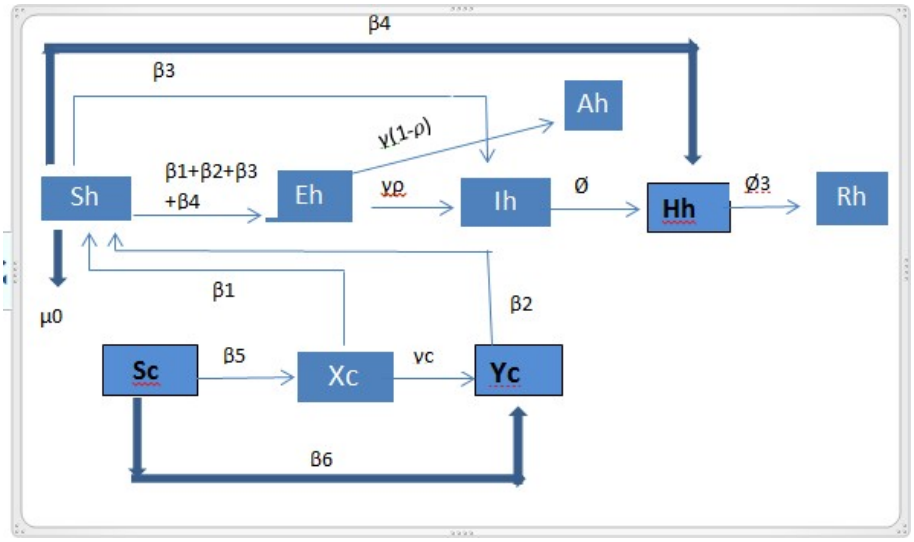


Figure 1
Flow chart for the transmission of MERS-CoV between camel and human population.

he camel population is represented as *SI* model, where b_c is the birth rate of camel population, S_c represents susceptible camels, X_c represents asymptomatic infected camels, and Y_c represents symptomatic camels. γ_c is the moving rate from asymptomatic camel to symptomatic camel population. k_1, k_2, k_3 are the natural death rate of susceptible, asymptomatic camel, and symptomatic infected camels. α_c is the death rate due to MERS- CoV. N_h and N_c are human and camel populations, respectively.

3 Positivity and boundedness

Lemma 1. *All the variables given in model* (1) *are positive and bounded.*

Proof. The positivity follow from the standard argument [16] with which we can show that if $R^{10} = (s_1, s_2, \ldots, s_n) \in R^{10}_+$, $s_i \geqslant 0$, for all $i \in 1, \ldots, n$, then R^{10}_+ is positively invariant under the flow induced by model (1).

For boundedness, we call total human population in model (1) by $N_h(t)$ and camel population by $N_c(t)$. *Let* $N_h(t)$ represents the total human population at time t, i.e.,

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + A_h(t) + H_h(t) + R_h(t).$$

The time differentiation of $N_h(t)$ and the use of equations in model (1) give

$$\begin{aligned}\frac{\mathrm{d}N_h}{\mathrm{d}t} &= b_h - \mu_0 S_h - \mu_0 E_h - (\mu_0 + \mu_1) I_h - (\mu_0 + \mu_2) A_h - \mu_0 H_h - \mu_0 R_h \\ &\leqslant b_h - \mu_0 N_h.\end{aligned}$$

This means that there exists $\limsup_{t\to\infty} b_h/\mu_0$. Let $N_c(t)$ represents the total camels population at time t,

$$N_c(t) = S_c(t) + X_c(t) + Y_c(t),$$

$$\frac{\mathrm{d}N_c}{\mathrm{d}t} = b_c - k_1 S_c - k_2 X_c - k_3 Y_c - \alpha_c Y_c. \leqslant b_c - k_1 N_c.$$

This means that there exists $\limsup_{t\to\infty} b_c/k_1$.

From the above results it follows that $S_h(t), E_h(t), I_h(t), A_h(t), H_h(t), R_h(t), S_c(t), X_c(t), Y_c(t)$ are bounded on their maximal domain. Therefore, for biological feasibility, we study (1) in the closed set

$$\Omega = \left\{ (S_h, E_h, I_h, A_h, H_h, R_h, S_c, X_c, Y_c) \in R_+^9 : \right.$$
$$\left. 0 < S_h + E_h + I_h + A_h + H_h + R_h + S_c + X_c + Y_c \leqslant \frac{b_h}{\mu_0} + \frac{b_c}{k_1} \right\}.$$

The Jacobian of model (1) is given by

$$J_0 = \begin{pmatrix} -A_2 & 0 & -A_3 & 0 & -A_4 & 0 & 0 & -A_5 & -A_6 \\ B_2 & -B_3 & B_4 & 0 & B_5 & 0 & 0 & B_6 & B_7 \\ 0 & C_1 & -C_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & C_3 & 0 & -C_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi_a & 0 & -D_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi_1 & 0 & \phi_\phi & -\mu_0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -D_2 & D_3 & -D_4 \\ 0 & 0 & 0 & 0 & 0 & 0 & D_5 & E_2 & \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_c & -E_3 \end{pmatrix} \quad [2]$$

Here

$$A_2 = -\frac{\beta_1 X_c + \beta_2 Y_c + \beta_3 I_h + \beta_4 q H_h}{N_h} - \mu_0, \quad A_3 = \frac{\beta_3 S_h}{N_h}, \quad A_4 = -\frac{\beta_4 q S_h}{N_h},$$
$$A_5 = \frac{\beta_1 S_h}{N_h}, \quad A_6 = \frac{\beta_2 S_h}{N_h}, \quad B_2 = \frac{\beta_1 X_c + \beta_2 Y_c + \beta_3 I_h + \beta_4 q H_h}{N_h},$$
$$B_3 = \gamma + \mu_0, \quad B_4 = \frac{\beta_3 S_h}{N_h}, \quad B_5 = \frac{\beta_4 q S_h}{N_h}, \quad B_6 = \frac{\beta_1 S_h}{N_h},$$
$$B_7 = \frac{\beta_2 S_h}{N_h}, \quad C_1 = \rho\gamma, \quad C_2 = -(\phi_a + \phi_1 + \mu_0 + \mu_1), \quad C_3 = \gamma(1-\rho),$$
$$C_4 = \mu_0 + \mu_2, \quad D_1 = \phi_\phi + \mu_0, \quad D_2 = -\frac{\beta_5 X_c + \beta_6 Y_c + \beta_7 I_h - k_3}{N_c},$$
$$D_3 = -\frac{\beta_5 S_c}{N_c}, \quad D_4 = \frac{\beta_6 S_c}{N_c}, \quad D_5 = \frac{\beta_5 X_c + \beta_6 Y_c + \beta_7 I_h - k_3}{N_c},$$
$$E_2 = \frac{\beta_5 S_c}{N_c} - (k_3 + \alpha_c) - \frac{\beta_6 S_c}{N_c}, \quad E_3 = k_3 + \alpha_c.$$

4 Stability analysis

We investigate the positivity and boundedness of the proposed model. We then study the qualitative behaviour of the proposed model (1). For this, first, we find equilibria and the threshold quantity R_0.

4.1 Equilibrium analysis and threshold quantity

For disease-free equilibria, we set all the variables and rate of change equal to zero except $S_h = S_{h0}, S_c = S_{c0}$., the disease-free equilibrium of the proposed model (1) become $d_0 = (b_h/\mu_0, 0, 0, 0, 0, 0, b_c/k_1, 0, 0)$. The dynamic of this equilibrium will be discuss with the help of linear stability analysis theory. On the basis of stability theory, we find those condition for which the model become stable and disease spreading will be under control. We state the dynamic of the proposed model around disease-free equilibrium with the help of the following result.

The disease-free equilibrium point $(b_h/\mu_0, 0, 0, 0, 0, 0, b_c/k_1, 0, 0)$, is lo- cally stable if $R_0 < 1$, otherwise, the disease-free equilibrium is unstable when $R_0 > 1$.

n epidemiology the basic reproduction ratio of an infection can be thought of as the expected number of cases directly generated by one case in a population when all the population are susceptible to infection. We use next-generation matrix method to find the basic reproductive number [6]. In Eq. (3) the matrices $\bar{F}$ and $\bar{V}$ contain the nonlinear and linear terms, respectively, that is,

$$\bar{F} = \begin{pmatrix} \frac{(\beta_1 X_c + \beta_2 Y_c + \beta_3 I_h + \beta_4 q H_h) S_h}{N_h} \\ 0 \\ 0 \\ \frac{(\beta_5 X_c + \beta_6 Y_c)}{N_c} S_c \\ 0 \end{pmatrix},$$

$$\bar{V} = \begin{pmatrix} (\gamma + \mu_0) E_h \\ \rho\gamma E_h - (\phi_a + \phi_1) I_h - (\mu_0 + \mu_1) I_h \\ \phi_a I_h - \phi_\phi H_h - \mu_0 H_h \\ (k_2 + \gamma_c) X_c \\ \gamma_c X_c - (k_3 + \alpha_c) Y_c \end{pmatrix}. \quad [3]$$

The Jacobian matrices of $\bar{F}$ and $\bar{V}$ at disease-free equilibrium d_0 are the following:

$$F = \begin{pmatrix} 0 & \frac{\beta_3 S_h}{N_h} & \frac{\beta_4 q S_h}{N_h} & \frac{\beta_1 S_h}{N_h} & \frac{\beta_2 S_h}{N_h} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta_5 S_c}{N_c} & \frac{\beta_6 S_c}{N_c} \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} M_1 & 0 & 0 & 0 & 0 \\ -M_2 & M_3 & 0 & 0 & 0 \\ 0 & -\phi_a & M_4 & 0 & 0 \\ 0 & 0 & 0 & M_5 & 0 \\ 0 & 0 & 0 & -\gamma_c & M_6 \end{pmatrix},$$

where $M_1 = \gamma + \mu_0$, $M_2 = \rho\gamma$, $M_3 = (\phi_a + \phi_1) + (\mu_0 + \mu_1)$, $M_4 = \phi_\phi + \mu_0$, and $M_5 = k_2 + \gamma_c$, $M_6 = k_3 + \alpha_c$.

The basic reproduction number R0 is therefore the spectral radius of next-generation matrix $\bar{H} = FV^{-1}$. $R_0 = R_h + R_c$., where

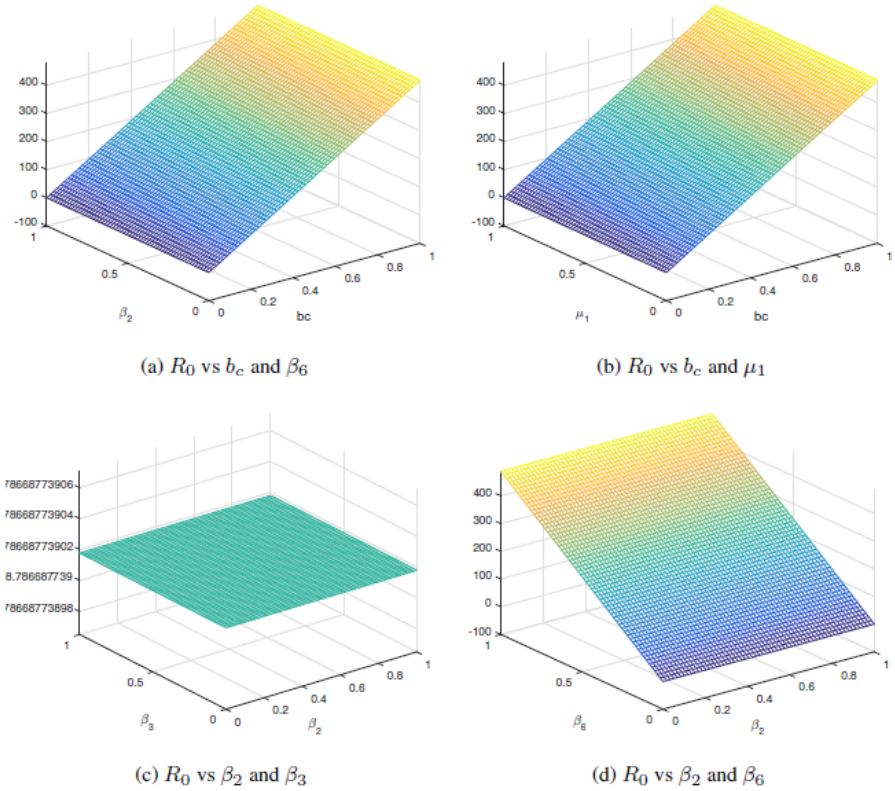
$$R_h = \frac{\gamma\beta_2\rho S_h}{N_h(\gamma+\mu_0)(\phi_a+\phi_1+\mu_0+\mu_1)} + \frac{\gamma\phi_a\beta_3 q\rho S_h}{N_h(\phi_1+\mu_0)(\phi_a+\mu_0)(\phi_1+\phi_\phi+\mu_0+\mu_1)}$$

$$R_c = \frac{\beta_6 S_c}{N_c(k_3+\alpha_c)}.$$

4.2 Sensitivity analysis

We present analysis of the sensitivity of a few parameters using in the proposed model. This make it easier for us to know the parameters that have a significant effect on repro- ductive number. We apply the technic given in [9]. Sensitivity index of basic reproductive number R_0 is given by $\Delta_h^{R_0} = \partial R_0/\partial h(h/R_0)$, where h is parameter.

Both Figs. 2 and 3 show the sensitivity of R_0. They show the importance of different parameters in the transmission of disease and also allow to measure the change in the



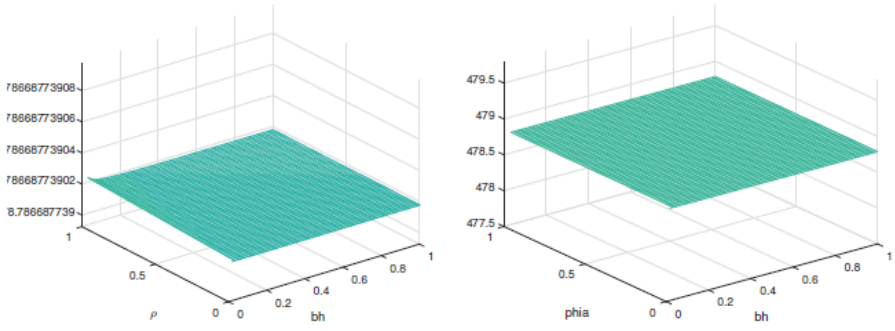
(a) R_0 vs b_c and β_6

(b) R_0 vs b_c and μ_1

(c) R_0 vs β_2 and β_3

(d) R_0 vs β_2 and β_6

Figure 2
The graphs show the variation of different parameters and its effect on the basic reproductive number.



(a) R_0 vs bh and ρ

(b) R_0 vs bh and ϕ_a

Figure 3
The graphs show the variation of different parameters and its effect on the basic reproductive number.

Parameter	Sensitivity indices	Parameter	Sensitivity indices
α_c	−0.8091519	β_2	0.002044
γ	0.0014879	ρ	0.00204588
μ_0	−0.0025667	μ_1	−0.0001346
b_h	0.0820448	b_c	0.9979541
ϕ_1	−0.000124034	ϕ_a	0.0023932
N_h	−1.1975449	N_c	−0.00204588
ϕ_ϕ	−0.0095502	β_3	0.00010744
N_h	−1.1975449	N_c	−0.00204588
κ_3	−0.1888021	β_6	0.4975411

Table 1
Values of parameters obtained in the sensitivity analysis.

reproduction number with the change in a parameter. Using these indices, we find the parameters that highly affect the reproduction number (see Table 1).

5 Endemic equilibria and backward bifurcation

We will find the endemic equilibria of system (1), where at least one of the infected component is non zero. Let $D_1 = (S_h^*, E_h^*, I_h^*, A_h^*, H_h^*, R_h^*, X_c^*, Y_c^*, S_c^*)$ represent any endemic equilibrium point. Solving equations of (1) at steady state gives

$$S_h^* = \frac{\gamma_c\beta_5(Q_1 + b\beta_6\gamma_c + Q_2^2Q_1\beta_5)I_h^*}{\beta_1Q_1Q_3 + \beta_2Q_3(1-R_0)}, \qquad I_h^* = \frac{Q_1Q_3(\beta_4\gamma_c\beta_6\gamma_c)(1-R_{0h})}{\gamma_cb\beta_5 + Q_1\beta_6\gamma_c},$$

$$E_h^* = \frac{\gamma_c\beta_5(Q_3 + \beta_6\gamma_c + Q_2^2Q_1\beta_5 + Q_2Q_1\beta_6\gamma_c)I_h^*}{(\gamma+\mu_0)(\beta_1Q_1Q_3 + \beta_2Q_3 + \beta_4Q_2\phi_a)(Q_1 + \beta_6\gamma_cQ_2^2Q_3\beta_5 - Q_2Q_3\beta_6\gamma_c)},$$

$$A_h^* = \frac{\gamma(1-\rho)(\gamma_c\beta_5)(Q_1+\beta_6\gamma_c)I_h^*}{(\mu_0+\mu_2)(\gamma+\mu_0)(\beta_1Q_1Q_3 + \beta_2Q_3 + \beta_4Q_3\phi_a)(1-R_{0h})},$$

$$H_h^* = \frac{\phi_aI_h^*}{\phi_\phi+\mu_0}, \qquad R_h^* = \frac{\phi_1(\phi_\phi+\mu_0) + \phi_a\phi_\phi)I_h^*}{\mu_0(\phi_\phi+\mu_0)},$$

$$X_c^* = \frac{Q_1Q_2(\beta_5\gamma_cb\beta_6\gamma_c)I_h^*}{\gamma_cb\beta_5 + (1-R_{0c})}, \qquad Y_c^* = \frac{\gamma_cQ_1Q_2(\beta_5\gamma_c\beta_4\gamma_c)I_h^*}{Q_1(\gamma_c\beta_5Q_1 + b\beta_6\gamma_c + Q_2Q_1\beta_5Q_2)},$$

$$S_c^* = \frac{bN_c(\beta_1Q_3Q_3 + \beta_2Q_3 + \beta_4Q_2\phi_a)(1-R_{0c})}{b\gamma_c\beta_5(Q_3+b\beta)},$$

where $Q_1 = k_3 + \alpha_c$, $Q_2 = k_2 + \alpha_c$, $Q_3 = (Q_2Q_3\beta_6\gamma_c - \beta_6\gamma_c)I_h^*$. The significance of backward bifurcation in the epidemiological model is that the classical requirement of the basic reproduction number R_0 should be less than one [13], while it is necessary for the elimination of the MERS-CoV virus from the population. The presence of backward bifurcation in the proposed model suggests that the feasibility of MERS virus elimination when the basic reproduction number is less than one depends on the initial size of the subpopulation of the model.

Now $I_h \neq 0$, then putting $S_h^*, X_c^*, Y_c^*, H_h^*$ in the first equation of model (1) at steady state and using of simple algebraic manipulation, we obtain the equation

$$f(I_h) = aI_h^2 + bI_h + c = 0. \quad [4]$$

In Eq. (4), *a*, *b*, and *c* are defined as

$$\begin{aligned} a &= b\gamma_c\beta_5 QQ_2Q_3 + Q_3^2\beta_2 + b\beta_6\gamma_c Q_3^2, \\ b &= b\beta_1 Q_2 Q_3^2(1 - R_0)\beta_6 \\ c &= b\beta_6\gamma_c Q_3^2 + \beta_3 qQ_1Q_3. \end{aligned} \quad [5]$$

In Eq. (5), $Q = N_h(\gamma + \mu_0)(\phi_a + \phi_1 + \mu_0 + \mu_1) + N_c(k_3 + \alpha_c)$. Clearly, the coefficient a is always positive, while *b* is positive or negative depend on the value of R_0. As $a > 0$, the existence of positive solution of Eq. (4) depend on the sign of *b*, *c*, which shows that the equilibrium depend continuously on the basic reproductive number. For $b^2 < 4ac$, Eq. (4) has no positive solutions, and there is no endemic equilibrium.

For $R_0 = 1$, the following result holds.

Lemma 2. *If R. = 1, model (1) posses the phenomena of backward bifurcation when c < 0; (see Fig. 4).*

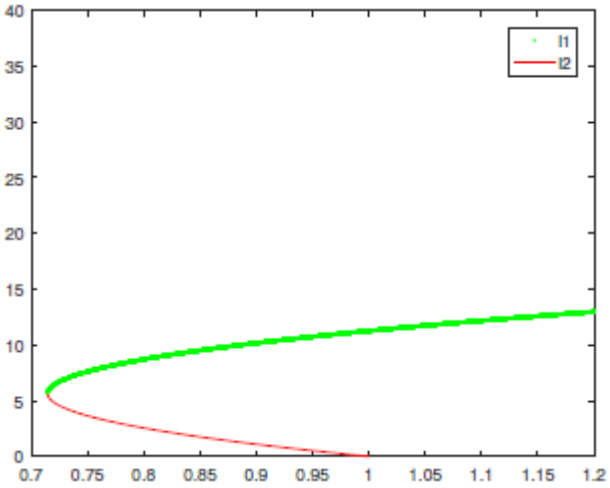


Figure 4
Bifurcation diagram of model (1) showing backward bifurcation.

6 Stability of endemic equilibrium

We prove the local stability of model (1) at endemic equilibria (EE). For this, we have the following result.

Theorem 2. The endemic equilibrium point $E_2 = (S_h^*, E_h^*, I_h^*, A_h^*, H_h^*, R_h^*, X_c^*, Y_c^*, S_c^*)$ is locally asymptotically stable if $R_0 > 1$, and it is unstable for $R_0 < 1$.

Proof. By applying the row operation and some simplification to the matrix (2) we have the following Jacobian matrix:

$$J_0 = \begin{pmatrix} -A_2 & 0 & -\frac{\beta_3 S_h}{N_h} & 0 & -\frac{\beta_4 q S_h}{N_h} & 0 & 0 & -\frac{\beta_1 S_h}{N_h} & -\frac{\beta_2 S_h}{N_h} \\ 0 & -A_2 & T_1 & 0 & T_2 & 0 & 0 & T_3 & T_4 \\ 0 & 0 & -T_5 & T_6 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -C_2 T_5 & T_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -T_7 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -T_8 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -D_2 & T_7 & -\frac{\beta_6 S_c}{N_c} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -E_2 & \frac{\beta_6 S_c}{N_c} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_c & -T_9 \end{pmatrix}. \quad [6]$$

In Eq. (6), the values of T_i for $i = 1, 2, \ldots, 8$ are given as

$$T_1 = \frac{\beta_3 S_h}{N_h} A_2 - \frac{\beta_3 S_h}{N_h} B_2, \qquad T_2 = \frac{\beta_4 q S_h A_2}{N_h} - \frac{\beta_4 q S_h B_2}{N_h},$$
$$T_3 = \frac{\beta_1 S_h A_2}{N_h} - \frac{\beta_1 S_h B_2}{N_h}, \qquad T_4 = \frac{\beta_2 S_h A_2}{N_h} - \frac{\beta_2 S_h B_2}{N_h},$$
$$T_5 = \gamma(1-\rho), \qquad T_6 = (\mu_0 + \mu_2)\gamma\rho,$$
$$T_6 = (\phi_\phi + \mu_0) - \phi_\phi \phi_a, \qquad T_7 = (\mu_0 + \mu_2)\gamma\rho\phi_a,$$
$$T_8 = \phi_\phi(\phi_\phi + \mu + \mu_0) + (\phi_\phi + \mu_0)\phi_a, \qquad T_9 = k_3 + \alpha_c.$$

Thus the eigenvalues of the Jacobian matrix (6) become

$$\lambda_1 = -G < 0, \qquad \lambda_2 = -(\gamma + \mu_0)A_2 < 0,$$
$$\lambda_3 = -C_2\gamma(1-\rho) = -T_5 < 0, \qquad \lambda_4 = -C_2\gamma(1-\rho) < 0,$$
$$\lambda_5 = -(\mu_0 + \mu_2)\gamma\rho\phi_a < 0, \qquad \lambda_6 = -\phi_\phi(\phi_\phi + \mu + \mu_0) + (\phi_\phi + \mu_0)\phi_a < 0,$$
$$\lambda_7 < 0, \qquad \lambda_8 < 0, \qquad \lambda_9 < 0.$$

Here

$$G = \frac{\gamma\beta_2\rho}{(\gamma + \mu_0)(\phi_a + \phi_1 + \mu_0 + \mu_1)} - \frac{\gamma\phi_a\beta_3 q\rho}{(\phi_1 + \mu_0)(R_0 - 1)}.$$

If $R_0 > 1$, all the eigenvalues have negative real parts. $\lambda_1 < 0$ if $R_0 > 1$, which prove the result.

6.1 Global stability of endemic equilibrium

Theorem 3. For $R_0 > 1$, the endemic equilibrium point D_1 of model (1) is stable globally, and it is unstable for $R_0 < 1$.

Proof. We define the Lyapunov function as [7, 17]

$$U(t) = \frac{1}{2}\big[(S_h - S_h^*) + (E_h - E_h^*) + (I_h - I_h^*) + (A_h - A_h^*) + (H_h - H_h^*) + (S_c - S_c^*) + (X_c - X_c^*) + (Y_c - Y_c^*)\big]^2_{[7]}$$

Differentiating (7) with respect to time, we obtain

$$U'(t) = \big[(S_h - S_h^*) + (E_h - E_h^*) + (I_h - I_h^*) + (A_h - A_h^*) + (H_h - H_h^*) + (S_c - S_c^*) + (X_c - X_c^*) + (Y_c - Y_c^*)\big] \times \left[\frac{\mathrm{d}S_h}{\mathrm{d}t} + \frac{\mathrm{d}E_h}{\mathrm{d}t} + \frac{\mathrm{d}I_h}{\mathrm{d}t} + \frac{\mathrm{d}A_h}{\mathrm{d}t} + \frac{\mathrm{d}H_h}{\mathrm{d}t} + \frac{\mathrm{d}S_c}{\mathrm{d}t} + \frac{\mathrm{d}X_c}{\mathrm{d}t} + \frac{\mathrm{d}Y_c}{\mathrm{d}t}\right].$$

After some rearrangement and using the values of system (1), we have

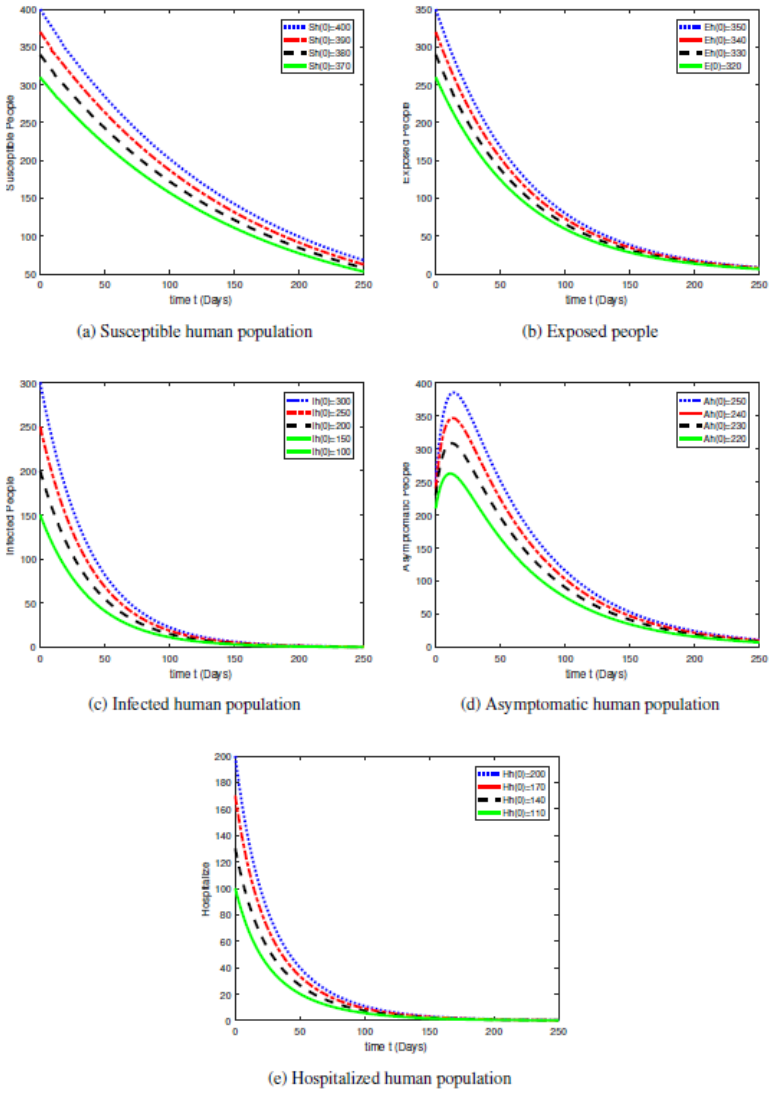
$$\begin{aligned} U'(t) = -\big[&(S_h - S_h^*) + (E_h - E_h^*) + (I_h - I_h^*) + (A_h - A_h^*) \\ &+ (H_h - H_h^*) + (S_c - S_c^*) + (X_c - X_c^*) + (Y_c - Y_c^*)\big] \\ &\times \Big[\mu_0(S_h - S_h^*) + \mu_0(E_h - E_h^*) + (\mu_0 + \mu_1)(I_h - I_h^*) \\ &+ \mu_0(H_h - H_h^*) + (\mu_0 + \mu_2)(A_h - A_h^*) - \mu_0 R_h + \mu_0(S_c - S_c^*) \\ &+ \mu_0\frac{(\phi_a + \phi_1)(\mu_0 + \mu_1)}{\gamma\rho} + \frac{\gamma\beta_2\rho S_h}{N_c(\gamma + \mu_0)} + k_1 S_c \\ &+ (k_2 + \alpha_c)X_c^*(R_0 - 1) + \frac{\gamma\beta_2\rho S_h}{N_h(\gamma + \mu_0)(\phi_a + \phi_1 + \mu_0 + \mu_1)}\Big]. \end{aligned}$$

The last equation implies that $U'(t) < 0$ for all t, while $U'(t) = 0$ if $S_h = S_h^*$, $S_c = S_c^*$, and $R_0 > 1$, which prove the global stability of model (1) around the endemic equilibrium [14].

7 Numerical simulation

To study the dynamical behavior of the MERS-CoV model, a numerical algorithm was developed and implemented in an extensive numerical simulations by using Runge–Kutta fourth-order method using the parameter values listed in Table 2. For the simulation purpose, some parameters are chosen, and some are taken from the published data. The choice of parameters are taken in such a away that would be more biological feasible.

This numerical results are given in Figs. 5–6.



(a) Susceptible human population

(b) Exposed people

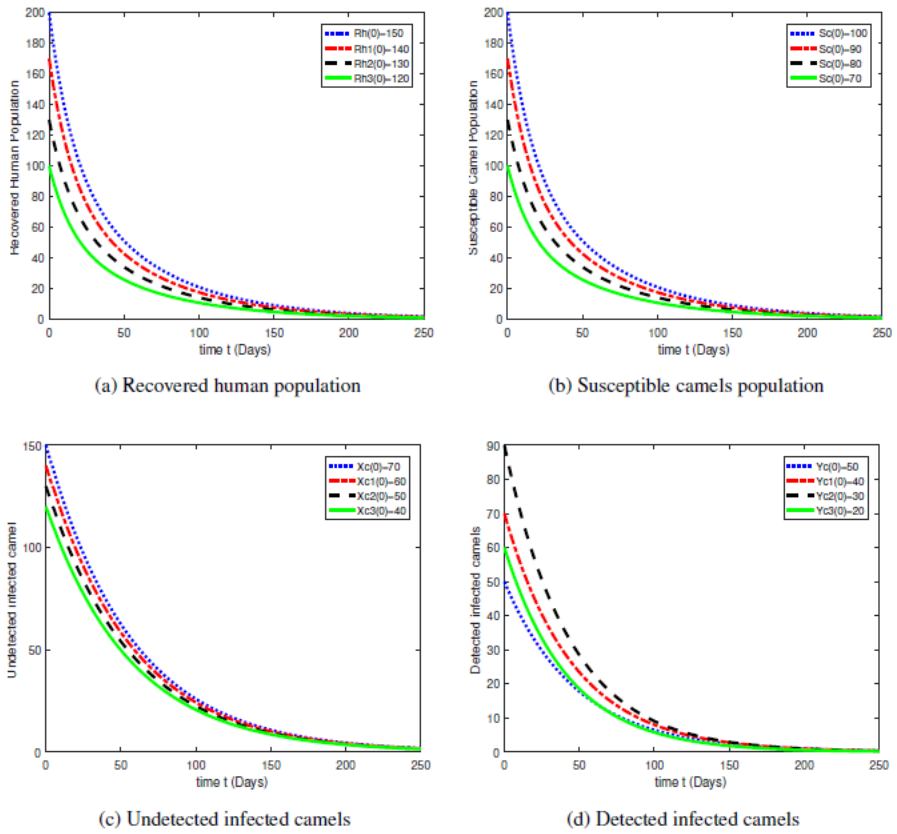
(c) Infected human population

(d) Asymptomatic human population

(e) Hospitalized human population

Figure 5

The plots demonstrate the time dynamics of different compartmental population susceptible, exposed, symptomatic and infected, infected but asymptomatic, hospitalized when $R_0 < 1$.



(a) Recovered human population

(b) Susceptible camels population

(c) Undetected infected camels

(d) Detected infected camels

Figure 6

The plots demonstrate the time dynamics of different compartmental population recover, susceptible camels, undetected infected camels, detected infected camels when $R_0 < 1$.

Table 2

Parameters and its values

Notation	Value	Source	Parameter	Value	Source
b_h	0.09	estimated	bc	0.022	estimated
β_1	0.09	estimated	β_2	0.022	estimated
β_3	0.09	estimated	β_4	0.022	estimated
μ_0	0.09	estimated	k_1	0.022	estimated
φa	0.09	estimated	γc	0.022	estimated
φa	0.09	estimated	μ_0	0.022	estimated
γ	0.026	estimated	k_2	0.000 2	[15]
k_3	0.05	estimated	ρ	0.065	estimated
μc	0.023	estimated	αc	0.04	[11]
q	0.004	estimated	φ_φ	0.014	estimated
μ_1	0.01	estimated	μ_2	0.008	estimated
β_6	0.000 1	estimated	β_7	0.008	estimated

8 Conclusion

In this work, we showed using a deterministic model the transmission of MERS-CoV between human population and camel population. We developed a model formulation and discussed the stability of the proposed model. The stability conditions are obtained in terms of R_0 . We found those condition under which the model become stable. The threshold quantity R_0 measures transmission potential of disease. We found R_0 by using next-generation matrix method. We performed sensitivity analysis of the basic reproduc- tive number in order to find the most sensitive parameter. We shown that the proposed model exhibits the phenomena of backward bifurcation. Finally, to support our analytical work, we have shown the numerical simulation for our proposed model.

Acknowledgments

We would like to thank our institutions for all the support provided while preparing this article.

References

1. J.A. Al-Tawfiq, K. Hinedi, J. Ghandour, Middle East respiratory syndrome coronavirus: A case-control study of hospitalized patients, *Clin. Infect. Dis.*, **59**(2):160–165, 2014, https://doi.org/10.1093/cid/ciu226.
2. A.N. Alagaili, T. Briese, N. Mishra, V. Kapoor, S.C. Sameroff, E. de Wit, V.J. Munster,L.E. Hensley, I.S. Zalmout, A. Kapoor, J.H. Epstein, W.B. Karesh, P. Daszak, O.B. Moham- med, W.I. Lipkin, Middle East respiratory syndrome coronavirus infection in dromedary camels in Saudi Arabia, *MBio*, .(2):e00884–14, 2014, https://doi.org/10.1128/mBio.00884-14.
3. Y.M. Arabi, A.A. Arifi, H.H. Balkhy, Clinical course and outcomes of critically ill patients with Middle East respiratory syndrome coronavirus infection, *Ann. Intern. Med.*, **160**(6):389–397, 2014, https://doi.org/10.7326/M13-2486.
4. A. Assiri, J.A. Al-Tawfiq, J.A. Al-Rabeeah, Epidemiological, demographic, and clinical characteristics of 47 cases of Middle East respiratory syndrome coronavirus disease from Saudi Arabia a descriptive study, *Lancet Infect. Dis.*, **13**(9):752–761, 2013, https://doi.org/10.1016/S1473-3099(13)70204-4.
5. E.I. Azhar, S.A. El-Kafrawy, S.A. Farraj, A.M. Hassan, M.S. Al-Saeed, A.M. Hashem,T.A. Madani, Evidence for camel to human transmission of MERS coronavirus, *N. Engl. J. Med.*, **370**(26):2499–2505, 2014, https://doi.org/10.1056/NEJMoa1401505.
6. P. Van Den Driessche, J. Watmough, Reproduction numbers and sub threshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci.,* **180**(1–2):29–48, 2002, https://doi.org/10.1016/S0025-5564(02)00108-6.

7. D.N. Fisman, A.R. Tuite, The epidemiology of MERS-CoV, *Lancet Infect. Dis.*, **14**(1):6–7, 2014, https://doi.org/10.1016.

8. W.O. Kermack, A.G. McKendrick, A Contribution to the mathematical theory of epidemics, *Proc. R. Soc. Lond., A, Math. Phys. Eng. Sci.*, **115**(772):700–721, 1927, https://doi. org/10.1098/rspa.1927.0118.

9. T. Khan, Z. Ullah, Z.N. Ali, G. Zaman, Modeling and control of the hepatitis B virus spreading using an epidemic model, *Chaos Solitons Fractals*, **124**:1–9, 2019, https://doi.org/ 10.1016/j.chaos.2019.04.033.

10. T. Khan, G. Zaman, M.I. Chohan, The transmission dynamic and optimal control of acute and chronic hepatitis B, *J. Biol. Dyn.*, **11**(1):172–189, 2017, https://doi.org/10.1080/ 17513758.2016.1256441.

11. Y. Kim, S. Lee, C. Chu, S. Choe, S. Hong, Y. Shin, The characteristics of Middle Eastern respiratory syndrome coronavirus transmission dynamics in South Korea, *Osong Public Health Res. Perspect.*, .(1):49–55, 2016, htt ps://doi.org/10.1016/j.phrp.2016.01. 001.

12. J.P. LaSalle, Stability of nonautonomous systems, *Nonlinear Anal., Theory Methods Appl.*,**61**(1):83–90, 1976, https://doi.org/10.1016/0362-546X (76)90011-0.

13. A.A. Lashari, K. Hattaf, G. Zaman, X.Z. Li, Backward bifurcation and optimal control of a vector borne disease, *Appl. Math. Inf. Sci.*, .(1):301–309, 2013, https://doi.org/10. 12785/amis/070138.

14. A.A. Lashari, G. Zaman, Global dynamics of vector borne disease with horizontal transmission in host population, *Comput. Math. Appl.*, **61**:745–754, 2011, https://doi.org/10. 1016/j.camwa.2010.12.018.

15. C. Poletto, C. Pelat, D. Lévy-Bruhl, Y. Yazdanpanah, P.Y. Boëlle, V. Colizza, Assessment of the Middle East respiratory syndrome coronavirus (MERS-CoV) epidemic in the Middle East and risk of international spread using a novel maximum likelihood analysis approach, *Eurosurveillance*, **19**(23):20824, 2014, https://doi.org/10.2807/1560-79 17. es2014.19.23.20824.

16. H.R. Thieme, *Modelling in Population Biology*, Princeton Univ. Press, Princeton, 2003,https://doi.org/10.1515/9780691187655.

17. G. Zaman, Y.H. Kang, I.H. Jung, Stability analysis and optimal vaccination of an SIR epidemic model, *Biosystems*, **93**(3):240–249, 2008, https://doi .org/10.1016/j. biosystems.2008.05.004.

18. G. Zaman, Y.H. Kang, I.H. Jung, Optimal treatment of an SIR epidemic model with time delay, *Biosystems*, **98**(1):43–50, 2009, https://doi.org/1 0.1016/j.biosystems. 2009.05.006.

19. A. Zumla, D.S. Hui, S. Perlman, Middle East respiratory syndrome, *Lancet*, **386**(9997):995– 1007, 2015, https://doi.org/10.1016/S0140-6736(15) 60454-8.

Appendix: Proof of Theorem 1

The Jacobian matrix of the system at $(F_0, 0, 0, 0, 0, 0, E_0, 0, 0)$ is given by

$$J_0 = \begin{pmatrix} -\mu_0 & 0 & -\frac{\beta_3 S_h}{N_h} & 0 & -\frac{\beta_4 q S_h}{N_h} & 0 & 0 & -\frac{\beta_1 S_h}{N_h} & -\frac{\beta_2 S_h}{N_h} \\ 0 & -(\gamma+\mu_0) & \frac{\beta_3 S_h}{N_h} & 0 & \frac{\beta_4 q S_h}{N_h} & 0 & 0 & \frac{\beta_1 S_h}{N_h} & \frac{\beta_2 S_h}{N_h} \\ 0 & \rho\gamma & -C_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & L_1 & 0 & -L_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi_a & 0 & -L_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi_1 & 0 & \phi_\phi & -\mu_0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\frac{k_3}{N_c} & -\frac{\beta_5 S_c}{N_c} & -\frac{\beta_6 S_c}{N_c} \\ 0 & 0 & 0 & 0 & 0 & 0 & D_4 & -\frac{k_3}{N_c} & \frac{\beta_6 S_c}{N_c} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_c & -T_9 \end{pmatrix},$$

Where $L_1\gamma(1-\rho)$, $L_2 = \mu_0 + \mu_2$, and $L_3 = \phi_\phi + \mu_0$.

After elementary row operation, we get the characteristic equation of the Jacobian matrix (3):

$$(\zeta+\mu_0)(\zeta+\gamma+\mu_0)\big(\zeta+C_3(\gamma+\mu_0)\big)\big(\zeta+C_3\gamma(1-\rho)\big) \times (\zeta+C_4)(\zeta+\mu_0\phi_a)\big(\zeta^3+a_1\zeta^2+a_2\zeta+a_3\big) = 0,$$

Where

$$a_1 = k_3 + 2\frac{k_3}{N_c} + k_3 + \alpha_c, \qquad a_2 = 2\frac{k_3^2}{N_c} + 2\frac{k_3}{N_c}\alpha_c + \frac{\beta_5 S_c}{N_c}D_4,$$

$$a_3 = \frac{k_3^3}{N_c^2}\alpha_c + \frac{\gamma_c\beta_6 S_c}{N_c D_4} + \frac{\beta_5 S_c D_4}{N_c}\alpha_c,$$

$$a_1 a_2 = \frac{k_3}{N_c}\Big[2k_3^2 + 2k_3\alpha_c + \beta_5 S_c D_4 + 2\frac{k_3^2}{N_c} + 4\frac{k_3}{N_c}\alpha_c + 2k_5 S_c D_4 + 2\frac{k_3}{N_c}\alpha_c + 2\alpha_c^2 + \frac{\beta_5 D_4\alpha_c}{k_3}\Big].$$

The application of Routh–Hurwitz criteria [12]

(H_1) $a_i > 0$ for $i = 1, 2, 3$ and $a_1 a_2 > a_3$

implies that the eigenvalues have negative real parts, thus model (1) is locally asymptotically stable at DFE.