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Stability Behaviour of Mathematical Model MERS Corona Virus Spread in Population

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Abstract. In this subsection, we first formulated the proposed model in there infectious classes and then we derived the basic key value reproductive number, R_0 with the help of *next generation approach*. Then we obtained all the endemic equilibrium points, as well as, local stability analysis, at disease free equilibria and, at endemic equilibria of the related model and shown stable. Further the global stability analysis either, at disease free equilibria, and at endemic equilibria is discussed by constructing *Lyapunov function* which show the validity of the concern model exist. In the last part of the article numerical simulation is presented for the model which support the model existence with the help of RK-4 method.

1. Introduction

MERS-CoV(Middle East respiratory syndrome corona virus) considered chronic disease for respiration was reported in Saudi Arabia, in 2012. Mostly it linked to the Arabian countries. This virus is perhaps came from animals, like, camels. Still MERS-CoV have no cure or vaccination developed, so why it is fatal disease and considered pandemic. Here we considered MERS-CoV virus which gradually spread in population through camel to owner, shepherd and those who care and treat the camels. The study open the secrete that the said disease if once attack it round from patient to family member, family member to hospital, from hospital to clinic centre and then spread in care centre. For the cure purpose treat the camels and patients in protective way because still no medication or vaccine has been invented, so special precaution are advise to those who being a part of camels. Also blood, saliva, meat and milk of infected camel is prohibited in any case. The disease is mainly in Arabic counties but have to care all main kind.

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 [1] considered acute respiration disordered mainly occurs from MERS-corona virus

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(MERS-Cov) [2]. The source which spread the virus is probably camels in humans but this assumption is not clear[3], however the virus is Spread from humans to human requires close relation from infected person [2]. Up to 2016 no vaccine or treatment has been developed against the disease [3,4]. But currently many antiviral medications is studied now a day [3]. According to World Health Organization the people how touch and connected to camels need to wash hands frequently and avoid to touch sick camels [2] in any.

In April 4, 2017 approximately 2000 cases have reported containing 36 percent diagnosed the virus and die [5]. 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 [6]. Mathematical model has been presented for Ebola virus by Tahir et al [24]. 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 [11,12] 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 [15]. 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 [16,17].

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 [18,19,20]. 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. Majority cases of MERS virus 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. Philippine MERS virus is reported on 6 July 2015 when a thirty six year foreigner came from Middle East have positive test [22]. In South Korea a first MERS case diagnosed at May 2015 when a man traveled United Arab Emirates, Bahrain and Saudi Arabia [21]. While in Middle East camel urine is one of the medicine considered for various illnesses [13]. 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 [23].

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 [7,8,9]. From a laboratory test its found MERS-CoV cases individuals having low defence cells(lymphocytes) and also low white blood cell present in number [14]. MERS corona-virus generally grows in LLC-MK2 cells and also in Vero cells [10]. 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 article we focus on the disease MERS corona virus which is considered chronic in Arabic countries while the main agent of the transmission of the disease is considered camels. The model is new in respect of this disease. Disease virus is spread in population if no precaution is used, we try to aware and established such work through by applying some conditions we avoid the said disease but if not, we see that virus is then transferred from an infected camel to healthy person and gradually from their it round in different places, community, country and in last effect the whole world. So for this order, we first formulated the model with all of its infectious classes and derived the basic reproductive number R_0 with the next generation approach. Then all endemic equilibrium points are derived. In the presence of reproductive number we discussed all the equilibria, that is, local stability analysis, at disease free and, at endemic equilibria are shown stable for $R_0 < 1$. Similarly we shown the global stability at both respect with the help of *Lyapunov function*. In the last we discussed the model by numerical approach of Runge-Kutta method of order four 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 centre and clinic centre to care centre. For this purpose the papulation and virus transmission are classified in the model as: S_c, represent susceptible camel population, I_c represent infected camels population, I_p represent infected human population by infected camels, H_h represent human to human transmission population, F_m represent infected individual to family member, P_c represent patient to clinic centre transmission and C_c represent infected patient to care centre transmission population. From characteristics of MERS-CoV in the concern model lead a mathematical model of the following differential equations which we developed as,

$$\begin{aligned}
\frac{dS_c}{dt} &= \mu_s - d_{ns_c}S_c - \beta_1 S_c I_c,\\
\frac{dI_c}{dt} &= \beta_1 S_c I_c - (d_{dI_c} + d_{nI_c})I_c - \beta_2 I_c I_p,\\
\frac{dI_p}{dt} &= \beta_2 I_c I_p - (d_{dI_p} + d_{nI_p})I_p - \beta_3 I_p H_h,\\
\frac{dH_h}{dt} &= \beta_3 I_p H_h - (d_{dH_h} + d_{nH_h})H_h - \beta_4 H_h F_m,\\
\frac{dF_m}{dt} &= \beta_4 H_h F_m - (d_{dF_m} + d_{nF_m})F_m - \beta_5 F_m P_c,\\
\frac{dP_c}{dt} &= \beta_5 F_m P_c - (d_{dP_c} + d_{nP_c})P_c - \beta_6 P_c C_c,\\
\frac{dC_c}{dt} &= \beta_6 P_c C_c - (d_{dC_c} + d_{nC_c})C_c.
\end{aligned} \tag{1}$$

Concerning the initial conditions, we fix the following conditions as,

$$S_c(t) \geq 0, I_c(t) \geq 0, I_p(t) \geq 0, H_h(t) \geq 0, F_m(t) \geq 0, P_c(t) \geq 0, C_c(t) \geq 0.$$

Here we drawn certain assumptions in model (1) which are classified as: μ_c and d_{ns_c} represent new birth rate, and natural death rate in susceptible camels population, β_1 represent transmission rate of infection in susceptible camels population from infected camel population, d_{dI_c} and d_{nI_c} represent natural death rate, and infectious death rate in infected camels population, β_2 represent transmission rate of infection in human population by infected camels population, d_{dI_p} and d_{nI_p} represent natural death rate, and infectious death rate of infected human population, β_3 represent infection transmission rate from infected individual to healthy infected individual population(human to human), while d_{dH_h} and d_{nH_h} represent natural death rate, and infectious death rate in healthy infected individual population(human to human), β_4 represent transmission rate of infection from infected individual to own family member population, d_{dF_m} and d_{nF_m} represent natural death rate and infectious death rate in family member, β_5 represent infection transmission rate from family member to patient in clinic, d_{dP_c} and d_{nP_c} represent natural death rate, and infectious death rate of individuals to clinic centre, β_6 represent infection transmission rate of clinic patient to care center, d_{dC_c} and d_{nC_c} represent natural death rate and infectious death rate in care center patient respectively.
Also we considered the total population of the model is $Z(t)$ as,

$$Z(t) = S_c + I_c + I_p + H_h + F_m + P_c + C_c. \tag{2}$$

By putting values in equation (2) from model (1) we get the following,

$$\frac{dZ(t)}{dt} \leq \mu_c - d_{ns_c} S_c.$$

Then after simplification we get,

$$\lim_{t\to\infty} supZ \leq \frac{\mu_c}{d_{ns_c}}.$$

Thus biological feasible region for the study of model (1) is,

$$\mho = \{(S_c, I_c, I_p, H_h, F_m, P_c, C_c) \in R_+^7, Z \leq \frac{\mu_c}{d_{ns_c}}\}.$$

There for, to study the model dynamics the sufficient and feasible region is $\mho$.

3. Endemic Equilibrium Points of The Proposed Mode

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,

$$\begin{aligned}
S_c^\star &= \frac{\beta_2 I_p + d_{dI_c} + d_{nI_c}}{\beta_1},\\
I_c^\star &= \frac{\beta_3}{\beta_2\beta_4\beta_6}(\beta_5(d_{dC_c} + d_{nC_c}) + \beta_6(d_{dF_m} + d_{nF_m}) + \beta_4\beta_6(d_{dI_p} + d_{nI_p})),\\
I_p^\star &= \frac{\beta_4 F_m + d_{dH_h} + d_{nH_h}}{\beta_3},\\
H_h^\star &= \frac{1}{\beta_4\beta_6}(\beta_5(d_{dC_c} + d_{nC_c}) + \beta_6(d_{dF_m} + d_{nF_m})),\\
F_m^\star &= \frac{\beta_6 C_c + d_{dP_c} + d_{nP_c}}{\beta_5},\\
P_c^\star &= \frac{d_{dC_c} + d_{nC_c}}{\beta_6},\\
C_c^\star &= \frac{\beta_5 F_m - (d_{dP_c} + d_{nP_c})}{\beta_6}.
\end{aligned}$$

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"[25]. 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 [24]. Now in order to find R_0 we using the next generation approach. 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,

$$F = \begin{bmatrix} \beta_1 S_c I_c - \beta_2 I_c I_p \\ \beta_2 I_c I_p - \beta_3 I_p H_h \\ \beta_3 I_p H_h - \beta_4 H_h F_m \\ \beta_4 H_h F_m - \beta_5 F_m P_c \\ \beta_5 F_m P_c - \beta_6 P_c C_c \\ \beta_6 P_c C_c \end{bmatrix}.$$

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

$$\bigvee = -\begin{bmatrix} (d_{dI_c} + d_{nI_c})I_c \\ (d_{dI_p} + d_{nI_p})I_p \\ (d_{dH_h} + d_{nH_h})H_h \\ (d_{dF_m} + d_{nF_m})F_m \\ (d_{dP_c} + d_{nP_c})P_c \\ (d_{dC_c} + d_{nC_c})C_c \end{bmatrix}.$$

Now Jacobian of F of model (1) is given below,

$$\overline{F} = \begin{bmatrix} \beta_1 S_c - \beta_2 I_p & -\beta_2 I_c & 0 & 0 & 0 & 0 \\ \beta_2 I_p & \beta_2 I_c - \beta_3 H_h & -\beta_3 I_p & 0 & 0 & 0 \\ 0 & \beta_3 H_h & \beta_3 I_p - \beta_4 F_m & -\beta_4 H_h & 0 & 0 \\ 0 & 0 & \beta_4 F_m & \beta_4 H_h - \beta_5 P_c & -\beta_5 F_m & 0 \\ 0 & 0 & 0 & \beta_5 P_c & \beta_5 F_m - \beta_6 C_c & -\beta_6 P_c \\ 0 & 0 & 0 & 0 & 0 & \beta_6 P_c \end{bmatrix}.$$

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

$$\overline{V} = \begin{bmatrix} (d_{dI_c} + d_{nI_c}) & 0 & 0 & 0 & 0 & 0 \\ 0 & (d_{dI_p} + d_{nI_p}) & 0 & 0 & 0 & 0 \\ 0 & 0 & (d_{dH_h} + d_{nH_h}) & 0 & 0 & 0 \\ 0 & 0 & 0 & (d_{dF_m} + d_{nF_m}) & 0 & 0 \\ 0 & 0 & 0 & 0 & (d_{dP_c} + d_{nP_c}) & 0 \\ 0 & 0 & 0 & 0 & 0 & (d_{dC_c} + d_{nC_c}) \end{bmatrix}.$$

Now R_0, the basic reproductive for the model (1) is,

$$R_0 = [\frac{\mu_s - d_n S_c}{(d_{dI_c} + d_{nI_c})I_c}].$$

Now Local Stability Analysis At Disease Free Equilibrium of The Model

To find the local stability analysis at disease free equilibrium of the model (1) the points of local stability analysis are, $E_{p_0} = (S_c, d_{I_c}, d_{I_p}, d_{H_h}, d_{F_m}, d_{P_c}, d_{C_c})$ which implies $E_{p_0} = (\frac{\mu_s - d_n S_c}{\beta_1 I_c}, 0, 0, 0, 0, 0, 0)$. Thus we

processed by *Jacobian matrix*, as,

$$E_{p_0} = \begin{pmatrix} 0 & -\beta_1 S_c & 0 & 0 & 0 & 0 & 0 \\ 0 & A & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(d_{dI_p} + d_{nI_p}) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(d_{dH_h} + d_{nH_h}) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(d_{dF_m} + d_{nF_m}) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(d_{dP_c} + d_{nP_c}) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -B \end{pmatrix}. \tag{3}$$

Where,
$A = \beta_1 S_c - (d_{dI_c} + d_{nI_c})$,
and,
$B = (d_{dC_c} + d_{nC_c})$.

Now the following known result will stated for local stability analysis at disease free equilibrium.

Theorem 4.1. *If the reproductive number $R_0 < 1$, model (1) is* locally asymptotically stable *at disease free equilibrium, $E_{p_0} = (\frac{\mu_s - d_n S_c}{\beta_1 I_c}, 0, 0, 0, 0, 0, 0)$ and if $R_0 > 1$ then unstable.*

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

$$\lambda_1 = 0, \tag{4}$$
$$\lambda_2 = \beta_2 S_c - (d_{dI_c} + d_{nI_c}), \tag{5}$$
$$\lambda_3 = -(d_{dI_p} + d_{nI_p}), \tag{6}$$
$$\lambda_4 = -(d_{dH_h} + d_{nH_h}), \tag{7}$$
$$\lambda_5 = -(d_{dF_m} + d_{nF_m}), \tag{8}$$
$$\lambda_6 = -(d_{dP_c} + d_{nP_c}), \tag{9}$$
$$\lambda_7 = -(d_{dC_c} + d_{nC_c}). \tag{10}$$

We observed from equations (6), (7), (8), (9) and (10) that all the eigenvalues are negative except from equation (4) and (5), that is $\lambda_1 = 0$ and $\lambda_2 = \beta_2 S_c - (d_{dI_c} + d_{nI_c})$, Clearly $\lambda_2 = \beta_2 < 0$ if and only if, $R_0 < 1$. Now at the disease free equilibrium all the values of the system (1) are less then unity. So model (1) is locally asymptotically stable with $\lambda_1 = 0$ which complete the proof.

Local stability Analysis of The Model At Endemic Equilibrium

Theorem 4.2. *The model (1) defined in equation (3) is locally asymptotically stable at endemic equilibrium $S_c(t) = S_c^\star(t), dI_c(t) = d^\star I_c(t), dI_p(t) = d^\star I_p(t), dH_h(t) = d^\star H_h(t), dF_m(t) = d^\star F_m(t), dP_c(t) = d^\star P_c(t), dC_c(t) = d^\star C_c(t)$. If $R_0 > 1$, then model (1) is stable if not, then unstable.*

$$E_{P_E} = \begin{pmatrix} -\beta_1 I_c^\star & -\beta_1 S_c^\star & 0 & 0 & 0 & 0 & 0 \\ \beta_1 I_c^\star & A & -\beta_2 I_c^\star & 0 & 0 & 0 & 0 \\ 0 & \beta_2 I_p^\star & B & -\beta_3 I_p^\star & 0 & 0 & 0 \\ 0 & 0 & \beta_3 H_h^\star & C & -\beta_4 H_h^\star & 0 & 0 \\ 0 & 0 & 0 & \beta_4 F_m^\star & D & -\beta_5 F_m^\star & 0 \\ 0 & 0 & 0 & 0 & \beta_5 P_c^\star & E & -\beta_6 P_c^\star \\ 0 & 0 & 0 & 0 & 0 & \beta_6 C_c^\star & F \end{pmatrix}.$$

From simplification we get the following eigenvalues.

$$\begin{aligned}
\lambda_1 &= -\beta_1 I_c^\star, && (11)\\
\lambda_2 &= A - \beta_1 S_c^\star, && (12)\\
\lambda_3 &= -G, && (13)\\
\lambda_4 &= -L, && (14)\\
\lambda_5 &= M, && (15)\\
\lambda_6 &= P, && (16)\\
\lambda_7 &= PF. && (17)
\end{aligned}$$

The terms used above are classified by the following,

$$\begin{aligned}
A &= \beta_1 S_c^\star - (d_{dI_c} + d_{nI_c}) - \beta_2 I_p^\star,\\
B &= \beta_2 I_c^\star - (d_{dI_p} + d_{nI_p}) - \beta_3 H_h^\star,\\
C &= \beta_3 I_p^\star - (d_{dH_h} + d_{nH_h}) - \beta_4 F_m^\star,\\
D &= \beta_4 H_h^\star - (d_{dF_m} + d_{nF_m}) - \beta_5 P_c^\star,\\
E &= \beta_5 F_m^\star - (d_{dP_c} + d_{nP_c}) - \beta_6 C_c^\star,\\
F &= \beta_6 C_c^\star - (d_{dC_c} + d_{nC_c}),\\
G &= -(\beta_2^2 I_c^\star I_p^\star + B(A - \beta_1 S_c^\star)), && (18)\\
L &= -(GC + \beta_3^2 I_p^\star H_h^\star),\\
K &= G\beta_4 H_h^\star - (A - \beta_1 S_c^\star)\beta_3 H_h^\star,\\
M &= -DL - K\beta_4 F_m^\star,\\
N &= L\beta_5 F_m^\star,\\
P &= ME - N\beta_5 P_c^\star,\\
R &= -M\beta_6 P_c^\star.
\end{aligned}$$

From above equations it is clear that equations (11) and (12), that is, λ_1 and λ_2 are negative eigne values. Also equation (13) $\lambda_3 < 0$ iff $\beta_3 < 1$. Using equation (14) $\lambda_4 < 0$ iff $\beta_4 < 0$. Taking equation (15) $\lambda_5 < 0$ if $\beta_2 < 1$. Also from equation (16) $\lambda_6 < 0$ iff $\beta_2 < 1$ and $\beta_3 < 0$. Now taking equation (17) $\lambda_7 < 0$ iff $\beta_2 < 1$, with $\beta_3 < 0$ and $(d_{dC_c} + d_{nC_c}) < 1$. All the values of model (1) are negative eigne values, so model (1) is asymptotically stable at endemic equilibrium.

5. 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* [26, 27] is an interesting and easy rule to study stability analysis. Many authors [25, 27] use this technique for the same job in there work. Now for global stability analysis of model (1) we define the following *Lyapunov function* for stability and also we have the following stability results which are stated as,

Global Stability Analysis of The Model At Disease Free Equilibrium

Theorem 5.1. *Model* (1) *at disease free equilibrium is said globally asymptotically stable, if* $R_0 \leq 1$, *at* $S_c = S_c0$ *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*,

$$P(S_c, dI_c, dI_p, dH_h, dF_m, dP_c, dC_c) = \frac{1}{2}[S_c - S_c^\star + I_c - I_c^\star + I_p - I_p^\star + H_h - H_h^\star + F_m - F_m^\star + P_c - P_c^\star + C_c - C_c^\star]^2 \tag{19}$$

We see clearly that the above function $P(S_c, dI_c, dI_p, dH_h, dF_m, dP_c, dC_c) \geq 0$, and it also equal to zero if and only if $S_c = S_c^\star, I_c = I_c^\star, I_p = I_p^\star, H_h = H_h^\star, F_m = F_m^\star, P_c = P_c^\star, C_c = C_c^\star$. Now to show the required result, let us differentiate equation (19) *with respect to t*, we get,

$$\frac{dP}{dt}(S_c, dI_c, dI_p, dH_h, dF_m, dP_c, dC_c) = (S_c - S_c^\star + I_c - I_c^\star + I_p - I_p^\star + H_h - H_h^\star + F_m - F_m^\star + P_c - P_c^\star + C_c - C_c^\star)(\frac{dS_c}{dt} + \frac{dI_c}{dt} + \frac{dI_p}{dt} + \frac{dH_h}{dt} + \frac{dF_m}{dt} + \frac{dP_c}{dt} + \frac{dC_c}{dt}). \tag{20}$$

Using values from model (1), then equation (20) becomes,

$$\frac{dP}{dt}(S_c, dI_c, dI_p, dH_h, dF_m, dP_c, dC_c) = (S_c - S_c^\star + I_c - I_c^\star + I_p - I_p^\star + H_h - H_h^\star + F_m - F_m^\star + P_c - P_c^\star + C_c - C_c^\star)[\mu_s - (d_n S_c + (d_{dI_c} + d_{nI_c})I_c + (d_{dI_p} + d_{nI_p})I_p + (d_{dH_h} + d_{nH_h})H_h + (d_{dF_m} + d_{dF_m})F_m + (d_{dp_c} + d_{nP_c})P_c + (d_{dP_c} + d_{nP_c})C_c)].$$

From above it is clear that $\frac{dP}{dt}(S_c, dI_c, dI_p, dH_h, dF_m, dP_c, dC_c) = 0$ if and only if $S_c = S_c^\star, I_c = I_c^\star, I_p = I_p^\star, H_h = H_h^\star, F_m = F_m^\star, P_c = P_c^\star, C_c = C_c^\star$, further it also be clear that
$\frac{dP}{dt}(S_c, dI_c, dI_p, dH_h, dF_m, dP_c, dC_c) < 0$ for $\mu_s < K$, where $K = (d_n S_c + (d_{dI_c} + d_{nI_c})I_c + (d_{dI_p} + d_{nI_p})I_p + (d_{dH_h} + d_{nH_h})H_h + (d_{dF_m} + d_{dF_m})F_m + (d_{dp_c} + d_{nP_c})P_c + (d_{dP_c} + d_{nP_c})C_c)]$.
Which show that the global asymptotically stability at disease free equilibrium is stable, which complete the proof.

Global Stability Analysis of The Model At Endemic Equilibrium

Theorem 5.2. *Endemic equilibrium for model* (1) *is* globally asymptotically stable *if* $R_0 > 1$, *if* $S_c = S_c^\star$, $I_c = I_c^\star$, $I_p = I_p^\star$, $H_h = H_h^\star$, $F_m = F_m^\star$, $P_c = P_c^\star$, $C_c = C_c^\star$ *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 (1), considered the following *Lyapunov function*,

$$R(S_c, dI_c, dI_p, dH_h, dF_m, dP_c, dC_c) = \frac{1}{2}(S_c - S_c^\star)^2 + \frac{1}{2}(I_c - I_c^\star)^2 + \frac{1}{2}(I_p - I_p^\star)^2 + \frac{1}{2}(H_h - H_h^\star)^2 + \frac{1}{2}(F_m - F_m^\star)^2 + \frac{1}{2}(P_c - P_c^\star) + \frac{1}{2}(C_c - C_c^\star)^2.$$

We see the above define function, $R(S_c, dI_c, dI_p, dH_h, dF_m, dP_c, dC_c) \geq 0$, and its equal to zero, if and only if $S_c = S_c^\star$, $I_c = I_c^\star$, $I_p = I_p^\star$, $H_h = H_h^\star$, $F_m = F_m^\star$, $P_c = P_c^\star$, $C_c = C_c^\star$,now Differentiating the above equation *with respect to* "t" we have,

$$\frac{dR}{dt}(S_c, dI_c, dI_p, dH_h, dF_m, dP_c, dC_c) = [(S_c - S_c^\star) + (I_c - I_c^\star) + (I_p - I_p^\star) + (H_h - H_h^\star) + (F_m - F_m^\star) + (P_c - P_c^\star) + (C_c - C_c^\star)][\mu_s - (d_n S_c + (d_{dI_c} + d_{nI_c})I_c + (d_{dI_p} + d_{nI_p})I_p + (d_{dH_h} + d_{nH_h})H_h + (d_{dF_m} + d_{dF_m})F_m + (d_{dp_c} + d_{nP_c})P_c + (d_{dP_c} + d_{nP_c})C_c)],$$

After some simplification we get the following,

$$\frac{dR}{dt}(S_c, dI_c, dI_p, dH_h, dF_m, dP_c, dC_c) = -[(S_c - S_c^\star) + (I_c - I_c^\star) + (I_p - I_p^\star) + (H_h - H_h^\star) + (F_m - F_m^\star) + (P_c - P_c^\star) + (C_c - C_c^\star)](M - \mu_s) \tag{21}$$

Where the value of "M" is given by,

$$M = d_n S_c + (d_{dI_c} + d_{nI_c})I_c + (d_{dI_p} + d_{nI_p})I_p + (d_{dH_h} + d_{nH_h})H_h + (d_{dF_m} + d_{dF_m})F_m \\ +(d_{dp_c} + d_{nP_c})P_c + (d_{dP_c} + d_{nP_c})C_c)].$$

From equation (21) it is clear that $\frac{dR}{dt}(S_c, dI_c, dI_p, dH_h, dF_m, dP_c, dC_c) = 0$ if $S_c = S_c^\star$, $I_c = I_c^\star$, $I_p = I_p^\star$, $H_h = H_h^\star$, $F_m = F_m^\star$, $P_c = P_c^\star$, $C_c = C_c^\star$ and also equation (21) $\frac{dR}{dt}(S_c, dI_c, dI_p, dH_h, dF_m, dP_c, dC_c) < 0$ if and only if $M < \mu_s$. From above the model (1) is globally asymptotically stable at endemic equilibria which is required.

6. 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[28,29] 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 $S_c(t)$, infected camel population $I_c(t)$, infected camel to infect human population $I_p(t)$, Infected individual to infect healthy population $H_h(t)$, infected individual to, infect family member population $F_m(t)$, family member to clinic centre population $P_c(t)$ and clinic centre to care centre population $C_c(t)$ 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 (8), while Fig(1) represents combine behaviour of all population. Only susceptible camels Fig (2) after some time reach to zero showing no infection in population. Fig (3) showing sharply decreasing in behaviour mean, effectiveness occur after some time rapidly in this class. Figs (4) and (6) initially increasing and then show rapid decreasing in behaviour, while Figs (5), (7) and (8) are increasing, showing some infection exist in these classes rapidly.

Figures .

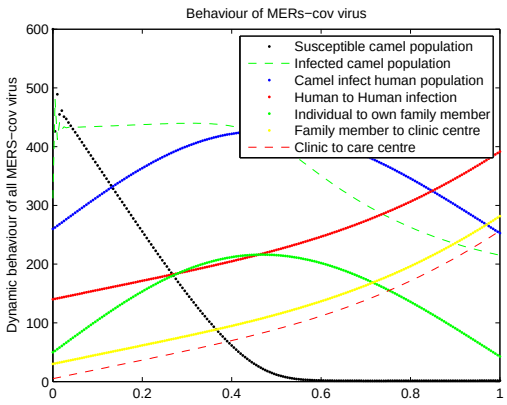

Figure 1: The plot represents combine time dynamics of all categories population.

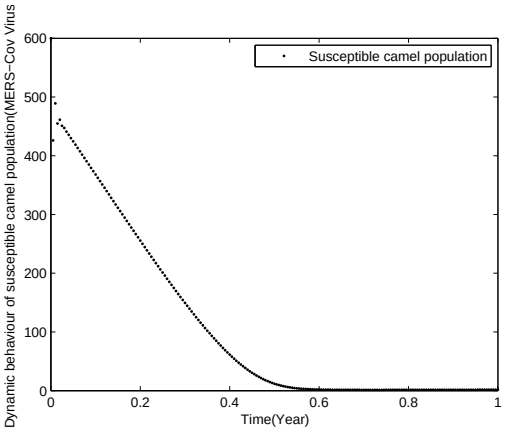

Figure 2: The plot represents the time dynamics of the susceptible camel population.

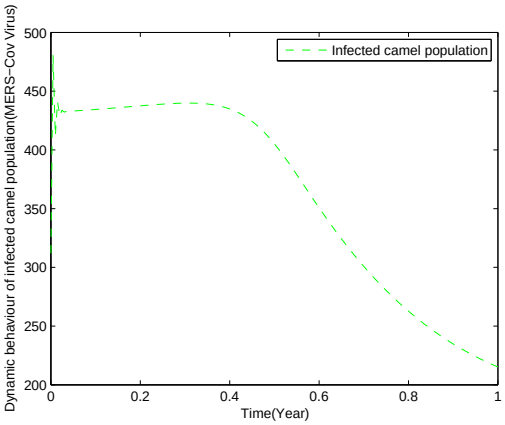

Figure 3: The plot represents the time dynamics of infected camel population.

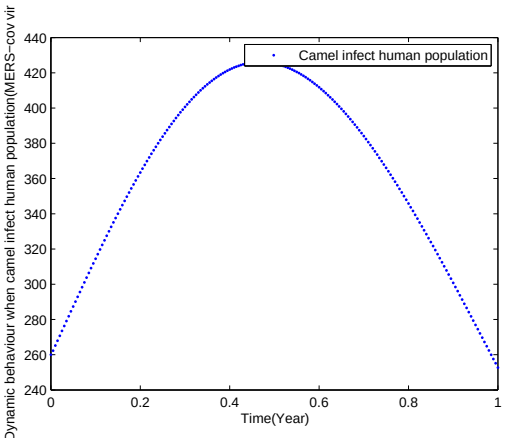

Figure 4: The plot represents the time dynamics of infected camel to infect human population.

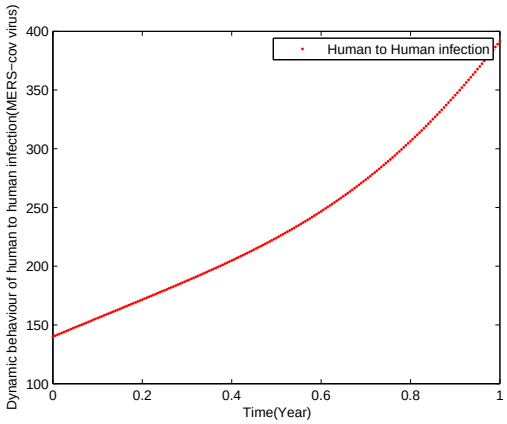

Figure 5: The plot show time dynamics of infected individual to healthy population.

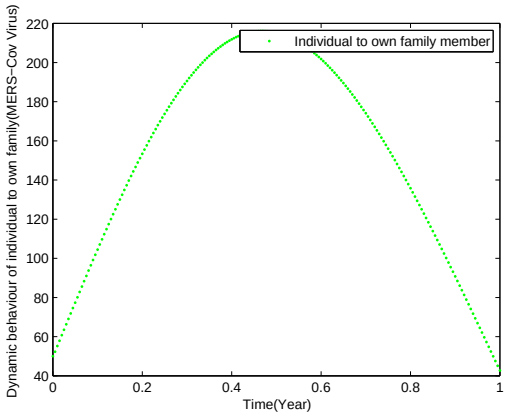

Figure 6: The plot represents the time dynamics of individual to family member population.

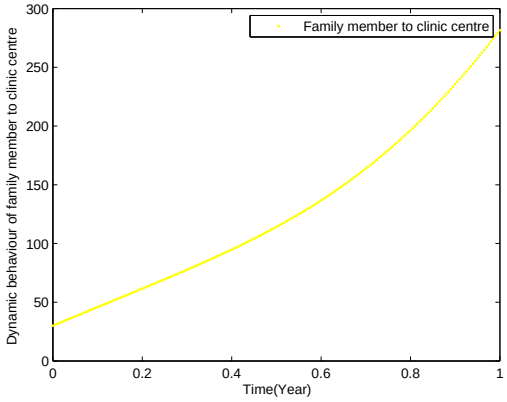

Figure 7: The plot represents the time dynamics of family member to clinic centre population.

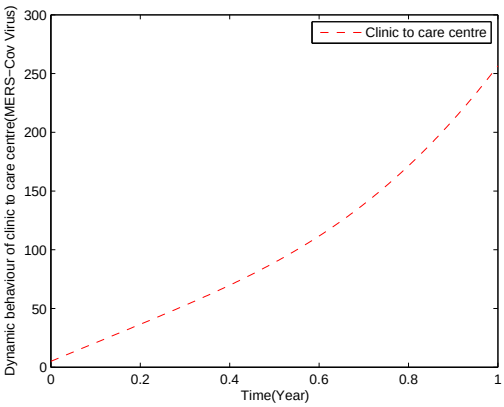

Figure 8: The plot represents the time dynamics of clinic centre to care centre population.

Notation	Parameter	Value
$Z(t)$	Total population	00-600
S_c	Susceptible camel population	00-600
I_c	Infected camel population	200-500
I_p	Infected camel to infect human population	240-440
H_h	Infected individual to healthy(human to human)population	100-400
F_m	Individual to own family member population	40-220
P_c	Family member to clinic centre population	00-300
C_c	Clinic centre to care centre population	00-300
μ_s	New birth rate in susceptible camels population	1.5000
$d_n s_c$	Natural death rate in susceptible camels population	1.7000
d_{nI_c}	Infectious death rate in infected camels population	0.0143
d_{dI_c}	Natural death rate in infected camels population	0.1340
d_{nI_p}	Infectious death rate of infected camels to infect human population	0.3002
d_{dI_p}	Natural death rate of infected camels population to infect human population	0.1343
d_{nH_h}	Infectious death rate of infected individual to healthy individual(human to human)	0.0054
d_{dH_h}	Natural death rate of infected individual to healthy individual(human to human)	0.0024
d_{nF_m}	Infectious death rate of individual to infect own family member	0.0019
d_{dF_m}	Natural death rate of individual to infect own family member	0.0074
d_{nP_c}	Infectious death rate of family member to clinic center	0.0640
d_{dP_c}	Natural death rate of family member to clinic center	0.3440
d_{nC_c}	Infectious death rate of clinic patient to care center	0.4400
d_{dc_c}	Natural death rate of clinic patient to care center	0.5410
β_1	Transmission rate from susceptible camels population to infected camel population	1.2300
β_2	Transmission rate of infected camels population to infect human population	0.1000
β_3	Transmission rate of infected individual to healthy individual(human to human)	0.0060
β_4	Transmission rate of infected individual to own family member	1.0090
β_5	Transmission rate of infected family member to clinic centre	0.0040
β_6	Transmission rate of clinic centre individual to care center	0.0900

7. Conclusion

In this article, we have established a model for the transmission dynamic of MERS-CoV by taking into account the classification of different phases of its spread in population. We presented different mathematical analysis including, biological feasibility, positivity and equilibrium analysis of the proposed model. For that order we obtained the basic reproduction number by using *next generation matrix approach* and then discussed its feasibility region. Moreover, we derived all the endemic equilibriums points and discussed the stability analysis and showed that the model is locally, as well as, globally asymptotically stable for the disease free equilibria and for endemic equilibria. The global stability is retrieved by using *Lyapunov function* theory approach. Finally, the numerical simulation and sensitivity analysis are presented to show the feasibility of the proposed model.

In future, we will consider the proposed model with septic and spatial effect. We will also design the optimal control strategy and will be reported in a near future publication very soon.

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