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ON MULTI-STRAIN FRACTIONAL ORDER MERS-COV MODEL

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Abstract. Mers-Cov is an important epidemic. Its behavior in the Middle East in Saudi Arabia differs significantly from that in Korea. Consequently we propose a new fractional order multi-strain model studying the possibility of its spread.

1. Introduction

It is known that the transmission route of MERS-Cov in Saudi Arabia depends on Camels as an animal source of the virus (animal to human transmission). The outbreak in the republic of Korea didn't need camels (human to human transmission) so, we expect that MERS-Cov may be a multi-strain disease. MERS-Cov outbreak occurred in 27 countries, so it has the property of the small world network [1]. It is of the same family as common cold so, it can be represented by SIS model. Hence the equations of Moreno et al (2014) are valid. We will generalize their results to the fractional order case. In sec.2 a brief introduction to Mers-Cov is given. In sec.3 multi-strain diseases are studied. In sec.4 the model is given. Conclusions are given in sec.5.

2. Middle East Respiratory Syndrome (MERS-Cov)

Many diseases ranging from common cold, flu and even severe acute respiratory syndrome (SARS) occur because of a type of viruses called Corona viruses. Middle East respiratory syndrome (MERS-Cov) is considered one of the viral respiratory diseases which occur by a novel type of Coronaviruses family named MERS-Cov. The first appearance of the new type happened in the Middle East, especially in Saudi Arabia in 2012. This virus is classified among the zoonotic viruses which are transmitted from animals to humans [2]. The death rate of infected patients is estimated to be approximately 36%.

The transmission route has two ways either from animal to human or from human to human. Camels are the largest animal source of the virus, which helps spread the infection to humans. We are also not fully learned about the manner of spread from animal to human. On the other hand, the transmission of the virus from one person to another is not done in an easy way, except in the case of close contact

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and the absence of unprotected care to an infected patient. In Korea, the death rate of infected patients was 17%.

The first appearance of MERS-Cov was found in the Arabian Peninsula, where most of the cases are present (85%), especially in Saudi Arabia. Many other cases outside the Middle East believed to have occurred in the Middle East and then moved outside the region. This outbreak occurred in 27 countries, so it has the property of the small world network. The largest outbreak outside the Middle East happened in the republic of Korea without sustained human to human transmission.

3. Multi-Strains for Infected Diseases

Frequently, more than one pathogen species helps to form infections contrary popular belief that pathogens are uniform entities. It was observed that multi-strain infections appears in 51 human pathogens and is expected to arise in most pathogen species. A few notable cases in most multi-strain diseases are beginning to be considered despite much work has been done on infections with multi pathogen species. Due to the evolution of pathogens and the consequences of multi-strain infections there is a great theoretical interest. There are technical challenges that hinder the efficient distinction between strains elements among many disease agents. Because of this, we find it difficult to study the effects of multi-strain infections in terms of vaccination, treatment, pathogenicity and the dynamics of the disease. This is because experimental investigations to study these effects are limited because of these challenges [3].

4. The Multi-Strain Model for MERS-Cov

This epidemiological model describes the dynamics of the spread of two infectious diseases. In this model we will express the host population that is exposed to the two diseases with an epidemiological network in which the two diseases are spread at the same time. The status of the infected individual, whether with one or two diseases, affects the rates of infectiousness and recovery (dynamical parameters). We also consider that the contact networks through which each disease is spread are different from each other, in addition to the difference in the evolution mechanisms of each disease. Each node belonging to the population network can be found in four different statuses: susceptible for both diseases SS, infected by both diseases II , infected with disease 1 and still susceptible to disease 2 IS and infected with disease 2 and still susceptible to disease 1 SI. These quantities represent the proportion of individuals at each disease status so, we have that $SS + IS + SI + II = 1$. Our network is divided into two different parts of contacts according to the spread of the diseases [4].

Now, we will introduce our model in the fractional order formalism because of its usefulness to predict the outbreak of diseases. Also, fractional order formalism naturally contains both memory and non-locality effects which contribute significantly to study epidemic models. By using Caputo definition for fractional derivative of order $\alpha \in (n-1, n)$ of a function $f(t)$ which is defined by [5]

$$D_*^{\alpha} f(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t \frac{f^{(n)}(s)}{(t-s)^{\alpha+1-n}} ds. \tag{4.1}$$

We get the following model:

$$\begin{aligned}
D_*^\alpha SS &= -k_0\lambda_1 SS\ IS - l_0\lambda_2 SS\ SI - k_0\beta_1^b\lambda_1 SS\ II - l_0\beta_2^b\lambda_2 SS\ II \\
&\quad +\mu_1 IS + \mu_2 SI \\
D_*^\alpha IS &= k_0\lambda_1 SS\ IS + k_0\beta_1^b\lambda_1 SS\ II - l_0\beta_2^a\lambda_2 SI\ IS - l_0\beta_2^a\beta_2^b\lambda_2 IS\ II \\
&\quad -\mu_1 IS + \eta_2\mu_2 II \\
D_*^\alpha SI &= l_0\lambda_2 SS\ SI + l_0\beta_2^b\lambda_2 SS\ II - k_0\beta_1^a\lambda_1 IS\ SI - k_0\beta_1^a\beta_1^b\lambda_1 SI\ II \\
&\quad -\mu_2 SI + \eta_1\mu_1 II \\
D_*^\alpha II &= k_0\beta_1^a\lambda_1 IS\ SI + l_0\beta_2^b\lambda_2 SI\ IS + k_0\beta_1^a\beta_1^b\lambda_1 SI\ II + l_0\beta_2^a\beta_2^b\lambda_2 IS\ II \\
&\quad -(\eta_1\mu_1 + \eta_2\mu_2)\, II, \qquad (4.2)
\end{aligned}$$

where SS means susceptible for both strains, SI means susceptible for the first and infective for the second and so on. The domain of our variables is

$$\Psi = \left\{(SS, IS, SI, II) \in R^4 : SS, IS, SI, II \geq 0\right\},$$

the value of $\alpha \in (0, 1]$ and all other parameters have positive values. The following table illustrates the description of used parameters.

λ_1	Disease 1 infectiousness
λ_2	Disease 2 infectiousness
μ_1	Disease 1 recovery rate
μ_2	Disease 2 recovery rate
β_1^a	Represent the variation of disease (1) infectiousness when the uninfected person with the first disease and infected with the second disease communicates with infected person with the first disease.
β_2^a	Represent the variation of disease (2) infectiousness when the uninfected person with the second disease and infected with the first disease communicates with infected person with the second disease.
β_1^b	when the person who will transmit the first disease is infected with the second disease.
β_2^b	Represent the variation of disease (2) infectiousness when the person who will transmit the second disease is infected with the first disease.
η_1	Represent the variation of disease (1) recovery rate when the person who is recovering from the first disease is infected with the second disease.
η_2	Represent the variation of disease (2) recovery rate when the person who is recovering from the second disease is infected with the first disease.
k_0	Mean degree of disease 1 while propagates over network.
l_0	Mean degree of disease 2 while propagates over the network.

Table 1 the description of used parameters

5. Equilibrium Points and Stability

To calculate the equilibrium points from model (4.2), let

$$\begin{cases} D_*^\alpha SS = 0, \\ D_*^\alpha IS = 0, \\ D_*^\alpha SI = 0, \\ D_*^\alpha II = 0, \end{cases} \tag{5.1}$$

then, we get three disease-free equilibrium points which are equal to

$$\begin{aligned} E_1 &= (1,0,0,0), E_2 = \left(\frac{\mu_1}{\lambda_1 k_0}, \frac{\lambda_1 k_0 - \mu_1}{\lambda_1 k_0}, 0, 0\right) \text{ and} \\ E_3 &= \left(\frac{\mu_2}{\lambda_2 l_0}, 0, \frac{\lambda_2 l_0 - \mu_2}{\lambda_2 l_0}, 0\right). \end{aligned} \tag{5.2}$$

To simplify the studying of stability to the three disease-free equilibrium points we will reduce the dimension of model (4.2) by using

$$SS + IS + SI + II = 1, \tag{5.3}$$

to get another 3-dimensional model consists of three equations in the following form

$$\begin{aligned} D_*^\alpha IS &= (k_0\lambda_1 - \mu_1) IS - (k_0\lambda_1 + l_0\beta_2^a\lambda_2) SI\ IS \\ &\quad - \left(k_0\lambda_1 + k_0\beta_1^b\lambda_1 + l_0\beta_2^a\beta_2^b\lambda_2\right) IS\ II + \left(k_0\beta_1^b\lambda_1 + \eta_2\mu_2\right) II \\ &\quad - k_0\beta_1^b\lambda_1 SI\ II - k_0\lambda_1 (IS)^2 - k_0\beta_1^b\lambda_1 (II)^2 \\ D_*^\alpha SI &= (l_0\lambda_2 - \mu_2) SI - (l_0\lambda_2 + k_0\beta_1^a\lambda_1) SI\ IS \\ &\quad - \left(l_0\lambda_2 + l_0\beta_2^b\lambda_2 + k_0\beta_1^a\beta_1^b\lambda_1\right) SI\ II + \left(l_0\beta_2^b\lambda_2 + \eta_1\mu_1\right) II \\ &\quad - l_0\beta_2^b\lambda_2 IS\ II - l_0\lambda_2 (SI)^2 - l_0\beta_2^b\lambda_2 (II)^2 \\ D_*^\alpha II &= \left(k_0\beta_1^a\lambda_1 + l_0\beta_2^b\lambda_2\right) SI\ IS + k_0\beta_1^a\beta_1^b\lambda_1 SI\ II \\ &\quad + l_0\beta_2^a\beta_2^b\lambda_2 IS\ II - (\eta_1\mu_1 + \eta_2\mu_2) II. \end{aligned} \tag{5.4}$$

The eigenvalues of the Jacobian matrix for model (5.4) which is evaluated at E_1 are

$$\sigma_1 = k_0\lambda_1 - \mu_1, \quad \sigma_2 = l_0\lambda_2 - \mu_2, \quad \sigma_3 = -(\eta_1\mu_1 + \eta_2\mu_2) \tag{5.5}$$

Hence E_1 is locally asymptotically stable if $\sigma_{1,2} < 0$, i.e

$$\frac{k_0\lambda_1}{\mu_1} < 1 \quad \text{and} \quad \frac{l_0\lambda_2}{\mu_2} < 1 \tag{5.6}$$

For the second disease-free equilibrium point E_2 , if all the eigenvalues $\sigma_i (i = 1, 2, 3)$ of the Jacobian Matrix of model (5.4) evaluated at E_2 satisfy the following condition

$$|\arg(\sigma_i)| > \frac{\alpha\pi}{2}. \tag{5.7}$$

Then, E_2 is locally asymptotically stable and the same condition is valid for the third disease-free equilibrium point E_3 [6]-[8].

Figure 1 Stability Region for fractional order system

6. Conclusions

This paper discusses the dynamical transmission of two strains of MERS-Cov over a regular network which is divided into two parts according to the spread of the diseases. The model has three equilibrium points E_1 , E_2 and E_3 the first is a disease free state and the other two are endemic. We obtained the generalized stability conditions by the fractional order model for the three disease-free equilibrium points. The new finding of fractional order formulation is that it naturally includes both memory and non-locality properties. Moreover stability regions increase in this formulation as shown in fig.1 where the stability region for the integer order systems is the region $Re(\sigma_i) < 0$. Mers-Cov is nonlocal since it has reached 27 countries. Therefore fractional order formulation is suitable to model it.

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Competing Interests

All authors declare that there are no competing interests regarding the publication of this paper.

Authors contribution:

The first author did the calculations. The idea was proposed by authors two and three. The last author reviewed the paper.

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