Punjab University
Journal of Mathematics (ISSN 1016-2526)
Vol. 51(4)(2019) pp. 57-71

A Dynamic Compartmental Mathematical Model Describing The Transmissibility Of MERS-CoV Virus In Public

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Received: 31 May, 2018 / Accepted: 13 September, 2018 / Published online: 15 January, 2019

Abstract. In this subsection, We considered a mathematical model Middle East Respiratory Syndrome(Mers-Corona) virus and shown its spreading effect from infected camel individual to family members, clinic and care center staff. Here we apprized the transmission associated with different infection stages which generated an epidemic model. In order to do this, first we formulate the model by splitting in infectious categories and presented the basic properties of the proposed model. The basic reproduction number of the model is obtained by next generation matrix approach. Then the biological sense of threshold condition investigated and discussed in detail. Next we derived the endemic equilibrium points of our proposed model. After that we find all conditions to investigate possible equilibrium of model(local equilibrium and endemic equilibrium) in the presence of basic reproductive number. Further, global stability is discussed by the help of *Lyapunov function*. Finally, numerical simulations is perform with and with out control or vaccination by RK-4 method which support the analytical work and existence of the model.

AMS (MOS) Subject Classification Codes: 35S29; 40S70; 25U09
Key Words: MERS-Cov Virus; Mathematical Model; Basic Reproduction Number; Endemic equilibrium points; Stability Analysis; Lyapunov function; Numerical Simulation.

1. Introduction

Middle East respiratory syndrome (MERS) is a viral respiratory illness caused by a coronavirus (Middle East respiratory syndrome coronavirus, or MERS-CoV) that was first identified in Saudi Arabia in 2012. Coronaviruses are a large family of viruses that can cause

diseases in humans, ranging from the common cold to Severe Acute Respiratory Syndrome (SARS). The Middle East respiratory syndrome which is also called camel flu [24] considered acute respiration disordered mainly occurs from MERS-corona virus (MERS-Cov) [37]. The source which spread the virus is probably Camels in humans but this assumption is not clear [41], however the virus is Spread from humans to human requires close relation from infected person [37]. Up to 2016 no vaccine or treatment has been developed against the disease [41, 19]. But currently many antiviral medications is studied now a day [41]. According to World Health Organization the people how touch and connected to camels need to wash hands frequently and avoid to touch sick camels [37] in any. In April 4, 2017 approximately 2000 cases reported containing 36 percent diagnosed the virus and die [38]. Since 2012, MERS has been reported in 27 countries including Algeria, Austria, Bahrain, China, Egypt, France, Germany, Greece, Islamic Republic of Iran, Italy, Jordan, Kuwait, Lebanon, Malaysia, the Netherlands, Oman, Philippines, Qatar, Republic of Korea, Kingdom of Saudi Arabia, Thailand, Tunisia, Turkey, United Arab Emirates, United Kingdom, United States, and Yemen. However first case related in Saudi Arabia reported in June 2012 where a person having 7-days cough, fever, expectoration, and shortness of breath recorded [40]. Muhammad Ans et al. presented Decay of a Potential Vortex of Fractional Model[23]. Muhammad Irfan presented the Numerical Treatment for the Stability of Josephson Vortices in BEC with numerical approach[25]. Tahir et.al presented mathematical model for epidemic diseases[34, 29]. Further it is noticed that by applying some medicine to that person then a camel and that person have identical strains of MERS-CoV virus [10, 3] were found, while most of them occurred in Arabian Peninsula [2,3]. According to survey conducted in Saudi Arabia, at April 2014 MERS virus infected 688 persons in which 282 death occurred from MERS relate virus since 2012 [13]. The Centers for Disease Control and Prevention(CDC) reported first case diagnosis which related to MERS in United States on 2 May 2014 in Community Hospital of Munster Indiana. They reported that the infected man was healthy care worker who,s from Saudi Arabia before a week [31, 6]. Then the second case individual also belong to Saudi Arabia and reported by Florida and Orlando in 12 may 2014 by officials of Netherland reported that first case is appeared [7, 27, 18]. Philippine MERS is reported on 6 July 2015 [5] when a thirty six year foreigner came from Middle East have positive test [8]. In South Korea a first MERS case diagnosed at May 2015 when a man traveled United Arab Emirates, Bahrain and Saudi Arabia [33]. While in Middle East Camel urine is one of the medicine considered for various illnesses [39]. Similarly Fawad el al. was discussed the Modeling and Control of Zoonotic Cutaneous Leishmaniasis [22]. In United Kingdom the department of Accident and Emergency reported at 27 July 2015 the Manchester Royal Infirmary treated 2 patients for suspected MERS virus [17]. Dynamical Behavior of Mathematical Model was presented by Sultan et. al[11]. Transmission between people has been limited to-date, and has been identified among family members, patients, and health care workers. While, the majority of reported MERS cases to date have occurred in health care settings, thus far, no sustained human to human transmission has been documented anywhere in the world. According to hospital based survey one out-break of MERS had examined an incubation time period of 5.5 days who can range MERS-Cov an asymptomatic disease to pneumonia having (ARDS) or acute respiratory distress syndrome where reported that patient may have occurred (DIC) disseminated intravascular coagulation, pericarditis and a Kidney failure [2, 1, 12]. From a laboratory test its found

MERS-CoV cases individuals having low defence cells(lymphocytes) and also low white blood cell present in number [36]. MERS corona virus generally grows in LLC-MK2 cells and also in Vero cells [28]. MERSE Corona was first occurred in Saudi Arabia in 2012. Corona-viruses having big family viruses which cause infection from a common cold to Severe Acute Respiratory Syndrome (SARS). General MERS have fever, cough also shortness of the breath [9].
While Pneumonia considered common symptom, but not always. While diarrhoea is present in Gastrointestinal problem. Some laboratories shown MERS-CoV having no clinical symptoms. According to a general survey 35 percent of patients have died with MERS Corona. Majority cases of MERS spread from human-to-human connection in health care center, a recent scientific research shown camels is a major step of MERS-CoV in humans. These viruses do not spread easily unless there a close connection among peoples and camels. The most outbreaks in health occurred in various countries, while most outbreaks observed in Saudi Arabia second United Arab Emirates third in Republic of Korea [21]. Severe illness can cause respiratory failure in human. The virus appears to cause more severe disease in people with weakened immune systems, older people, and people with chronic diseases as renal disease, diabetes, cancer, and chronic lung disease.
In this paper we processed as follows that: In the first section we presented introduction. The formulation and infectious classes are given in second section. Then in third section, we given all endemic equilibrium points of the model. In section forth reproduction number and biological feasibility is discussed. Local and endemic stability analysis, at disease free equilibria are discussed in section fifth and sixth. In section seventh, eighth and ninth sensitivity analysis of global stability, at disease free ,as well as, at endemic equilibria is presented. Finally in the tenth and eleventh sections, numerical simulation, discussed and conclusion are presented respectively.

2. Model Formulation And Methodology

Here we developed a mathematical model of MERS-CoV which is zoonotic and spread from infected animals to population. According to the biological characteristics of MERS-CoV, the transmission of the virus is spread from infected animal to human or non human to human, in family member, patient to clinic center and clinic center to care center. For this purpose the papulation and virus transmission are classified in the model as: S represent susceptible camel population, A represent infected camels population, B represent infected human population by infected camels, H represent human to human transmission population, G represent infected individual to family member, P represent patient to clinic center transmission. From characteristics of MERS-CoV in the concern model lead a mathematical model of the following differential equations which we developed as,

$$\begin{aligned} S^{\bullet} &= \alpha - dS - \xi SA, \\ A^{\bullet} &= \xi SA - \beta_1 A - \beta_2 A - \chi AB, \\ B^{\bullet} &= \chi AB - \phi_1 B - \phi_2 B - \gamma BH, \\ H^{\bullet} &= \gamma BH - \theta_1 H - \theta_2 H - \phi HG, \\ G^{\bullet} &= \phi HG - \rho_1 G - \rho_2 G - \epsilon GP, \\ P^{\bullet} &= \epsilon GP - \eta_1 P - \eta_2 P - \eta PE. \end{aligned} \tag{2. 1}$$

Concerning the initial conditions, we fix the following conditions as, $S(t) \geq 0, A(t) \geq 0, B(t) \geq 0, H(t) \geq 0, G(t) \geq 0, P(t) \geq 0$, Here we drawn certain assumptions in model (1) which are classified as: α represent new birth rate in camel population, d show natural death rate of susceptible camels, β_1 represent the natural death rate of infected camels, β_2 represent infectious death rate of infected camels, χ represent the rate of infected camels to infected human population, ϕ_1 represent natural death rate of infected human individuals, ϕ_2 show the death rate of infected individual due to infection, γ represent the rate of transmission of infected individual to individual, θ_1 represent natural death rate of infected individuals, θ_2 show the death rate of infected human individuals due to infection, θ represent the rate of infection from patient to family members, ρ_1 show natural death rate of family members, ρ_2 represent infectious death rate of family members, ϵ represent the infection transmission rate from family members to clinic individuals, η_1 show natural death rate of patient in clinic, η_2 represent infectious death rate of patient in clinic, η represent infection transmission rate from clinic individual to care center individuals, π_1 represent natural death rate and π_2 represent infectious death rate in care center individuals respectively. Also we considered the total population of the model is $R(t)$ as below. $R(t) = S + A + B + H + G + P$.
By putting values in equation (2.2) from model (2.1) we get the following, $R^{\bullet} \leq \alpha - dS$. Then after simplification we get, $\lim_{t\to\infty} supR \leq \frac{\alpha}{d}$. Thus biological feasible region for the study of model (2.1) is, $\circledR = \{(S, A, B, H, G, P,) \in R_+^7, R \leq \frac{\alpha}{d}\}$. There for, to study the model dynamics the sufficient and feasible region is $\circledR$.

3. Endemic Equilibrium Points of The Proposed Model

In this subsection, we discussed the endemic equilibrium points of the said model. In any mathematical epidemiological model endemic equilibrium points play very important role. The potential existence of a disease-free equilibrium points are now discussed. As we know that the points of disease-free equilibrium results to be locally asymptotically stable when the basic reproduction number, that is, $(R_0) < 1$, while the endemic equilibrium points are not locally asymptotically stable when the reproductive number exceeds unity, that is, greater then 1. Now all the endemic equilibrium points of our proposed mathematical model are given below,

$$\begin{aligned}
S^{\star} &= \frac{1}{\xi}(\chi B + \beta_1 + \beta_2),\\
A^{\star} &= \frac{\gamma\epsilon}{\chi\phi\eta}((\pi_1 + \pi_2) + \frac{\gamma}{\chi\phi}(\rho_1 + \rho_2) + \frac{\gamma}{\chi}(\phi_1 + \phi_2)),\\
B^{\star} &= \frac{\phi G + \theta_1 + \theta_2}{\gamma},\\
H^{\star} &= \frac{\epsilon}{\phi\eta}(\pi_1 + \pi_2) + \frac{\rho_1 + \rho_2}{\phi},\\
G^{\star} &= \frac{1}{\epsilon}(\eta E + \eta_1 + \eta_2),\\
P^{\star} &= \frac{\pi_1 + \pi_2}{\eta}.
\end{aligned}$$

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4. Reproductive Number R_0 And Local Stability Analysis

In epidemiological models the role of basic reproduction number is a key concept and play very important role. It represents the expected average number of new infections produced directly and indirectly by a single infective, when introduced into a completely susceptible population. Now let us define R_0 the basic reproductive number, it is an essential and fundamental parameter having one simple definition is "An average number when an secondary infection developed by an monad person in susceptible cohort in its entire period of infection in the whole susceptible cohort" [14]. Many approaches are adopted to find the reproductive number but here we developed a technique which is known next generation matrix approach. For biological purpose the said technique is useful to determine R_0 specially in epidemic model with continuous time means system of differential equations [35]. Now in order to find R_0 we using the next generation approach. To find the basic reproductive number for our proposed model (1), we follow Driessche and Watmough [32, 30]. According to this approach the whole system of model is need to divide in two classes, infected and non infected. After that we defining the Jacobian Matrix for infectious group of the model. Then the Jacobian Matrix further divide in two sub classes and represented by (J = F -V) where J is stand for Jacobian Matrix, and F and V are new matrices. Then we find inverse of the matrix V and multiplying with F, that is, FV^{-1}. Then we derive the biggest eigne value which is the required R_0, that is, the basic reproduction number. Now the basic reproductive number of our model by taking infectious class is given by,

$$F = \begin{bmatrix} \xi SA - \chi AB \\ \chi AB - \gamma BH \\ \gamma BH - \phi HG \\ \phi HG - \varepsilon GP \\ \varepsilon GP - \eta P \\ \eta P \end{bmatrix}.$$

Now the non infectious class of the model (2.1) is represented by $\bigvee$,

$$\bigvee = \begin{bmatrix} -\beta_1 A - \beta_2 A \\ -\phi_1 B - \phi_2 B \\ -\theta_1 H - \theta_2 H \\ -\rho_1 G - \rho_2 G \\ -\eta_1 P - \eta_2 P \end{bmatrix}.$$

Now Jacobian of F of the model (2.1) is given below,

$$\overline{F} = \begin{bmatrix} \xi S - \chi B & -\chi A & 0 & 0 & 0 & 0 \\ \chi B & \chi A - \gamma H & -\gamma B & 0 & 0 & 0 \\ 0 & \gamma H & \gamma B - \phi G & -\phi H & 0 & 0 \\ 0 & 0 & \phi G & \phi H - \epsilon P & -\epsilon G & 0 \\ 0 & 0 & 0 & \epsilon P & \epsilon G & -\eta P \\ 0 & 0 & 0 & 0 & 0 & \eta P \end{bmatrix}.$$

Now Jacobian of $\bigvee$, its inverse and the required value of R_0 is given by,

$$\overline{V} = \begin{bmatrix} (\beta_1+\beta_2) & 0 & 0 & 0 & 0 & 0 \\ 0 & (\phi_1+\phi_2) & 0 & 0 & 0 & 0 \\ 0 & 0 & (\theta_1+\theta_2) & 0 & 0 & 0 \\ 0 & 0 & 0 & (\rho_1+\rho_2) & 0 & 0 \\ 0 & 0 & 0 & 0 & (\eta_1+\eta_2) & 0 \\ 0 & 0 & 0 & 0 & 0 & (\pi_1+\pi_2) \end{bmatrix}.$$

Now R_0, the basic reproductive for the model (2.1) is,

$$R_0 = [\frac{\alpha - dS}{(\beta_1+\beta_2)A}].$$

5. Local Stability Analysis At Disease Free Equilibrium of The Model

To find the local stability analysis at disease free equilibrium of the model (2.1) the points of local stability analysis are, $E_{p_0} = (S, d_A, d_B, d_H, d_G, d_P)$ which implies $E_{p_0} = (\frac{\alpha - dS}{\xi A}, 0, 0, 0, 0, 0)$. Thus we processed by *Jacobian matrix,* as,

$$E_{p_0} = \begin{pmatrix} 0 & -\xi S & 0 & 0 & 0 & 0 & 0 \\ 0 & A_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\phi_1-\phi_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\theta_1-\theta_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\rho_1-\rho_2 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\eta_1-\eta_2 & 0 \end{pmatrix} \quad (5.\ 2)$$

Where,
$A_1 = \xi S - \beta_1 - \beta_2$
and,
$B_1 = (\pi_1 + \pi_2)$. Now the following known result will stated for local stability analysis at disease free equilibrium.

Theorem 5.1. *If the reproductive number $R_0 < 1$, model (1) is* locally asymptotically stable *at disease free equilibrium, $E_{p_0} = (\frac{\alpha - dS}{\xi A}, 0, 0, 0, 0, 0)$ and if $R_0 > 1$ then unstable.*

Proof: We have the following eigenvalues from Jacobian matrix defined in equation (3),

$$\lambda_1 = \chi S - \beta_1 - \beta_2, \quad (5.\ 3)$$
$$\lambda_2 = -\phi_1 - \phi_2, \quad (5.\ 4)$$
$$\lambda_3 4 = -\theta_1 - \theta_2, \quad (5.\ 5)$$
$$\lambda_4 = -\rho_1 - \rho_2, \quad (5.\ 6)$$
$$\lambda_5 = -\eta_1 - \eta_2, \quad (5.\ 7)$$
$$\lambda_6 = -\pi_1 - \pi_2. \quad (5.\ 8)$$

We observed from equations (5.5), (5.6), (5.7), (5.8) and (5.9) that all the eigenvalues are negative except from equation (5.4), that is $\lambda_2 = \chi S - \beta_1 - \beta_2$, Clearly $\lambda_2 = \chi < 0$ if and only if, $R_0 < 1$.
Now at the disease free equilibrium all the values of the system (2.1) are less then unity. So system (1) is locally asymptotically stable which complete the proof.

6. Local stability Analysis of The Model At Endemic Equilibrium

Theorem 6.1. *The model (2.1) defined in equation (5.3) is locally asymptotically stable at endemic equilibrium* $S(t) = S^\star(t), dA(t) = d^\star A(t), dB(t) = d^\star B(t), dH(t) = d^\star H(t), dG(t) = d^\star G(t), dP(t) = d^\star P(t)$. *If* $R_0 > 1$, *then model (2.1) is stable if not, then unstable.*

$$E_{P_E} = \begin{pmatrix} -\xi A^\star & -\xi S^\star & 0 & 0 & 0 & 0 & 0 \\ \xi A^\star & A_1 & -\chi A^\star & 0 & 0 & 0 & 0 \\ 0 & \chi B^\star & B_1 & -\gamma B^\star & 0 & 0 & 0 \\ 0 & 0 & \gamma H^\star & K & -\gamma H^\star & 0 & 0 \\ 0 & 0 & 0 & \phi G^\star & K_1 & -\phi G^\star & 0 \\ 0 & 0 & 0 & 0 & \epsilon P^\star & F & -\eta P^\star \end{pmatrix}.$$

From simplification we get the following eigenvalues.

$$\begin{aligned} \lambda_1 &= -\xi A^\star, && (6.\ 9)\\ \lambda_2 &= A_1 - \xi S^\star, && (6.\ 10)\\ \lambda_3 &= -G_1, && (6.\ 11)\\ \lambda_4 &= -K_2, && (6.\ 12)\\ \lambda_5 &= K_3, && (6.\ 13)\\ \lambda_6 &= P_1. && (6.\ 14) \end{aligned}$$

The terms used above are classified by the following,

$$\begin{aligned} A_1 &= \xi S^\star - \beta_1 - \beta_2 - \chi B^\star,\\ B_1 &= \chi A^\star - \phi_1 - \phi_2 - \gamma H^\star,\\ K &= \gamma B^\star - \theta_1 - \theta_2 - \phi G^\star,\\ K_1 &= \phi H^\star - \rho_1 - \rho_2 - \eta P^\star,\\ F &= \eta - \pi_1 - \pi_2,\\ G &= -(\chi^2 A^\star B^\star + B_1(A_1 - \xi S^\star)), && (6.\ 15)\\ K_2 &= -(G_1 C + \gamma^2 B^\star H^\star),\\ K_4 &= G_1 \phi H^\star - (A_1 - \xi S^\star)\gamma H^\star,\\ K_3 &= -K_1 K_2 - K_4 \phi G^\star,\\ K_5 &= K_2 \epsilon G^\star,\\ P_1 &= M_1 - N_5 \epsilon P^\star. \end{aligned}$$

From above equations it is clear that equations (6.10) and (6.11), that is, λ_1 and λ_2 are negative eigne values. Also equation (6.12),e.g. $\lambda_3 < 0$ iff $\gamma < 1$. Using equation (6.13), that is, $\lambda_4 < 0$ iff $\phi < 0$. Taking equation (6.14), implies $\lambda_5 < 0$ if $\chi < 1$. Also from equation (6.15), that is $\lambda_6 < 0$ iff $\chi < 1$ and $\gamma < 0$.All the values of model (1) are negative eigne values, so model (2.1) is asymptotically stable at endemic equilibrium.

7. Global Stability Analysis of The Proposed Model

In this subsection, we find the global stability analysis of the proposed model. In mathematical epidemiology global stability analysis is an interesting and especial work. For this purpose we construct a *Lyapunov function* for global stability at disease free equilibrium and at endemic equilibrium of the model. The Lyapunov function [20, 16] is an interesting and easy rule to study stability analysis. Many authors [20, 35] use this technique for the same job in there work. Now for global stability analysis of model (2.1) we define the following Lyapunov function for stability and also we have the following stability results which are stated below.

8. Global Stability Analysis of The Model At Disease Free Equilibrium

Theorem 8.1. *Model* (2.1) *at disease free equilibrium is said globally asymptotically stable, if* $R_0 \leq 1$*, at* $S = S_0$ *and the model unstable for* $R_0 > 1$.

Proof: To find the global stability analysis at disease free equilibrium, we define the following *Lyapunov function,*

$$Z(S, dA, dB, dH, dG, dP) = \frac{1}{2}[S - S^\star + A - A^\star + B - B^\star + H - H^\star + G - G^\star + P - P^\star]^2 \quad (8.\ 16)$$

We see clearly that the above function $Z(S, dA, dB, dH, dG, dP) \geq 0$, and it also equal to zero if and only if $S = S^\star, A = A^\star, B = B^\star, H = H^\star, G = G^\star, P = P^\star$. Now to show the required result, let us differentiate equation (8.17) *with respect to* t, we get,

$$\frac{dZ}{dt}(S, dA, dB, dH, dG, dP) = (S - S^\star + A - A^\star + B - B^\star + H - H^\star + G - G^\star + P - P^\star)(\frac{dS}{dt} + \frac{dA}{dt} + \frac{dB}{dt} + \frac{dH}{dt} + \frac{dG}{dt} + \frac{dP}{dt}) \quad (8.\ 17)$$

Using values from model (2.1), then equation (8.18) becomes,

$$\frac{dZ}{dt}(S, dA, dB, dH, dG, dP) = (S - S^\star + A - A^\star + B - B^\star + H - H^\star + G - G^\star + P - P^\star)[\alpha - (dS + (\beta_1 + \beta_2)A + (\phi_1 + \phi_2)B + (\theta_1 + \theta_2)H + (\rho_1 + \rho_2)G + (\eta_1 + \eta_2)P)].$$

From above it is clear that
$\frac{dZ}{dt}(S, dA, dB, dH, dG, dP) = 0$ if and only if $S = S^\star, A = A^\star, B = B^\star,$
$H = H^\star, G = G^\star, P = P^\star$, further it also be clear that
$\frac{dZ}{dt}(S, dA, dB, dH, dG, dP) < 0$ for $\alpha < K$,
where $K = (dS + (\beta_1 + \beta_2)A + (\phi_1 + \phi_2)B + (\theta_1 + \theta_2)H + (\rho_1 + \rho_2)G + (\eta_1 + \eta_2)P]$. Which show that the global asymptotically stability at disease free equilibrium is stable, which complete the proof.

9. Global Stability Analysis of The Model At Endemic Equilibrium

Theorem 9.1. *Endemic equilibrium for model* (2.1) *is* globally asymptotically stable *if* $R_0 > 1$ *, if* $S = S^\star$*,* $A = A^\star$*,* $B = B^\star$*,* $H = H^\star$*,* $G = G^\star$*,* $P = P^\star$*, and unstable, if* $R_0 < 1$*, that is,* R_0 *is less then unity.*

Proof: Now to show the global stability analysis at endemic equilibrium of the model (2.1), considered the following *Lyapunov function*,

$$X(S, dA, dB, dH, dG, dP) = \frac{1}{2}(S - S^\star)^2 + \frac{1}{2}(A - A^\star)^2 + \frac{1}{2}(B - B^\star)^2 + \frac{1}{2}(H - H^\star)^2 + \frac{1}{2}(G - G^\star)^2 + \frac{1}{2}(P - P^\star)^2.$$

We see the above define function, $X(S, dA, dB, dH, dG, dP) \geq 0$, and its equal to zero, if and only if $S = S^\star$, $A = A^\star$, $B = B^\star$, $H = H^\star$, $G = G^\star$, $P = P^\star$, now Differentiating the above equation *with respect to* "t" we have,

$$\frac{dX}{dt}(S, dA, dB, dH, dG, dP) = [(S - S^\star) + (A - A^\star) + (B - B^\star) + (H - H^\star) + (G - G^\star) + (P - P^\star)][\alpha - dS + (\beta_1 + \beta_2)A + (\phi_1 + \phi_2)B + (\theta_1 + \theta_2)H + (\rho_1 + \rho - 2)G + (\eta_1 + \eta_2)P],$$

After some simplification we get the following,

$$\frac{dX}{dt}(S, dA, dB, dH, dG, dP) = -[(S - S^\star) + (A - A^\star) + (B - B^\star) + (H - H^\star) + (G - G^\star) + (P - P^\star)](W - \alpha) \qquad (9.\ 18)$$

Where the value of "W" is given by,

$$W = dS + (\beta_1 + \beta_2)A + (\phi_1 + \phi_2)B + (\theta_1 + \theta_2)H + (\rho_1 + \rho_2)G + (\eta_1 + \eta_2)P].$$

From equation (9.19) it is clear that $\frac{dX}{dt}(S, dA, dB, dH, dG, dP) = 0$ if $S = S^\star$, $A = A^\star$, $B = B^\star$, $H = H^\star$, $G = G^\star$, $P = P^\star$, and also equation (9.19)
$\frac{dX}{dt}(S, dA, dB, dH, dG, dP) < 0$ if and only if $W < \alpha$.
From above the model (2.1) is globally asymptotically stable at endemic equilibria which is required.

10. Numerical Simulation

In this section, we want to observe the dynamical behavior of our proposed model. In order to do this, we purpose numerical results by using Runge-Kutta of order 4th scheme [16, 15, 4, **?**] which have used several authors for a wide range of problems consisting of ordinary differential equations. For the simulation purpose, we use different value of parameters used in the proposed model are given in the Table. The parameters are taken in such away which would be more biologically feasible. Moreover the time interval is taken $0 - 1$ year. While the different positive population size for the compartmental population susceptible camel population $S(t)$, infected camel population $A(t)$, infected camel to infect human population $B(t)$, Infected individual to infect healthy population $H(t)$, infected individual to, infect family member population $G(t)$ and family member to clinic center population $P(t)$ are presented in Table below. By using the parameters value, non-negative initial population sizes and the time interval $0 - 1(year)$, we obtain the simulation Figs (1) to (6) are presented with and without vaccine or control to reduce the numbers of infected individuals and maximize the numbers of susceptible individuals, so the differences can be easily seen from the simulation. Also we observe that with control or vaccine the recovery is very fast as compared without control or vaccine.

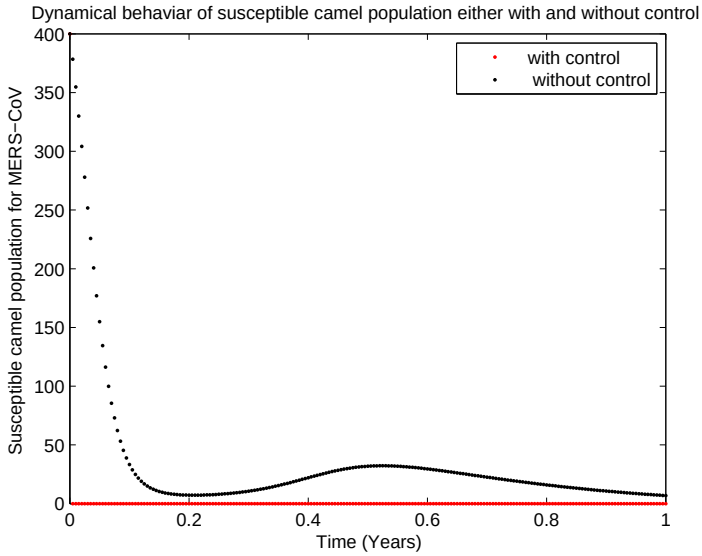

FIGURE 1. The plot represents the time dynamics of the susceptible camel population with and without control .

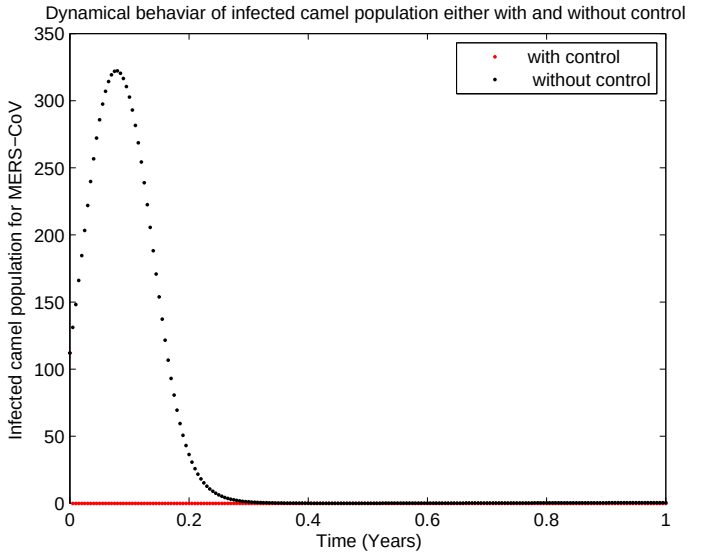

FIGURE 2. The plot represents the time dynamics of infected camel population with and without control.

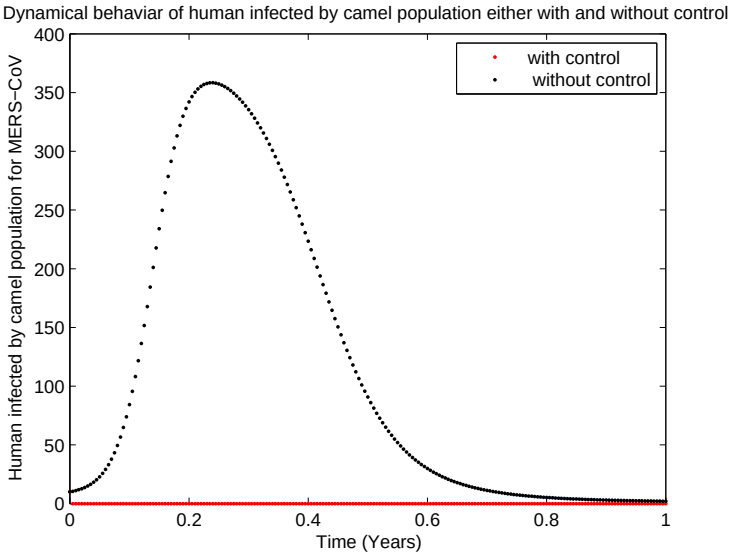

FIGURE 3. The plot represents the time dynamics of infected camel to infect human population with and without control.

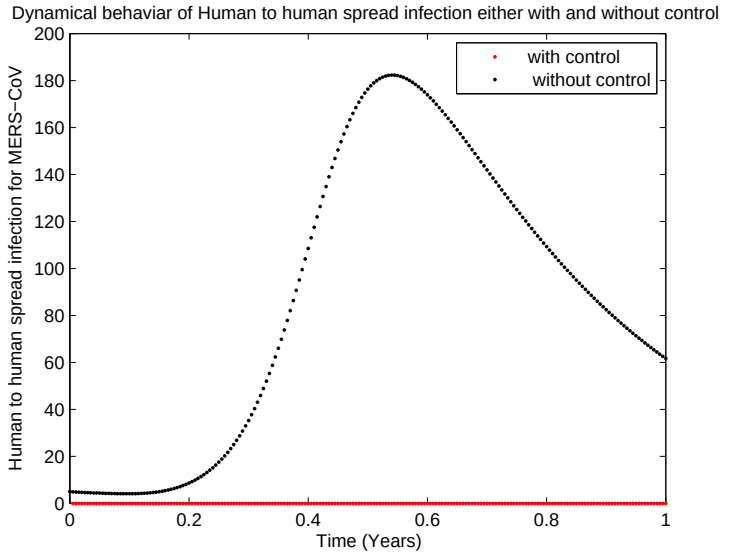

FIGURE 4. The plot show time dynamics of infected individual to healthy population with and without control.

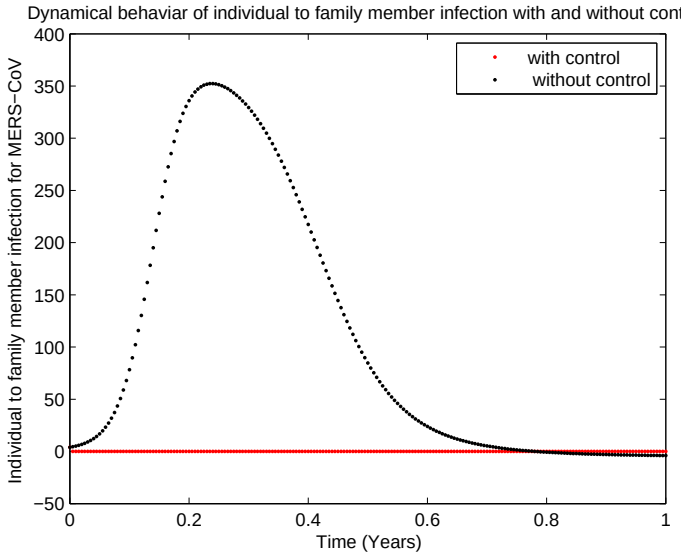

FIGURE 5. The plot represents the time dynamics of individual to family member population with and without control.

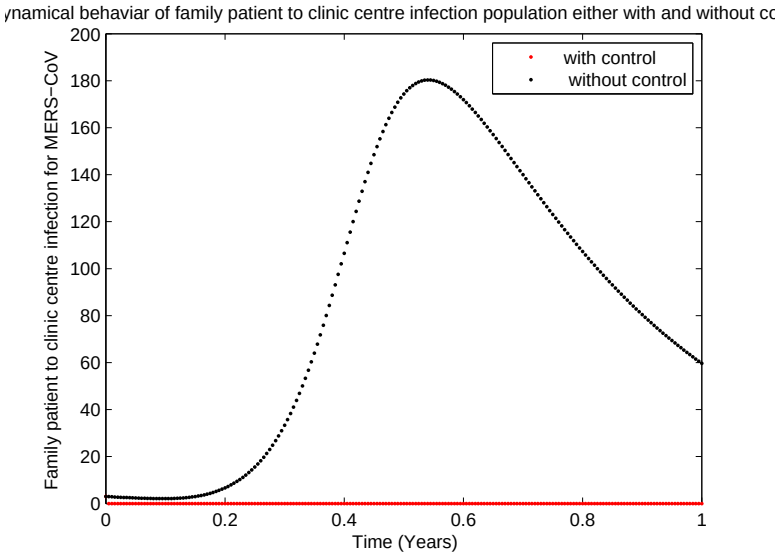

FIGURE 6. The plot represents the time dynamics of family member to clinic population with and without control.

Notation	Parameter	Value
$R(t)$	Total population Taken	00-500
S	Susceptible camel population	00-400
A	Infected camel population	00-350
B	Infected camel to infect human population	00-400
H	Infected individual to healthy population	00-200
G	Individual to own family member population	00-400
P	Family member to clinic center population	00-200
α	New birth rate in camels population	1.5000
d	Natural death rate in camels population	1.7000
β_2	Natural death rate in infected camels population	0.0143
β_1	Infectious death rate in infected camels population	0.1340
ϕ_2	Natural death rate of infect human population	0.3002
ϕ_1	Infectious death rate of infect human population	0.1343
θ_2	Natural death rate of infected to healthy individual	0.0054
θ_1	Infectious death rate of infected to healthy individual	0.0024
ρ_2	Natural death rate of individual to family member	0.0019
ρ_1	Infectious death rate of individual to family member	0.0074
η_2	Natural death rate of family member to clinic center	0.0640
η_1	Infectious death rate of family member to clinic center	0.3440
π_2	Natural death rate of clinic patient to care center	0.4400
π_1	Infectious death rate of clinic patient to care center	0.5410
ξ	Transmission rate from susceptible to infected camel population	1.2300
χ	Transmission rate of infected to infect human population	0.1000
γ	Transmission rate of infected to healthy individual	0.0060
ϕ	Transmission rate of infected to family member	1.0090
ϵ	Transmission rate of infected family to clinic center	0.0040
η	Transmission rate of clinic individual to care center	0.0900

11. CONCLUSION

In this article, we have established a model for the transmission dynamic of MERS-Cov virus by taking into account the classification of different phases of its spread in population. We presented different mathematical analysis including, biological feasibility and positivity analysis of the proposed model. We obtained the basic reproduction number by using next generation matrix approach and then discussed its feasibility. Moreover, we discussed the stability analysis and showed that the proposed model is both locally, as well as, globally asymptotically stable for the disease free as well as for endemic equilibriums, while the global stability is retrieved by using *Lyapunov function theory* and geometrical approach. Finally, the numerical simulation are presented with and with out control or vaccination to show the feasibility of the proposed work.

12. ACKNOWLEDGMENTS

The author would like to express their deep sincere thanks to the referees for their careful assessment and fruitful remarks and suggestions regarding this manuscript. The corresponding author Muhammad Tahir, is highly thankful to the Islamia College, University, Peshawar, 25000, Pakistan and University of Malakand, chakdara, District, Lower Dir, Pakistan for facilitating this research work. I also pay thanks to my supervisor, Prof. Dr. Syed Inayat Ali Shah and Co-supervisor Dr. Gul Zaman, for the patient guidance, encouragement and advice. I also pay thanks and gramercy to Tahir Khan who provided extra knowledge and courage.

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