SIMULATION OF MATHEMATICAL MODEL OF CORONAVIRUS

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Abstract:

We propose a mathematical model to investigate the current outbreak of the coronavirus disease 2019 (COVID-19). Our model describes the multiple transmission pathways in the infection dynamics, and emphasizes the role of the environmental reservoir in the transmission and spread of this disease. In addition to this, our model employs non-constant transmission rate changes that reflect the effect of continuous diseases control measures with regard to epidemiological condition and environmental conditions. We evaluate this model in depth and use the publicly reported data to explain its application. Our analytical and numerical results, among others, suggest that the coronavirus infection is still widespread and that long-term disease prevention and intervention programs are important. In particular, a new approach takes into account the fraction of observed cases over the actual total infected cases, which allows the influence of this relationship on the effect of COVID-19 to be examined. In addition, the model will estimate bed requirements in clinics. This is difficult enough to define the most significant but also easy effects so that its parameters can be affordably identified using the data on the pandemic published by the authorities. We are analyzing the specific case of the country that spreads the disease and using its reporting data to identify models that can be useful to estimate COVID-19 spreading in other countries (including the Chinese mainland, Macau, Hong Kong and Taiwan, as per the World Health Organization report in its report COVID-19). We show a strong understanding between the data reported and our model's estimates. In addition, when considering incomplete reported data (through cutting it down on some dates prior to and after the maximum number of reports), we analyze the actions of the tests that our model returns. By comparing these effects, the error produced by the model can be calculated by defining the parameters in the early stage of the pandemic. Finally, we research different scenarios, which show how the various values of the percentage of identified cases have altered China's global magnitude of COVID-19 that may be of concern to policymakers, taking account of the advantages of developments implemented by our model.

Keywords: *Novel Corona virus, Mathematical model, Basic reproduction number, Next generation matrix, Transmissibility*

INTRODUCTION

A new case of coronavirus (2019-nCoV), which has been identified as extreme acute breathing Syndrome Coronavirus 2 (SARS-CoV-2) by the International Committee for Virus Taxonomy on 11 February, 2 December 2019, has been notified the World Health Organization (WHO) China country office of pneumonia of unknown etiology (unknown cause) in the City of Wuhan, Hubei, China and the WHO. The virus is reported to originate from bats[2], while viral transmission may relate to an exposure to the wholesale market for seafood (Huanan Seafood Market) [3, 4]. Recently the genetic characteristics and some clinical results have been reported[4–6]. There has been an assessment of potential for international spread through commercial aviation[7]. The number of people contaminated and suspected is compensated globally for public health concerns. A mathematical model is therefore urgent to determine the transmissibility of the virus and its structure. Mathematical modeling was investigated in several ways[3, 8]. These studies were based on the simple reproductive number (R0) measurement using the serial intervals, intrinsic growth rates[3, 9, 10] as well as on the ordinary differential equations and the Monte Carlo Markov Chain[8] methods.

Modeling and simulation are important decision-making tools that can help manage animal and human diseases. As each disease has its own biological characteristics, however, the models must be modified to fit individual circumstances to tackle real situations. Coronovirus 2019 (COVID-19) is an infectious disease that has spread rapidly in China and many countries and originated in December 2019 in China. The World Health Organization (WHO) renamed 2019-nCoV infectious disease a strain of severe acute respiratory syndrome 2 (SARS-CoV-2) on 11 February 2020. This is an entirely new scenario and a new virus. The WHO announced that this was an international emergency for public health on 30 January 2020. The disease has been confirmed in more than 118, 000 cases reported in over 114 countries worldwide since 11 March 2020. More than 90 percent of cases have declined significantly in only four countries (two – China and the Republic of Korea) and WHO has declared this to be a corona virus, the first pandemic. 872481 and 43275 confirmed cases and deaths were reported respectively on 1 April 2020. There is no validated vaccine explicitly developed for this virus. Several mathematical models attempt to explain the evolution dynamics of COVID-19 in the literature. Three phenomenologic models are validated with outbreaks of diseases other than COVID-19 which aim to generate and evaluate short-term cumulative cases predictions. Many works provide SEIR models with minor variations and some have stochastic components. COVID-19 is a new virus disease that causes a worldwide emergency and requires a model that takes into account its established characteristics. In particular, it would be convenient to develop a model which incorporates the following:

• The effect of undetected infected people, being able to show the dependence of the impact of COVID-19 on the percentage of detected cases over the real total infected cases.

• The effect of different sanitary and infectiousness conditions of hospitalized people (differentiating those with mild and severe conditions that will recover from those who will finally die),

• The estimation of the needs of beds in hospitals (which is one of the major problems for policy makers addressing COVID-19).

General description

The model is used to evaluate the spread of a human disease within some territories during a fixed time interval. At the beginning of the simulation, the model parameters are set by the user. We consider as time t = 0 the 1 December 2019 (7 days before the date that appears in the literature as the most probable date for the index case in China. Here we took into account that,according to the World Health Organization's website, the first confirmed COVID-19 case in China was on December 8. Furthermore, according to the earliest symptom onset of confirmed patients can be traced back to 7 December 2019. We set then t = 0 that day at 10AM CET (this is a technical adjustment, since each day the WHO provides data as reported by national authorities).

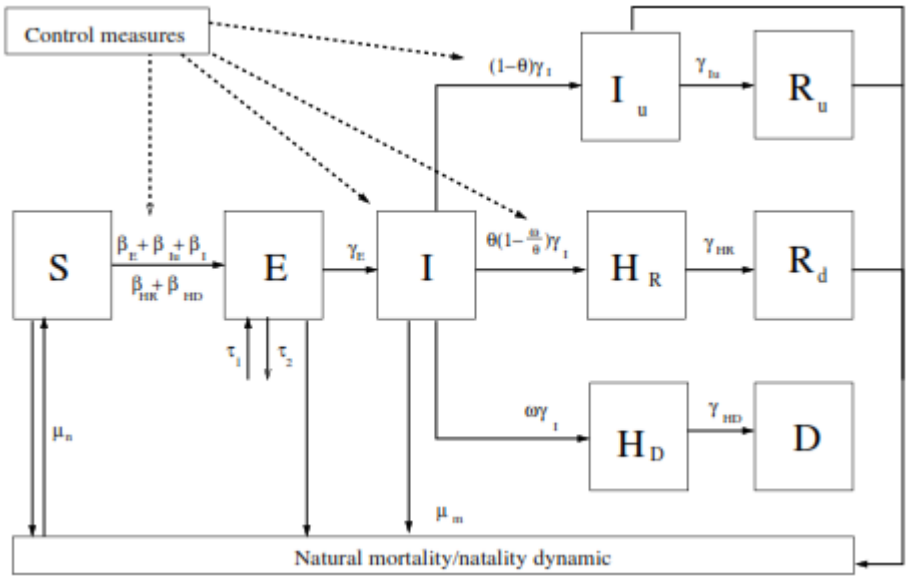

Figure 1: Diagram summarizing the model for COVID-19

We can start our simulation at any initial time $t0 \geq 0$, considering that only susceptible people live in the territories that are free of the disease, whereas the number of people in compartments S, E, I, Iu, HR, HD, Rd, Ru, and D of the infected territories are set to their corresponding values. Then, during the time interval [t0, t0 + Tmax], with Tmax$\in$ IN being Apply regular spreading procedures within the country for the maximum number of simulation days. When the number of people in each compartment E, I, Iu, HR and HD at the end of the simulation day t is less than 1 in all the territories covered, the simulation shall be stopped. Otherwise, when t = t0 + Tmax the simulation is halted. To order to calculate their efficacy and the the severity and duration of a COVID-19 outbreak, control measures are introduced and enabled or disabled at the launch of the model.

Figure 1 provides a diagram that summarizes the principal structure of our whole plan. We will present a streamlined version (which we use for our simulations) with a simplified diagram (see Figure 2) after explaining the complete model. The choice of using a deterministic model rather than a stochastic model is a first solution, as such models have some benefits, for example: a low machine complexity that enables the model parameters to be calibrated more efficiently, or the theory of normal differential equations for the correct analysis and interpretation in the model. In addition, deterministic models should be the first method for modeling a new problem that involves few data according to [5]. The authors also note that stochastic models are not sufficient if the distribution probability is difficult or unable to determine, are difficult to analyze, and require additional data to calibrate the model. As described above, in this work we only consider the spread of diseases within countries in areas where the COVID-19 pandemic is already spreading independently from the correct values of t0 and with a relatively poor reliance on international movement of people. In another job, the spread of cross-country disease is considered.

THE MATHEMATICAL MODEL

A deterministic compartmental model is used for the complex disease distribution through a specific infected territory I. In order to be simple, we assume that the population is homogenously distributed within a territory each time (this can be improved if some territories are divided into a group of smaller areas with similar characteristics). The spatial propagation of the disease within a region is therefore omitted. We always take for granted that new births are vulnerable people. Here we are not talking about people moving between territories. The evolution of these groups is based on the following system of standard differential equations under those assumptions (which is simplified below):

$$\begin{aligned}
\frac{\mathrm{d}S^{(i)}}{\mathrm{d}t}(t) &= -\frac{S^{(i)}(t)}{N^{(i)}}\left(m_E^{(i)}(t)\beta_E^{(i)}E^{(i)}(t) + m_I^{(i)}(t)\beta_I^{(i)}I^{(i)}(t) + m_{I_u}^{(i)}(t)\beta_{I_u}^{(i)}(\theta^{(i)}(t))I_u^{(i)}(t)\right) \\
&\quad -\frac{S^{(i)}(t)}{N^{(i)}}\left(m_{H_R}^{(i)}(t)\beta_{H_R}^{(i)}(t)H_R^{(i)}(t) + m_{H_D}^{(i)}(t)\beta_{H_D}^{(i)}(t)H_D^{(i)}(t)\right) \\
&\quad -\mu_m^{(i)}S^{(i)}(t) + \mu_n^{(i)}\left(S^{(i)}(t) + E^{(i)}(t) + I^{(i)}(t) + I_u^{(i)}(t) + R_d^{(i)}(t) + R_u^{(i)}(t)\right), \\
\frac{\mathrm{d}E^{(i)}}{\mathrm{d}t}(t) &= \frac{S^{(i)}(t)}{N^{(i)}}\left(m_E^{(i)}(t)\beta_E^{(i)}E^{(i)}(t) + m_I^{(i)}(t)\beta_I^{(i)}I^{(i)}(t) + m_{I_u}^{(i)}(t)\beta_{I_u}^{(i)}(\theta^{(i)}(t))I_u^{(i)}(t)\right) \\
&\quad +\frac{S^{(i)}(t)}{N^{(i)}}\left(m_{H_R}^{(i)}(t)\beta_{H_R}^{(i)}(t)H_R^{(i)}(t) + m_{H_D}^{(i)}(t)\beta_{H_D}^{(i)}(t)H_D^{(i)}(t)\right) \\
&\quad -\mu_m^{(i)}E^{(i)}(t) - \gamma_E E^{(i)}(t) + \tau_1^{(i)}(t) - \tau_2^{(i)}(t),
\end{aligned}$$

$$\frac{\mathrm{d}I^{(i)}}{\mathrm{d}t}(t) = \gamma_E E^{(i)}(t) - (\mu_m^{(i)} + \gamma_I^{(i)}(t))I^{(i)}(t), \quad (1)$$

$$\frac{\mathrm{d}I_{\mathrm{u}}^{(i)}}{\mathrm{d}t}(t) = (1-\theta^{(i)}(t))\gamma_I^{(i)}(t)I^{(i)}(t) - (\mu_m^{(i)} + \gamma_{I_{\mathrm{u}}}^{(i)}(t))I_{\mathrm{u}}^{(i)}(t),$$

$$\frac{\mathrm{d}H_R^{(i)}}{\mathrm{d}t}(t) = \theta^{(i)}(t)\left(1 - \frac{\omega^{(i)}(t)}{\theta^{(i)}(t)}\right)\gamma_I^{(i)}(t)I^{(i)}(t) - \gamma_{H_R}^{(i)}(t)H_R^{(i)}(t),$$

$$\frac{\mathrm{d}H_D^{(i)}}{\mathrm{d}t}(t) = \omega^{(i)}(t)\gamma_I^{(i)}(t)I^{(i)}(t) - \gamma_{H_D}^{(i)}(t)H_D^{(i)}(t),$$

$$\frac{\mathrm{d}R_{\mathrm{d}}^{(i)}}{\mathrm{d}t}(t) = \gamma_{H_R}^{(i)}(t)H_R^{(i)}(t) - \mu_m^{(i)}R_{\mathrm{d}}^{(i)}(t),$$

$$\frac{\mathrm{d}R_{\mathrm{u}}^{(i)}}{\mathrm{d}t}(t) = \gamma_{I_{\mathrm{u}}}^{(i)}(t)I_{\mathrm{u}}^{(i)}(t) - \mu_m^{(i)}R_{\mathrm{u}}^{(i)}(t),$$

$$\frac{\mathrm{d}D^{(i)}}{\mathrm{d}t}(t) = \gamma_{H_D}^{(i)}(t)H_D^{(i)}(t),$$

Where:

- $i \in \{1, \ldots, N_C\}$, with $N_C \in \mathbb{N}$ being the number of countries/territories/areas considered.
- $N^{(i)}$ is the number of people in territory i before the start of the pandemic.
- $\mu_n^{(i)} \in [0,1]$ is the natality rate (day^{-1}) in territory i: the number of births per day and per capita.
- $\mu_m^{(i)} \in [0,1]$ is the mortality rate (day^{-1}) in territory i: the number of deaths per day and per capita (or, equivalently, the inverse of the mean life expectancy (day) of a person).
- $\omega^{(i)}(t) \in [\underline{\omega}^{(i)}, \overline{\omega}^{(i)}] \subset [0,1]$ is the case fatality rate in territory i at time t: the proportion of deaths compared to the total number of infectious people (detected or not detected). Here, ω (i) and ω (i) are the minimum and maximum case fatality rates for territory i, respectively.
- $\theta^{(i)}(t) \in [\overline{\omega}^{(i)}, 1]$ is the fraction of infected people that are detected and documented by the authorities in territory i at time t. We are assuming, for the sake of simplicity, that all deaths due to COVID-19 are detected and reported, so that $\theta^{(i)}(t) \geq \overline{\omega}^{(i)}$.
- $\beta_E^{(i)}, \beta_I^{(i)}, \beta_{I_{\mathrm{u}}}^{(i)}, \beta_{H_R}^{(i)}, \beta_{H_D}^{(i)} \in \mathbb{R}^+$ are the disease contact rates (day^{-1}) of a person in the corresponding compartments, in territory i (without taking into account the control measures).
- $\beta_{I_{\mathrm{u}}}^{(i)}(\theta) \in \mathbb{R}^+$ is the disease contact rate (day−1) of a person in the compartment Iu in territory i (without taking into account the control measures), where the fraction of infected people that are detected are θ.
- $\gamma_I^{(i)}(t) \in (0, +\infty)$ is the transition rate (day−1) from compartment E to compartment I. It is the same for all the territories.
- $\gamma_{I_{\mathrm{u}}}^{(i)}(t), \gamma_{H_R}^{(i)}(t)$ and $\gamma_{H_D}^{(i)}(t) \in (0, +\infty)$ denote the transition rates (day^{-1}) from compartments I_{u}, H_R and H_D to compartments R_{u}, R_{d} and D, respectively, in territory i at time t.
- $\gamma_{I_{\mathrm{u}}}^{(i)}(t), \gamma_{H_R}^{(i)}(t)$ and $\gamma_{H_D}^{(i)}(t) \in (0, +\infty)$ denote the transition rates (day^{-1}) from compartments I_{u}, H_R and H_D to compartments R_{u}, R_{d} and D, respectively, in territory i at time t.
- $m_E^{(i)}(t)$, $m_I^{(i)}(t)$, $m_{I_{\mathrm{u}}}^{(i)}(t)$, $m_{H_R}^{(i)}(t)$, $m_{H_D}^{(i)}(t) \in [0,1]$ (%) are functions representing the efficiency of the control measures applied to the corresponding compartments, in territory i at time t.

• τ (i) 1 (t) is the people infected that arrives to territory i from other territories per day. τ (i) 2 (t) is the people infected that leaves territory i per day. Both can be modeled following the between-country spread part of the Be-CoDiS model).

System (1) is completed with initial data $S^{(i)}(t_0)$, $E^{(i)}(t_0)$, $I^{(i)}(t_0)$, $I_u^{(i)}(t_0)$, $H_R^{(i)}(t_0)$, $H_D^{(i)}(t_0)$, $R_d^{(i)}(t_0)$, $R_u^{(i)}(t_0)$ and $D^{(i)}(t_0)$ given in $[0, \infty)$.

We point out that the 9th equation of system (1) is not coupled with the other equations. Thus, we can solve the first eight equations of that system and the solution of the last one can be computed as follows:

$$D^{(i)}(t) = D^{(i)}(t_0) + \int_{t_0}^{t} \gamma_{H_D}^{(i)}(s) H_D^{(i)}(s) \mathrm{d}s. \tag{2}$$

From a modeling point of view, the term ω (i) (t) θ (i)(t) in the 5th equation of system (1) corresponds to the apparent fatality rate of the disease (obtained by considering only detected cases) in territory i at time t, and ω (i) (t) is the real fatality rate of the COVID-19 (taking into account detected and undetected cases).

We may also have considered sub-compartments grouped around age ranges, etc., but this would make the model more complicated and hinder parameter recognition. For example, a different birth rate for infected people (depending on their age distribution) could be used in each compartiment. In addition, as there are no significant factors for COVID-19 (at least for relatively short periods of time), the birth rate and mortality (not from COVID-19) one can find a simplified model with μm = μn = 0. In the remainder of this article, we will find this case. Figure 2, and the resulting system equations, after eliminating the index and indicating different areas, can be seen in the corresponding diagram summarizing the main structure of the simplified model, (for simplicity), which is given in the below system (3).

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t}(t) &= -\frac{S(t)}{N}\Big(m_E(t)\beta_E E(t) + m_I(t)\beta_I I(t) + m_{I_u}(t)\beta_{I_u}(\theta(t))I_u(t)\Big) \\
&\quad -\frac{S(t)}{N}\Big(m_{H_R}(t)\beta_{H_R}(t)H_R(t) + m_{H_D}(t)\beta_{H_D}(t)H_D(t)\Big), \\
\frac{\mathrm{d}E}{\mathrm{d}t}(t) &= \frac{S(t)}{N}\Big(m_E(t)\beta_E E(t) + m_I(t)\beta_I I(t) + m_{I_u}(t)\beta_{I_u}(\theta(t))I_u(t)\Big) \\
&\quad +\frac{S(t)}{N}\Big(m_{H_R}(t)\beta_{H_R}(t)H_R(t) + m_{H_D}(t)\beta_{H_D}(t)H_D(t)\Big) - \gamma_E E(t) + \tau_1(t) - \tau_2(t), \\
\frac{\mathrm{d}I}{\mathrm{d}t}(t) &= \gamma_E E(t) - \gamma_I(t)I(t), \\
\frac{\mathrm{d}I_u}{\mathrm{d}t}(t) &= (1-\theta(t))\gamma_I(t)I(t) - \gamma_{I_u}(t)I_u(t), \\
\frac{\mathrm{d}H_R}{\mathrm{d}t}(t) &= \theta(t)\left(1 - \tfrac{\omega(t)}{\theta(t)}\right)\gamma_I(t)I(t) - \gamma_{H_R}(t)H_R(t), \\
\frac{\mathrm{d}H_D}{\mathrm{d}t}(t) &= \omega(t)\gamma_I(t)I(t) - \gamma_{H_D}(t)H_D(t), \\
\frac{\mathrm{d}R_d}{\mathrm{d}t}(t) &= \gamma_{H_R}(t)H_R(t), \\
\frac{\mathrm{d}R_u}{\mathrm{d}t}(t) &= \gamma_{I_u}(t)I_u(t), \\
\frac{\mathrm{d}D}{\mathrm{d}t}(t) &= \gamma_{H_D}(t)H_D(t).
\end{aligned} \tag{3}$$

We note that the 7th, 8th and 9th method equations (3) are not related to any other equations in such simplification. We can therefore solve the first six equations of this method and determine the solution of the last three equations using (2) and

$$R_d(t) = R_d(t_0) + \int_{t_0}^{t} \gamma_{H_R}(s)H_R(s)\mathrm{d}s,$$

$$R_u(t) = R_u(t_0) + \int_{t_0}^{t} \gamma_{I_u}(s)I_u(s)\mathrm{d}s.$$

The first five system equations (3) were numberally resolved for numerical simulation using the four-stage Runge – Kutta method (RK4) in a classic fourth order with a time-step of 4 hours (which has been checked to ensure that stable results are obtained).

Outputs of the model

Here, we present the outputs of a mathematical model (in territories I to simplify the study of the outcomes of the simulations we tend to avoid using this Index on the notation).

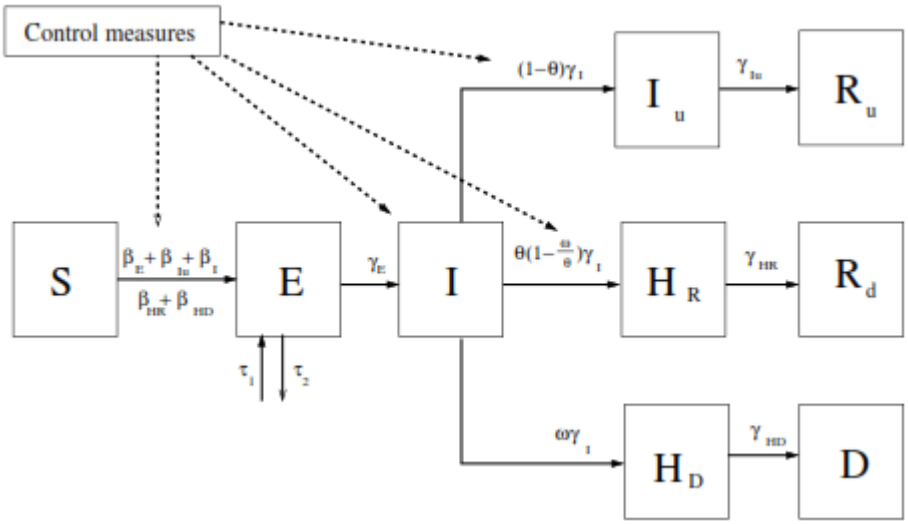

Figure 2: Diagram summarizing the simplified version of the model for COVID-19

- $c_m(t)$: The model cumulative number of COVID-19 cases (in country i) at day t, which is given by

$$c_m(t) = H_R(t) + H_D(t) + R_d(t) + D(t),$$

and can be also computed as follows:

$$c_m(t) = c_m(t_0) + \int_{t_0}^{t} \frac{d\left(H_R + H_D + R_d + D\right)}{dt}(s)ds = c_m(t_0) + \int_{t_0}^{t} \theta(s)\gamma_I(s)I(s)ds.$$

- $d_m(t)$: The model cumulative number of deaths (due to COVID-19), at day t (in territory i), which is given by $D(t)$.

• R_0 and R_e: The basic reproduction number and the effective reproduction number of COVID-19 (for territory i).

The fundamental reproductive number is the number of cases produced by an infected person, on average, in a non-infected population and without special control measures, over their infectious time. It depends on the population considered (so it may vary in different areas), but does not change as the disease spreads.

The efficient reproduction number is calculated by the average number of cases produced by an infected person during its time of infection. Some of the population could already be contaminated and/or special control measures could be applied. It depends on the populations considered and shifts during the disease spread. Furthermore, Re(0) = R0. Typically, the spread of the disease slows down when Re(i, t) < 1. We apply the Next Generation Matrix method to system (3) and obtain that:

$$R_0 = \frac{\left(\left(\left(\beta_{I_u}(1-\theta)\gamma_{H_R} + \beta_{H_R}\gamma_{I_u}(\theta-\omega)\right)\gamma_I + \beta_I\gamma_{H_R}\gamma_{I_u}\right)\gamma_E + \beta_E\gamma_I\gamma_{H_R}\gamma_{I_u}\right)\gamma_{H_D} + \beta_{H_D}\omega\gamma_E\gamma_I\gamma_{H_R}\gamma_{I_u}}{\gamma_E\gamma_I\gamma_{H_D}\gamma_{H_R}\gamma_{I_u}},$$

where, for the sake of simplicity of notation, all previous coefficients correspond to their particular values at time t = 0. Furthermore,

$$R_e(t) = \frac{U_e(t)}{\gamma_E\gamma_I\gamma_{H_D}\gamma_{H_R}\gamma_{I_u}}\frac{S(t)}{N},$$

where

$$\begin{aligned} U_e(t) &= \left(\left(\left(m_{I_u}\beta_{I_u}(1-\theta)\gamma_{H_R} + m_{H_R}\beta_{H_R}\gamma_{I_u}(\theta-\omega)\right)\gamma_I + m_I\beta_I\gamma_{H_R}\gamma_{I_u}\right)\gamma_E + m_E\beta_E\gamma_I\gamma_{H_R}\gamma_{I_u}\right)\gamma_{H_D} \\ &\quad + m_{H_D}\beta_{H_D}\omega\gamma_E\gamma_I\gamma_{H_R}\gamma_{I_u}, \end{aligned}$$

and, again in order to simplify the notation, all previous coefficients correspond to their particular values at time t.

• Hos(t): The number of people in hospital is estimated as follows

$$\mathrm{Hos}(t) = H_D(t) + p(t)\left(H_R(t) + (R_{\mathrm{d}}(t) - R_{\mathrm{d}}(t - C_o))\right),$$

The duration of convalescence (i.e. the time a person is still in hospital after recovery from VOCID-19) shall be where p(t) is the fraction at t time for people in the HR compartment who are hospitalized and Co day. This role will assist in determining and preparing the number of clinical beds needed for all COVID 19 cases.

• MHos: The maximum number of hospitalized people at the same time (in territory i) during the time interval [t0, T]. It is computed as

$$\mathrm{MHos} = \max_{t\in[t_0,T]} \mathrm{Hos}(t).$$

This number can help to estimate and plan the number of clinical beds needed to treat all the COVID-19 cases

- $\Gamma_E(t)$, $\Gamma_{I_u}(t)$ and $\Gamma_H(t)$: The number of people infected during the time interval $[t_0, T]$, by contact with people in compartment E, I_u and $H = H_R + H_D$, respectively. They are given by:

$$\begin{aligned}
\Gamma_E(t) &= \int_{t_0}^{t} m_E(s)\beta_E E(s)\frac{S(s)}{N}\mathrm{d}s,\\
\Gamma_{I_u}(t) &= \int_{t_0}^{t} m_{I_u}(s)\beta_{I_u} I_u(s)\frac{S(s)}{N}\mathrm{d}s,\\
\Gamma_H(t) &= \int_{t_0}^{t} \left(m_{H_R}(s)\beta_{H_R} H_R(s) + m_{H_D}(s)\beta_{H_D} H_D(s)\right)\frac{S(s)}{N}\mathrm{d}s.
\end{aligned}$$

We point out that cm(t) and dm(t) can be compared with the corresponding values reported by WHO.

MODEL FORMULATION AND ANALYSIS

Formulation

We split the total population into 4 separate parts: the susceptible population (denoted by S), the exposed population (denoted by E), and the infected population (denoted by I) and the recovered population (denoted by R). Persons in the contaminated community have fully developed signs of illness and are able to infect others. Individuals in the exposed class are in the time of incubation; they do not show any symptoms, but may also affect others. So the E and I portions of our model are further described as comprising asymptomatic infected individuals and symptomatic infected individuals.

We introduce the following model to describe the transmission dynamics of the COVID-19 epidemic:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta_E(E)S\,E - \beta_I(I)S\,I - \beta_V(V)S\,V - \mu S,\\
\frac{dE}{dt} &= \beta_E(E)S\,E + \beta_I(I)S\,I + \beta_V(V)S\,V - (\alpha+\mu)E,\\
\frac{dI}{dt} &= \alpha E - (w+\gamma+\mu)I,\\
\frac{dR}{dt} &= \gamma I - \mu R,\\
\frac{dV}{dt} &= \xi_1 E + \xi_2 I - \sigma V,
\end{aligned}$$

Where V is the natural reservoir concentration of coronavirus. μ is the average mortality rate of human hosts; α −1 is the incubation period between infection and the onset of symptoms; w is the death rate caused by the disease; α is the rate of recovery from infection; ±1 and −2 the respective rates of individuals exposed to and infected by the coronavirus contributing to the environmental storage tank; − is the rate of death of the human hosts; μ is the parameter The features βE(E) and β I (I) reflect the direct, human-to-human rate of transmission among the exposed and susceptible persons, and the indirect, environment-to-human transmission rate between the infected and susceptible persons, respectively, and the βV(V) function. We presume that all functions that are not increased are βE(E), βI(1) and βV(V) since higher E, I and V values will encourage greater controls to reduce transmission speeds. Specifically, we make the following assumptions:

(A1) $\beta_E(E), \beta_I(I), \beta_V(V)$ are all positive; and

(A2) $\beta'_E(E) \le 0$, $\beta'_I(I) \le 0$, $\beta'_V(V) \le 0$.

Apparently, systemhas a unique disease-free equilibrium (DFE) at

$$X_0 = (S_0, E_0, I_0, R_0, V_0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right).$$

The infection components in this model are E, I, and V. The new infection matrix F and the transition matrix V are given by

$$F = \begin{bmatrix} \beta_E(0)S_0 & \beta_I(0)S_0 & \beta_V(0)S_0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \alpha+\mu & 0 & 0 \\ -\alpha & w_1 & 0 \\ -\xi_1 & -\xi_2 & \sigma \end{bmatrix},$$

where w1 = w + γ + μ. The basic reproduction number of model is then defined as the spectral radius of the next generation matrix FV−1 [21]; i.e.,

$$\begin{aligned} \mathcal{R}_0 = \rho(FV^{-1}) &= \frac{\beta_E(0)S_0}{\alpha+\mu} + \frac{\alpha\beta_I(0)S_0}{w_1(\alpha+\mu)} + \frac{(w_1\xi_1+\alpha\xi_2)\beta_V(0)S_0}{\sigma w_1(\alpha+\mu)} \\ &:= \mathcal{R}_1 + \mathcal{R}_2 + \mathcal{R}_3, \end{aligned}$$

It offers a chance of disease quantification. The first two parts R1 and R2 assess human-to-human transmission routes (exposed and infected-to-susceptible, respectively) contribution and the third part R3 reflects the environment-to-human transmission route contribution. The cumulative probability of COVID-19 outbreaks is determined by these three modes of transmission.

Equilibrium analysis

They now examine the system's balance, which offers essential information about the disease's long-term dynamics. The following equations (S, E, I, R, V) are a standard equilibrium and therefore reach

$$\begin{aligned} \Lambda - \beta_E(E)SE - \beta_I(I)SI - \beta_V(V)SV - \mu S &= 0, \\ \beta_E(E)SE + \beta_I(I)SI + \beta_V(V)SV - (\alpha+\mu)E &= 0, \\ \alpha E - w_1 I &= 0, \\ \gamma I - \mu R &= 0, \\ \xi_1 E + \xi_2 I - \sigma V &= 0. \end{aligned}$$

Solving yields

$$\begin{aligned} S &= \frac{1}{\mu}(\Lambda - (\alpha+\mu)E), \\ E &= \frac{w_1}{\alpha} I, \\ R &= \frac{\gamma}{\mu} I, \\ V &= \frac{w_1\xi_1 + \alpha\xi_2}{\sigma\alpha} I. \end{aligned}$$

It follows from the first two equations that S can be denoted by a function of I, namely,

$$S = \phi(I) := \frac{1}{\mu}\left(\Lambda - \frac{w_1(\alpha+\mu)}{\alpha} I\right).$$

$$S = \psi(I) := (\alpha+\mu)\left(\beta_E\left(\frac{w_1}{\alpha} I\right) + \frac{\alpha}{w_1}\beta_I(I) + \frac{w_1\xi_1+\alpha\xi_2}{\sigma w_1}\beta_V\left(\frac{w_1\xi_1+\alpha\xi_2}{\sigma\alpha} I\right)\right)^{-1}.$$

Let us now consider curves $S = \phi(I),\ I \geq 0$ and $S = \psi(I),\ I \geq 0$. In particular, the intersections of these two curves in $\mathbb{R}^2_+$ determine the non-DFE equilibria. Clearly, $\phi(I)$ is strictly decreasing, whereas $\psi(I)$ is increasing since $\beta_E\left(\frac{w_1}{\alpha} I\right)$, $\beta_I(I)$, and $\beta_V\left(\frac{w_1\xi_1+\alpha\xi_2}{\sigma\alpha} I\right)$ are positive and decreasing functions of I. Additionally, one can easily verify that $\phi(0) = S_0$, $\phi(I_1) = 0$, where $I_1 = \frac{\alpha\Lambda}{(\alpha+\mu)w_1}$, and

$$\psi(0) = (\alpha+\mu)\left(\beta_E(0) + \frac{\alpha}{w_1}\beta_I(0) + \frac{w_1\xi_1+\alpha\xi_2}{\sigma w_1}\beta_V(0)\right)^{-1} = \frac{S_0}{\mathcal{R}_0}.$$

Thus, we conclude:

(1) If R0 > 1, these two curves have a unique intersection lying in the interior of R 2 + , since ψ(0) <φ(0) and ψ(I1) ≥ ψ(0) > 0 = φ(I1). Furthermore, at this intersection point, yields a unique endemic equilibrium (EE)

$$X_* = (S_*, E_*, I_*, R_*, V_*).$$

(2) If R0 ≤ 1, the two curves have no intersection in the interior of R 2 + as ψ(0) ≥ φ(0).
Therefore, we find that the model admits a unique equilibrium, the DFE X0, if R0 ≤ 1; and it admits two equilibria, the DFE X0 and the EE X∗, if R0 > 1.
In what follows, we perform a study on the global stability of the DFE. By a simple comparison principle, we find that $0 \le S + E + I + R \le S_0$ and $0 \le V \le \frac{(\xi_1+\xi_2)S_0}{\sigma}$. . Thus, it leads to a biologically feasible domain

$$\Omega = \left\{(S, E, I, R, V) \in \mathbb{R}^5_+ : S + E + I + R \le S_0,\ 0 \le V \le \frac{(\xi_1 + \xi_2)S_0}{\sigma}\right\}.$$

Theorem 1. The following statements hold for the model
(1) If R0 ≤ 1, the DFE of system is globally asymptotically stable in Ω.
(2) If R0 > 1, the DFE of system is unstable and there exists a unique endemic equilibrium. Moreover, the disease is uniformly persistent in the interior of Ω, denoted by Ω°; namely, liminf t→∞ (E(t), I(t), V(t)) > (ε, ε, ε) for some ε > 0.
Proof. Let X = (E, I, V)^T. One can verify that

$$\frac{d\mathbf{X}}{dt} \le (F - V)\mathbf{X},$$

Theorem 2. Assume that $\beta_E(E)E$, $\beta_I(I)I$ and $\beta_V(V)V$ are non-decreasing functions of variables E, I and V, respectively. If R_0>1, then the unique endemic equilibrium X∗ of system (2.1) is globally asymptotically stable in Ω° .

Proof. We let $L(y) = \int_{y_*}^{y} \frac{x-y_*}{x}dx$ for $y > 0$, where $y_* > 0$, and y can be replaced by S, E, I, or V. Clearly, L(y) ≥ 0 with the equality holding if and only if y = y∗. Differentiating the four functions L(S), L (E), L (I), L (V) along the solution of system and using the equilibrium equations yield

$$\begin{aligned}
\frac{dL(S)}{dt} =& \frac{S - S_*}{S}\frac{dS}{dt} \le \frac{S - S_*}{S}(\beta_E^* S_* E_* - \beta_E S E + \beta_I^* S_* I_* - \beta_I S I + \beta_V^* S_* V_* - \beta_V S V)\\
=& \beta_E^* E_* S_* \left(1 - \frac{S_*}{S} - \frac{\beta_E E S}{\beta_E^* E_* S_*} + \frac{\beta_E E}{\beta_E^* E_*}\right) + \beta_I^* I_* S_* \left(1 - \frac{S_*}{S} - \frac{\beta_I I S}{\beta_I^* I_* S_*} + \frac{\beta_I I}{\beta_I^* I_*}\right)\\
&+ \beta_V^* V_* S_* \left(1 - \frac{S_*}{S} - \frac{\beta_V V S}{\beta_V^* V_* S_*} + \frac{\beta_V V}{\beta_V^* V_*}\right),
\end{aligned}$$

$$\begin{aligned}
\frac{dL(E)}{dt} =& \frac{E - E_*}{E}\frac{dE}{dt} = \frac{E - E_*}{E}(\beta_E S E + \beta_I S I + \beta_V S V - (\alpha + \mu)E)\\
=& \beta_E^* E_* S_* \left(\frac{\beta_E E S}{\beta_E^* E_* S_*} - \frac{E}{E_*} - \frac{\beta_E S}{\beta_E^* S_*} + 1\right) + \beta_I^* I_* S_* \left(\frac{\beta_I I S}{\beta_I^* I_* S_*} - \frac{E}{E_*} - \frac{\beta_I I S E_*}{\beta_I^* I_* S_* E} + 1\right)\\
&+ \beta_V^* V_* S_* \left(\frac{\beta_V V S}{\beta_V^* V_* S_*} - \frac{E}{E_*} - \frac{\beta_V V S E_*}{\beta_V^* V_* S_* E} + 1\right).
\end{aligned}$$

Hence,

$$\begin{aligned}
&\frac{dL(S)}{dt} + \frac{dL(E)}{dt} \le \beta_E^* E_* S_* \left(2 - \frac{S_*}{S} - \frac{E}{E_*} + \frac{\beta_E E}{\beta_E^* E_*} - \frac{\beta_E S}{\beta_E^* S_*}\right)\\
&+ \beta_I^* I_* S_* \left(2 - \frac{S_*}{S} - \frac{E}{E_*} + \frac{\beta_I I}{\beta_I^* I_*} - \frac{\beta_I I S E_*}{\beta_I^* I_* S_* E}\right)\\
&+ \beta_V^* V_* S_* \left(2 - \frac{S_*}{S} - \frac{E}{E_*} + \frac{\beta_V V}{\beta_V^* V_*} - \frac{\beta_V V S E_*}{\beta_V^* V_* S_* E}\right)\\
\le& \beta_E^* E_* S_* \left(\frac{\beta_E E}{\beta_E^* E_*} - 1\right)\left(1 - \frac{\beta_E^*}{\beta_E}\right)\\
&+ \beta_I^* I_* S_* \left\{\left(\frac{\beta_I I}{\beta_I^* I_*} - 1\right)\left(1 - \frac{\beta_I^*}{\beta_I}\right) + \frac{I}{I_*} - \frac{E}{E_*} - \ln\frac{I}{I_*} + \ln\frac{E}{E_*}\right\}\\
&+ \beta_V^* V_* S_* \left\{\left(\frac{\beta_V V}{\beta_V^* V_*} - 1\right)\left(1 - \frac{\beta_V^*}{\beta_V}\right) + \frac{V}{V_*} - \frac{E}{E_*} - \ln\frac{V}{V_*} + \ln\frac{E}{E_*}\right\}\\
\le& \beta_I^* I_* S_* \left(\frac{I}{I_*} - \frac{E}{E_*} - \ln\frac{I}{I_*} + \ln\frac{E}{E_*}\right) + \beta_V^* V_* S_* \left(\frac{V}{V_*} - \frac{E}{E_*} - \ln\frac{V}{V_*} + \ln\frac{E}{E_*}\right).
\end{aligned}$$

The last inequality follows from the assumptions that $\beta_P(P)$ and $\beta_P(P)P$, where P can represent E, I, or V, are non-increasing and non-decreasing functions of P, respectively. This implies

$$1-\frac{\beta_P^*}{\beta_P}\le 0 \Longleftrightarrow P^*\le P \Longleftrightarrow \frac{\beta_P P}{\beta_P^* P_*}-1\ge 0.$$

Similarly, one can verify that

$$\frac{dL(I)}{dt}=\alpha E_*\left(\frac{E}{E_*}-\frac{I}{I_*}-\frac{I_*E}{IE_*}+1\right)\le \alpha E_*\left(\frac{E}{E_*}-\frac{I}{I_*}+\ln\frac{I}{I_*}-\ln\frac{E}{E_*}\right),$$

$$\begin{aligned}\frac{dL(V)}{dt}&=\xi_1 E_*\left(\frac{E}{E_*}-\frac{V}{V_*}-\frac{V_*E}{VE_*}+1\right)+\xi_2 I_*\left(\frac{I}{I_*}-\frac{V}{V_*}-\frac{V_*I}{VI_*}+1\right)\\&\le \xi_1 E_*\left(\frac{E}{E_*}-\frac{V}{V_*}+\ln\frac{V}{V_*}-\ln\frac{E}{E_*}\right)+\xi_2 I_*\left(\frac{I}{I_*}-\frac{V}{V_*}+\ln\frac{V}{V_*}-\ln\frac{I}{I_*}\right).\end{aligned}$$

Let $c_1=\frac{\beta_I^* I_* S_*}{\alpha E_*}+\frac{\xi_2\beta_V^* V_* S_*}{(w_1\xi_1+\alpha\xi_2)E_*}$ and $c_2=\frac{w_1\beta_V^* V_* S_*}{(w_1\xi_1+\alpha\xi_2)E_*}$. Then, we claim that

$$\mathcal{L}_1=L(S)+L(E)+c_1L(I)+c_2L(V)$$

is a Lyapunov function for system. Clearly, L1 ≥ 0 and

$$\begin{aligned}\frac{d\mathcal{L}}{dt}\le&(\beta_I^* I_* S_*+\beta_V^* V_* S_*-c_1\alpha E_*-c_2\xi_1 E_*)\left(\ln\frac{E}{E_*}-\frac{E}{E_*}\right)\\&+(\beta_I^* I_* S_*-c_1\alpha E_*+c_2\xi_2 I_*)\left(\frac{I}{I_*}-\ln\frac{I}{I_*}\right)\\&+(\beta_V^* V_* S_*-c_2(\xi_1 E_*+\xi_2 I_*))\left(\frac{V}{V_*}-\ln\frac{V}{V_*}\right)\\=&0\end{aligned}$$

with the equality holding if and only if (S, E, I, V) = (S ∗, E∗, I∗, V∗). Thus, one can easily see that the largest invariant set where $\frac{d\mathcal{L}}{dt}=0$ the singleton is $\{X_*=(S_*,E_*,I_*,R_*,V_*)\}$. Therefore, X∗ is globally asymptotically stable in Ω°.

CONCLUSION

Our model shows that SARS-CoV-2's transmissibility may be higher than MERS in the Near East, comparable to SARS, but lower than MERS in the Republic of Korea by measuring the published data. Based on a limited data published in a literature, the objective of this analysis was the provision of a mathmatical model for the transmissibility calculation of SARS-CoV-2. Further information was needed to accurately estimate the transmissibility. Since our model includes so many parameters, this study includes many limitations. First of all, instead of using data from the literature, we did not use the detailed data of SARS-CoV-2 for the assessment. We predicted the natural history of the infection that 50 percent of asymptomatic infections and half of symptomatic infections that were other than MERS and SARS were transmissible asymptomatic infections. The rate of asymptomatic MERS and SARS infection was reported to be less than 10%. Second, population mobility parameters were not based on a specific data set. Thirdly, as the initial prevalence of the seafood virus on the market has not yet been published, we assumed the initial 1/100 000 value. This assumption may lead to an underestimation or overestimation of the simulation. Furthermore, since we did not take account of the evolving rate of operation of the person (e.g. wear masks, increase social distance and not traveling to the town of Wuhan), it may not be right to estimate the export rate. All of these constraints will lead to our findings becoming unpredictable. Therefore, if the model fits first-hand data on population dynamics and data on natural history, epidemiological characteristics and the transmission mechanism of the virus the accuracy and validity of the prediction is stronger. We have also conducted a review on the effect of the percentage of cases identified and we find that when this proportion is increased the severity of the epidemic can be drastically reduced. This finding could be used as a guideline to the COVID-19 in all countries.

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