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## STABILITY OF A MATHEMATICAL MODEL DESCRIBING THE COVID-19 PANDEMIC IN TERMS OF THE ENVIRONMENT, SOCIAL DISTANCING AND REINFECTION

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**Abstract.** In this paper, we propose and study the stability of a mathematical model for the Covid-19 pandemic incorporating the environment, social distancing and reinfection. A numerical example is carried out to consider the obtained results. *Keywords:* stability, SIR model, disease-free-equilibrium point, the Covid-19 pandemic.

## 1. Introduction

The Covid-19 pandemic is an infectious disease caused by the SARS-CoV-2 virus, which is taking place in the world. For effective disease prevention and control measures, one needs to describe the pandemic with a mathematical model, such as using ordinary differential equations or partial differential equations. By using mathematical techniques such as statistics, stability analysis, combined with numerical methods, theoretical results will be obtained. These mathematical results allow estimating, evaluate and forecast the phenomenon.

The SIR model is a basic mathematical model of the pandemic, introduced in the classic paper of W.O. Kermack & A.G. McKendrick [1]. In this model, the population is divided into three groups, based on the status of the disease: (1) those who are likely to have the disease **S**usceptible, (2) those who are **I**nfected and can infect others, and (3) those who have recovered, are no longer able to get the disease, (**R**emoved or **R**ecovered). In this model, a person's state can only go from S to I (infected), or from I to R (recover or die, but cannot be reinfected).

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When Covid-19 broke out and became very complicated, mathematics was used in an attempt to model and predict the pandemic, etc. Scientists have built numerous models for Covid-19 (see, for example, [2, 3] and on international databases such as MathSciNet). Among these efforts, we are interested in the work of Mwalili [4]. This paper has described Covid-19 by a system of ordinary differential equations, shown the existence of a disease-free-equilibrium point and analyzed the data on MatLab.

A notable feature in Mwalili [4] (see a system of ordinary differential equations (2.1)) is that this mathematical model does not reflect the reinfection of recovered individuals. It could be good for diseases that are permanently immune (such as chickenpox, plague). But unfortunately, new developments of Covid-19 indicate that this disease has had a *reinfection*. Therefore, model (2.1) of Mwalili [4] needs to be improved to describe the spread of the Covid-19 pandemic.

We propose a new model from model (2.1) of Mwalili [4] by adding the quantity  $+\xi R$  (see disease transmission diagram in Figure 2 and model (\*) below). This addend represents that an individual who has recovered from Covid-19 can be reinfected at a rate of  $\xi$ . The next contribution of the paper is to provide a condition for the asymptotic stability of the disease-free-equilibrium point of the model under consideration.

### 2. The mathematical model

Mwalili [4] considers two interacting populations: (1) the human population as hosts and (2) pathogens. The model subdivides the total human population size at time t denoted as N(t) into susceptible S(t), exposed E(t), asymptomatic infectious  $I_A(t)$ , symptomatic infectious  $I_S(t)$  and the recovered as R(t). The pathogen in the environment) is denoted as P(t). Hence, for the human population we have N(t) = $S(t) + E(t) + I_A(t) + I_S(t) + R(t)$ .

Studies have shown that the virus can be transmitted in two ways, namely: from human to human and from environment to human. The epidemic data indicates that both asymptomatic  $I_A(t)$  and symptomatic  $I_S(t)$  infected individuals spread the Covid-19 virus to susceptible persons S(t) with whom they are in close contact. In addition, when infected individuals sneeze or cough, without taking the necessary precautions, the virus spreads to the environment they are in. Since the pathogen P(t) is known to survive in the environment for some days, susceptible individuals S(t) in close contact to this environment are likely to get exposed to these pathogens, especially in the early days of the Covid-19 outbreak before hygiene protocols are implemented. In the process of disease spread, the susceptible individual moves to the exposed population E(t) since the host has an incubation period Chen [5]. The exposed individual moves to either asymptomatic  $I_A(t)$  or symptomatic  $I_S(t)$  infectious population. P(t) is the number or quantity of pathogens present during the interaction of human beings at time t. The majority of infectious individuals recover and move to the recovered human population R(t).

The compartmental model that describes the interaction between human populations and pathogens in the environment is shown in Figure 1. Mwalili [4] has described the Covid-19 disease by a system of nonlinear ordinary differential equations with the parameters given in Table 1.



Figure 1. SEIR-P model of COVID-19 transmission (Source: Mwalili [4])

$$\begin{cases} \frac{dS}{dt} = b - \frac{\beta_1 SP}{1 + \alpha_1 P} - \frac{\beta_2 S(I_A + I_S)}{1 + \alpha_2 (I_A + I_S)} + \psi E - \mu S, \\ \frac{dE}{dt} = \frac{\beta_1 SP}{1 + \alpha_1 P} + \frac{\beta_2 S(I_A + I_S)}{1 + \alpha_2 (I_A + I_S)} - \psi E - \mu E - \omega E, \\ \frac{dI_A}{dt} = (1 - \delta) \omega E - (\mu + \sigma) I_A - \gamma_A I_A, \\ \frac{dI_S}{dt} = \delta \omega E - (\mu + \sigma) I_S - \gamma_S I_S, \\ \frac{dR}{dt} = \gamma_S I_S + \gamma_A I_A - \mu R, \\ \frac{dP}{dt} = \eta_A I_A + \eta_S I_S - \mu_P P. \end{cases}$$

$$(2.1)$$

The human population is born into the susceptible population at a rate b. The terms  $\beta_1 SP$  and  $\beta_2 S(I_A + I_S)$  describes the rate at which susceptible individuals S(t)

get infectious by pathogens in the environment, P(t) and from infectious humans  $I_A(t)$ and  $I_S(t)$ . Health experts and governments have been advising people, during this outbreak, to minimize contact with infectious individuals through social distancing. Therefore in our model we propose to have new infections occur in the form  $\frac{\beta_1 SP}{1+\alpha_2 (I_A+I_S)}$  and  $\frac{\beta_2 S(I_A+I_S)}{1+\alpha_2 (I_A+I_S)}$  respectively, where the interaction proportions  $\alpha_1$  and  $\alpha_2$  denotes reciprocal of the frequency with which susceptible individuals gets infected with Covid-19 from the environment and from infectious individuals, respectively.

Model parameter name	Symbol	Value
Birth rate of the human population	b	$0.00018 \ { m days}^{-1}$
Natural human death rate	$\mu$	$27375^{-1} \mathrm{days}^{-1}$
Human life expectancy	<u>1</u> //	27375 days (or 75
	μ.	years)
Natural death rate of pathogens in the environment	$\mu_P$	$0.1724 \text{ days}^{-1}$
Life expectancy of pathogens in the environment	$\frac{1}{\mu_P}$	5.8 days
Proportion of interaction with an infectious	$\alpha_1$	0.2
environment		
Proportion of interaction with an infectious	$\alpha_2$	0.10
individual		
Rate of transmission from S to E due to contact	$\beta_1$	0.00414
with P		
Rate of transmission from S to E due to contact	$\beta_2$	0.0115
with $I_A$ and/or $I_S$		
Proportion of symptomatic infectious people	δ	0.7
Progression rate from E back to S due to robust	$\psi$	0.0051
immune system		
Progression rate from $E$ to either $I_A$ or $I_S$	ω	0.09
Death rate due to the coronavirus	$\sigma$	0.0018
Rate of recovery of the symptomatic population	$\gamma_S$	$0.05 \text{ days}^{-1}$ or
		$\frac{1}{20 \text{ days}}$
Rate of recovery of the asymptomatic human	$\gamma_A$	$0.0714 \text{ days}^{-1}$
population		
Rate of virus spread to environment by	$\eta_S$	0.1 days <sup>-1</sup> or $\frac{1}{10 \text{ days}}$
symptomatic infectious individuals		10 days
Rate of virus spread to environment by	$\eta_A$	$0.05 \text{ days}^{-1}$ or
asymptomatic infectious individuals		$\frac{1}{20 \text{ days}}$

#### Table 1. Description of model parameters

The model (2.1) in Mwalili [4] doesn't describe reinfection. The diagram of the disease is shown in Figure 2. This paper proposes a mathematical model that describes the *reinfection* as follows:

$$\begin{aligned}
\frac{dS}{dt} &= b - \frac{\beta_1 SP}{1 + \alpha_1 P} - \frac{\beta_2 S(I_A + I_S)}{1 + \alpha_2 (I_A + I_S)} + \psi E - \mu S + \xi R, \\
\frac{dE}{dt} &= \frac{\beta_1 SP}{1 + \alpha_1 P} + \frac{\beta_2 S(I_A + I_S)}{1 + \alpha_2 (I_A + I_S)} - \psi E - \mu E - \omega E, \\
\frac{dI_A}{dt} &= (1 - \delta) \omega E - (\mu + \sigma) I_A - \gamma_A I_A, \\
\frac{dI_S}{dt} &= \delta \omega E - (\mu + \sigma) I_S - \gamma_S I_S, \\
\frac{dR}{dt} &= \gamma_S I_S + \gamma_A I_A - \mu R - \xi R, \\
\frac{dP}{dt} &= \eta_A I_A + \eta_S I_S - \mu_P P.
\end{aligned}$$
(\*)

In this model, thereinfection is described by terms  $+\xi R$  in the first equation and terms  $-\xi R$  in the fifth equation of model (\*).



Figure 2. SEIR-P model of COVID-19 transmission

# 3. Stability analysis of the mathematical model

The purpose of this section is to provide a condition for the disease-free-equilibrium point of the model  $(\star)$  to be asymptotic stability.

Putting

$$C_1 = \psi + \mu + \omega, \quad C_2 = \mu + \sigma + \gamma_S, \quad C_3 = \mu + \sigma + \gamma_A \tag{3.1}$$

$$p = \mu_P + C_1 + C_2 + C_3 \tag{3.2}$$

$$q = \frac{\beta_2 b \delta \omega (2 - \delta)}{\mu} - \mu_P (C_1 + C_2 + C_3) - C_2 (C_1 + \mu + \delta + \gamma_A) - C_1 C_3, \quad (3.3)$$

$$r = \frac{\beta_1 b\omega}{\mu} (\eta_A - \eta_A \delta + \eta_S \delta) + \frac{\beta_2 b\delta\omega}{\mu} (\mu_P + C_3) - \mu_P C_2 (C_1 + \mu + \delta + \eta_A) - C_1 C_3 (\mu_P + C_2) + \frac{\beta_2 b\omega}{\mu} (1 - \delta) (C_2 + \mu_P),$$
(3.4)

$$h = \frac{\beta_1 b \omega \eta_A (1 - \delta)}{\mu} (C_2) + \frac{\beta_1 b \eta_S \delta \omega}{\mu} (C_3) + \frac{\beta_2 b \delta \omega \mu_P}{\mu} (C_3) - \mu_P C_1 C_2 C_3 + \frac{\beta_2 b \omega \mu_P (1 - \delta)}{\mu} (C_2).$$
(3.5)

The main result of this paper is to state the asymptotic stability of the disease-free-equilibrium point  $\bar{\mathbf{x}} = \left(\frac{b}{\mu}, 0, 0, 0, 0, 0\right)$  as follows:

**Theorem 3.1.** If the constants p, q, r, and h satisfy the following conditions

$$h > 0, \quad pq - r > 0, \quad (pq - r)r - p^2h > 0,$$
 (3.6)

*then the disease-free-equilibrium point of the model* (\*) *is asymptotically stable.* 

*Proof.* First of all, the model (\*) takes the form of ordinary differential equation  $\frac{d\mathbf{x}(t)}{dt} = \mathbf{F}(\mathbf{x})$  in space  $\mathbb{R}^6$  with the setting

$$\mathbf{x} := \begin{bmatrix} S\\ E\\ I_A\\ I_S\\ P\\ \end{bmatrix}, \quad \mathbf{F}(\mathbf{x}) := \begin{bmatrix} b - \frac{\beta_1 SP}{1+\alpha_1 P} - \frac{\beta_2 S(I_A+I_S)}{1+\alpha_2 (I_A+I_S)} + \psi E - \mu S + \mu R + \xi R\\ \frac{\beta_1 SP}{1+\alpha_1 P} + \frac{\beta_2 S(I_A+I_S)}{1+\alpha_2 (I_A+I_S)} - \psi E - \mu E - \omega E\\ (1-\delta)\omega E - (\mu+\sigma)I_A - \gamma_A I_A\\ \delta\omega E - (\mu+\sigma)I_S - \gamma_S I_S\\ \gamma_S I_S + \gamma_A I_A - \mu R - \xi R\\ \eta_A I_A + \eta_S I_S - \mu_P P \end{bmatrix}.$$

Then, the Jacobi matrix  $D\mathbf{F}(\mathbf{x})$  of the mapping  $\mathbf{F}$  is

$$\begin{bmatrix} \frac{-\beta_1 P}{1+\alpha_1 P} - \frac{\beta_2 (I_A + I_S)}{1+\alpha_2 (I_A + I_S)} - \mu & \psi & \frac{-\beta_2 S}{[1+\alpha_2 (I_A + I_S)]^2} & \frac{-\beta_2 S}{[1+\alpha_2 (I_A + I_S)]^2} & \mu + \xi & \frac{-\beta_1 S}{(1+\alpha_1 P)^2} \\ \frac{\beta_1 P}{1+\alpha_1 P} + \frac{\beta_2 (I_A + I_S)}{1+\alpha_2 (I_A + I_S)} & -\psi - \mu - \omega & \frac{\beta_2 S}{[1+\alpha_2 (I_A + I_S)]^2} & \frac{\beta_2 S}{[1+\alpha_2 (I_A + I_S)]^2} & 0 & \frac{\beta_1 S}{(1+\alpha_1 P)^2} \\ 0 & (1-\delta)\omega & -(\mu + \sigma) - \gamma_A & 0 & 0 & 0 \\ 0 & \delta\omega & 0 & -(\mu + \sigma) - \gamma_S & 0 & 0 \\ 0 & 0 & \gamma_A & \gamma_S & -\mu - \xi & 0 \\ 0 & 0 & \eta_A & \eta_S & 0 & -\mu_P \end{bmatrix}$$

$$(3.7)$$

At the equilibrium point  $\bar{\mathbf{x}} = (\frac{b}{\mu}, 0, 0, 0, 0, 0)$ , put  $A = D\mathbf{F}(\bar{\mathbf{x}})$ , we have

$$A = \begin{bmatrix} -\mu & \psi & \frac{-\beta_2 b}{\mu} & \frac{-\beta_2 b}{\mu} & \mu + \xi & \frac{-\beta_1 b}{\mu} \\ 0 & -\psi - \mu - \omega & \frac{\beta_2 b}{\mu} & \frac{\beta_2 b}{\mu} & 0 & \frac{\beta_1 b}{\mu} \\ 0 & (1 - \delta)\omega & -(\mu + \sigma) - \gamma_A & 0 & 0 & 0 \\ 0 & \delta\omega & 0 & -(\mu + \sigma) - \gamma_S & 0 & 0 \\ 0 & 0 & \gamma_A & \gamma_S & -\mu - \xi & 0 \\ 0 & 0 & \eta_A & \eta_S & 0 & -\mu_P \end{bmatrix}.$$

Consider the characteristic polynomial  $\mathcal{P}(\lambda) = \det(A - \lambda I)$ ,

$$\begin{split} \det(A-\lambda I) \\ &= \begin{vmatrix} -\mu-\lambda & \psi & \frac{-\beta_2 b}{\mu} & \frac{-\beta_2 b}{\mu} & \mu+\xi & \frac{-\beta_1 b}{\mu} \\ 0 & -\psi-\mu-\omega-\lambda & \frac{\beta_2 b}{\mu} & \frac{\beta_2 b}{\mu} & 0 & \frac{\beta_1 b}{\mu} \\ 0 & (1-\delta)\omega & -(\mu+\sigma)-\gamma_A-\lambda & 0 & 0 & 0 \\ 0 & \delta\omega & 0 & -(\mu+\sigma)-\gamma_S-\lambda & 0 & 0 \\ 0 & 0 & \gamma_A & \gamma_S & -\mu-\xi-\lambda & 0 \\ 0 & 0 & \eta_A & \eta_S & 0 & -\mu_P-\lambda \end{vmatrix} \\ &= \begin{vmatrix} -\mu-\lambda & \psi & \frac{-\beta_2 b}{\mu} & \frac{-\beta_2 b}{\mu} & \mu+\xi & \frac{-\beta_1 b}{\mu} \\ 0 & -C_1-\lambda & \frac{\beta_2 b}{\mu} & \frac{\beta_2 b}{\mu} & 0 & \frac{\beta_1 b}{\mu} \\ 0 & (1-\delta)\omega & -C_3-\lambda & 0 & 0 & 0 \\ 0 & \delta\omega & 0 & -C_2-\lambda & 0 & 0 \\ 0 & 0 & \eta_A & \eta_S & 0 & -\mu_P-\lambda \end{vmatrix} \\ &= (-\mu-\lambda)(-1)^{1+1} \begin{vmatrix} -C_1-\lambda & \frac{\beta_2 b}{\mu} & \frac{\beta_2 b}{\mu} & 0 & \frac{\beta_1 b}{\mu} \\ (1-\delta)\omega & -C_3-\lambda & 0 & 0 & 0 \\ 0 & \eta_A & \eta_S & 0 & -\mu_P-\lambda \end{vmatrix} \\ &= (-\mu-\lambda)(-\mu-\xi-\lambda)(-1)^{4+4} \begin{vmatrix} -C_1-\lambda & \frac{\beta_2 b}{\mu} & \frac{\beta_2 b}{\mu} & \frac{\beta_2 b}{\mu} \\ (1-\delta)\omega & -C_3-\lambda & 0 & 0 \\ 0 & \eta_A & \eta_S & 0 & -\mu_P-\lambda \end{vmatrix} \\ &= (\mu+\lambda)(\mu+\xi+\lambda)\frac{\beta_1 b}{\mu}(-1)^{1+4} \begin{vmatrix} (1-\delta)\omega & -C_3-\lambda & 0 \\ \delta\omega & 0 & -C_2-\lambda \\ 0 & \eta_A & \eta_S \end{vmatrix} \\ &+ (\mu+\lambda)(\mu+\xi+\lambda)(-\mu_P-\lambda)(-1)^{4+4} \begin{vmatrix} -C_1-\lambda & \frac{\beta_2 b}{\mu} & \frac{\beta_2 b}{\mu} \\ (1-\delta)\omega & -C_3-\lambda & 0 \\ \delta\omega & 0 & -C_2-\lambda \end{vmatrix} \\ &= -(\mu+\lambda)(\mu+\xi+\lambda)\frac{\beta_1 b}{\mu}M(\lambda) + (\mu+\lambda)(\mu+\xi+\lambda)(-\mu_P-\lambda)N(\lambda), \end{split}$$

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where

$$M(\lambda) := \begin{vmatrix} (1-\delta)\omega & -C_3 - \lambda & 0\\ \delta\omega & 0 & -C_2 - \lambda\\ 0 & \eta_A & \eta_S \end{vmatrix}, \quad N(\lambda) := \begin{vmatrix} -C_1 - \lambda & \frac{\beta_2 b}{\mu} & \frac{\beta_2 b}{\mu}\\ (1-\delta)\omega & -C_3 - \lambda & 0\\ \delta\omega & 0 & -C_2 - \lambda \end{vmatrix}.$$
(3.8)

We have

$$\begin{split} M(\lambda) &= \left[-C_2 - \lambda\right](-1)^{2+3} \begin{vmatrix} (1-\delta)\omega & -C_3 - \lambda \\ 0 & \eta_A \end{vmatrix} + \eta_S(-1)^{3+3} \begin{vmatrix} (1-\delta)\omega & -C_3 - \lambda \\ \delta\omega & 0 \end{vmatrix} \\ &= (C_2 + \lambda)(1-\delta)\omega\eta_A + \eta_S\delta\omega(C_3 + \lambda), \\ N(\lambda) &= \frac{\beta_2 b}{\mu}(-1)^{1+3} \begin{vmatrix} (1-\delta)\omega & -C_3 - \lambda \\ \delta\omega & 0 \end{vmatrix} + \left[-C_2 - \lambda\right](-1)^{3+3} \begin{vmatrix} -C_1 - \lambda & \frac{\beta_2 b}{\mu} \\ (1-\delta)\omega & -C_3 - \lambda \end{vmatrix} \\ &= \frac{\beta_2 b}{\mu}\delta\omega(C_3 + \lambda) - (C_2 + \lambda) \left[ (C_1 + \lambda)(C_3 + \lambda) - \frac{\beta_2 b}{\mu}(1-\delta)\omega \right] \\ &= \frac{\beta_2 b}{\mu}\delta\omega(C_3 + \lambda) - (C_2 + \lambda)(C_1 + \lambda)(C_3 + \lambda) + (C_2 + \lambda)\frac{\beta_2 b}{\mu}(1-\delta)\omega. \end{split}$$

Thus,

$$\mathcal{P}(\lambda) = -(\mu + \lambda)(\mu + \xi + \lambda)\frac{\beta_1 b}{\mu}M(\lambda) + (\mu + \lambda)(\mu + \xi + \lambda)(-\mu_P - \lambda)N(\lambda)$$
$$= -(\mu + \lambda)(\mu + \xi + \lambda)\left[\frac{\beta_1 b}{\mu}M(\lambda) + (\mu_P + \lambda)N(\lambda)\right]$$
$$= -(\mu + \lambda)(\mu + \xi + \lambda)Q(\lambda),$$

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where

$$Q(\lambda) := \lambda^4 + p\lambda^3 + q\lambda^2 + r\lambda + h = 0, \qquad (3.9)$$

with the coefficients determined by (3.2), (3.3), (3.4), and (3.5).

Consider the equation  $\mathcal{P}(\lambda) = 0$ . We have  $\lambda = -\mu$ , or  $\lambda = -\mu - \xi$ , or  $\lambda$  is the roots of the algebraic equation  $Q(\lambda) = 0$ . Roots  $\lambda = -\mu$  and  $\lambda = -\mu - \xi$  are negative real numbers. Now, if the polynomial  $Q(\lambda)$  has roots with negative real parts, then according to the Principle of Linearized Stability in the Lyapunov Stability Theory, the equilibrium point  $\bar{\mathbf{x}}$  is asymptotically stable. Using the Routh-Hurwitz stability criterion for the polynomial  $Q(\lambda)$ , we get the conditions (3.6) (see Dingyü Xue [6, Example 7.4]). The proof is complete.

#### Remark 3.1.

1. Notice that, when  $\xi = 0$ , the model ( $\star$ ) will become the model (2.1) of Mwalili [4]. Therefore, from the proof of Theorem 3.1 we also get a condition for the asymptotic stability of the disease-free-equilibrium in Mwalili [4].

2. If the conditions (3.6) are satisfied then the graph of the solutions to the model ( $\star$ ) will be asymptotic to the *t*-axis. In particular, the graph of the function R(t) tends to the *t*-axis, which means that the number of people recovering from the disease will decrease. That is undesirable.

With the data in Table 1, we have p = 0.2912095891, h = 0.001141672667 pq - r = -0.01756288846 < 0,  $(pq - r)r - p^2h = -0.001977178664 < 0$ . We see that condition (3.6) is violated. In general, with a real data set, we need to analyze the evolution of the epidemic so that we can make estimates and predictions.

## 4. Numerical examples

This section presents some numerical examples to analyze the mathematical model under consideration. We use the values of the parameter given in Table 1, the reinfection parameter is  $\xi = 0.005$  (data is taken in 6 months according to [7]) and with the initial values S(0) = 93000, E(0) = 1000,  $I_A(0) = 50$ ,  $I_S(0) = 50$ , R(0) = 0, P(0) = 500.

Figure 3a depicts the epidemic during the first 90 days (equivalent to 3 months). During the first 10 days, the number of susceptible (S) sharply decreased, meaning the number of exposed (E) increased sharply due to exposure to asymptomatic infectious individuals  $I_A$  and symptomatic infectious  $I_S$ . Given that symptomatic infectious  $I_S$  individuals are more contagious than asymptomatic infectious  $I_A$ , the spread of Covid-19 through contact in households, workplaces, schools, public places will increase. This is depicted by the graph of the virus in P(t) medium from day 10 to 30. Notice that the curves  $I_A$ ,  $I_S$  and P get the peak about 20-25 days after the onset of the epidemic.



\* Change the parameter  $\alpha_2$ 

Figure 3. Developments of the Covid-19 pandemic with the change of the parameter  $\alpha_2$ 

The parameter  $\alpha_2$  describes the interaction between an individual and another infected individual.

For the model (\*), we increase  $\alpha_2$  by four times (meaning that susceptible individuals reduce contact with other infectious individuals four times), the graph obtained in Figure 3b shows the number of infected cases (E) also peaked around day 15 and the number was only about 25000 (compared to about 41000 in Figure 3a). At the same time, the peak value of the individual graph of asymptomatic infectious  $I_A$  and symptomatic infectious  $I_S$  is also peaked at a lower value.

Now, we will continue to increase  $\alpha_2 = 0.6$  and assume an individual can be reinfected after 1 year, i.e.  $\xi = 1/365$ . The disease evolution depicted in Figure 3d. Figure 3b shows that the number of cases (E) also peaks around on the 15th day with a quantity of 21000 and decreased immediately after the 20th day.

#### \* Change the parameter $\eta_A$ and $\eta_S$

The parameter  $\eta_A$  and  $\eta_S$  are the rates of viral spread to the environment of asymptomatic infectious and symptomatic infectious individuals, respectively.

Figure 4b depicts the parameters  $\eta_A$  and  $\eta_S$  tripple reduction,  $\eta_A = \frac{0.05}{3}$  and  $\eta_S = \frac{0.1}{3}$ . When reduced by three times, the pathogens in the P(t) [black] medium were reduced by about three times compared to the baseline.

Figure 4d depicts the parameters  $\eta_A$  and  $\eta_S$  increases three times,  $\eta_A = 3 \cdot 0.05 = 0.15$  and  $\eta_S = 3 \cdot 0.1 = 0.3$ . Observing the graph, we can see that pathogens in the environment P(t) increase about three times compared to the original. The P(t) graph peaked at about 45000 on day 25.



Figure 4. Developments of the Covid-19 pandemic with the change of the parameter  $\eta_A$  and  $\eta_S$ 

#### \* Change the parameter $\gamma_A$ and $\gamma_S$

The parameters  $\gamma_A$  and  $\gamma_S$  respectively are the recovery rates of asymptomatic infectious and symptomatic infectious individuals.

Observe the fifth differential equation in the model (\*), parameters  $\gamma_A$  and  $\gamma_S$  influence the recovery rate of recovered individuals R(t).

Figure 5b depicts the evolution of the epidemic when parameters  $\gamma_A$  and  $\gamma_S$  decrease three times. Looking at Figure 5b, we can see that the graph of the number of people with asymptomatic infectious  $I_A(t)$  and those with symptomatic infectious  $I_S(t)$  has increased by about one and a half. At the same time, the number of people recovering R(t) also decreased.

Figure 5d depicts the evolution of the epidemic when parameters  $\gamma_A$  and  $\gamma_S$  increase three times. Looking at Figure 5b, we see that the graph of the number of people with asymptomatic infectious  $I_A(t)$  and those with symptomatic infectious  $I_S(t)$  is reduced by about half. At the same time, the number of people recovering R(t) also increased and remained stable from day 50.

From the graph, we also see that, if the disease is well controlled, after about 1.5 months, the pandemic situation will be considered safe. And over 90 days period (about 3 months), the graph also shows that the number of people getting R reinstated increased. The above analysis leads to the conclusion that, if the isolated and environmental factors are ensured, the pandemic will be well controlled. In other words, the model ( $\star$ ) describes the impact of social distancing on the pandemic well.



Figure 5. Developments of the Covid-19 pandemic with the change of the parameter  $\gamma_A$  and  $\gamma_S$ 

Stability of a mathematical model describing the Covid-19 pandemic in terms of...

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