



INVESTIGATION ON SIMPLE MODELLING SPATIO-TEMPORAL WITH MOBILITY FOR CASE COVID-19 TRANSMISSION

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ABSTRACT

One of the factors that influences disease transmission is mobility. The increase in the frequency and reach of infectious disease epidemics is largely due to human mobility in the form of travel between cities and countries. One of the transmission of disease due to mobility is COVID-19. COVID-19 is caused by Coronavirus 2 (SARS-CoV-2) severe acute respiratory syndrome infection. Transmission of this disease can occur through direct contact with infected hosts. This transmission factor plays an important role in determining the occurrence of epidemics in a specific area, and even the transmission of disease in this area is influenced by the surrounding area due to mobility activities. In this study, we focus on discussing the COVID-19 transmission model. First, we discuss systems of time-dependent ordinary differential equations by dividing the population into four compartments: susceptible, exposed, symptomatic infectious, and asymptomatic infectious. The analysis of the model takes the form of determining the basic reproduction number, the existence of equilibrium points, and the stability of each equilibrium point. Sensitivity analysis is also carried out to see the parameters that affect the solution of this model. The transmission rate parameter affects the changes of the susceptible compartment with a negative relationship. Then, sensitivity analysis explains the positive relationship between transmission rate parameter changes and exposed compartments, symptoms, and asymptomatics. This model is modified to a partial differential equation. The effect of mobility, as a diffusion equation, affects the transmission from one region to another. Numerical simulations were given with two different diffusion coefficients, namely 0.0001 and 0.00005. The results showed that the spread of the disease transmission was faster with a diffusion coefficient of 0.0001 compared to 0.00005.

Keywords: Diffusion coefficient, SEIA Model, COVID-19, Partial Differential Equation, Mobility

1 Introduction

Since early 2020, the Coronavirus Disease 2019 (COVID-19) epidemic had an impact on the entire world. COVID-19 can cause respiratory problems and pneumonia. Clinical symptoms that appear include symptoms of the common cold (cough, runny nose, sore throat, muscle aches, headaches) to serious complications (pneumonia or sepsis) [1]. On January 30, 2020, the World Health Organization initially identified COVID-19 as a global health danger [2]. On March 11, 2020, it was identified as a pandemic [3]. More than 92 million infections and 2 million fatalities have been caused by the COVID-19 Pandemic since this outbreak [1]. This infection can develop and spread very quickly [4].

Many factors, such as demography, education, underlying medical conditions, and epidemiological features, influence the likelihood of infection and its manifestations [5]. Mobility has a big influence on the transmission of COVID-19 [6][7]. An understanding of human mobility is an important one in the transmission of human diseases with a large geographical scale [8]. The government implemented two main measures to stop the virus's transmission: reducing everyday activities that take place outside the home and limitations on inter-city travel [9]. These measures are predicated on the earlier understanding that human mobility is a major element in the global transmission of infectious diseases [9]. Many studies have been conducted in the literature to examine how COVID-19 affects how people use the transportation system and how the epidemic has altered travel habits [10][11]. The impact of government control measures on the reduction of human mobility, as well as the connection between human mobility patterns and the dire consequences of COVID-19, were studied by Hadjidemetriou et al. in 2020 [12].

Due to the pandemic, scientists have warned that the COVID-19 pandemic could happen again [13]. Therefore, it is necessary to take initial steps to access COVID-19 to facilitate the prevention of infection [14]. To do this, prevention requires understanding that can explain the process of transmission of infection [15]. One of the tools used to understand the phenomenon of COVID-19 transmission is mathematical modeling. Many researchers have developed a mathematical model of COVID-19 transmission, including the SEIR model (susceptible, exposed, infected, recovered) developed by [16], involving the incubation stage of the population before becoming an infectious population, and the viral compartment as a pathogen is considered a vector. This model does not consider waning immunity where the recovery population returns to a susceptible population and has no mobility. Next, the SEIAQFR model is studied by [17] involving the quarantined and fatal compartments. A spatio-temporal model was developed to investigate the spread of COVID-19 with inter-city mobility [18]. A stochastic mathematical model of COVID-19 with age structure was created by [19]. For COVID-19 in China, a simple model SIR was developed by [20], and utilizing the strong correlation between statistical data and numerical model simulation, COVID-19 transmission was predicted for an efficient measure. Not only the transmission of one disease but models of co-infection of COVID-19 with other diseases are also studied by [21].

In mathematical modeling, the transmission of infectious diseases with mobility can be expressed in a system of partial differential equations. The effect of mobility is expressed in the form of [22] diffusion equation. Analysis of diffusion reactions in the *SIR* (Susceptible, Infectious, Recovered) [23] model analyzes the asymptotic behavior of the solution in the spatially in-homogeneous case. In addition to the deterministic model, a simple SIS model is also analyzed in terms of reaction-diffusion [22][24]. The paper in [25] explains the five challenges for spatial models, which are the construction of network models; appropriate scale for intervention; modeling remote interactions; representation of population heterogeneity; and characterizing threshold behavior.

In this study, we have a scenario in which an area has a COVID-19 epidemic. In this study, we divide into several sections, i.e. introduction in Section 1. In Section 2, we adopted a deterministic model of influenza in [26] and performed the analysis of the model consisting of the equilibrium

point of existence, a stability analysis at each equilibrium point, and a basic reproduction number in Section 3. In addition, sensitivity analyzes are carried out in Section 4. In Section 5, the deterministic model in Section 2 is modified to a partial differential equation by adding the mobility factor. This factor is expressed as a diffusion equation. Numerical simulations in this equation are displayed with various diffusion coefficient values.

2 SEIA Model

For the first step, we adopt a deterministic model in [26] without mobility. Population is divided into four compartments: susceptible (S), exposed (E), symptomatic infectious (I), and asymptomatic infectious (A). A susceptible individual can be infected and become an exposed individual with a transmission rate β . In contrast to the model [26], we assume that the symptomatic infectious and asymptomatic infectious populations experience healing and return to being susceptible. In this model, we assume that COVID-19 can be transmitted by asymptomatic or symptomatic individuals with each reduction infection factor q and r .

An exposed individual becomes an infected individual after a period exposed $\frac{1}{\kappa}$ and becomes a symptomatic infected individual with a fraction p . Furthermore, An exposed individual becomes asymptomatic infectious with $1 - p$. The parameters γ and η are the recovery rates of infectious and asymptomatic compartments and back to susceptible, respectively. We assume that the total population is constant with the recruitment rate equal to the death rate, μ . For analysis of numeric simulation, we provide all parameter values in Table 1. Based on the assumption of the model, process infection is shown in Figure 1.

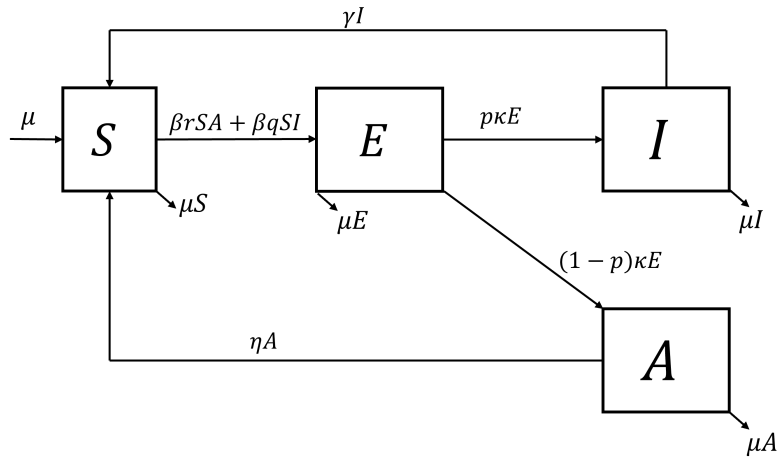


Figure 1: Transmission diagram of COVID-19.

The following system of differential equations that describes the COVID-19 transmission:

$$\begin{aligned}
 \frac{dS}{dt} &= \mu - \beta rSA - \beta qSI + \eta A + \gamma I - \mu S, \\
 \frac{dE}{dt} &= \beta rSA + \beta qSI - \kappa E - \mu E, \\
 \frac{dI}{dt} &= p\kappa E - \gamma I - \mu I, \\
 \frac{dA}{dt} &= (1-p)\kappa E - \eta A - \mu A,
 \end{aligned} \tag{1}$$

with non-negative initial condition $S(0) > 0, E(0) \geq 0, I(0) \geq 0, A(0) \geq 0$. The total population at

time t is denoted by $N(t)$ that is given by

$$N(t) = S(t) + E(t) + I(t) + A(t).$$

3 Dynamical Analysis

We will discuss the existence and local asymptotic stability of each equilibrium. Let

$$\Omega = \{(S, E, I, A) \in \mathbb{R}_+^4 : S + E + I + A = 1\}.$$

Clearly, the set Ω is positively invariant for System (1).

In epidemiology, the average number of secondary cases resulting from a single infection in a population that is fully susceptible is represented by the basic reproductive ratio, or R_0 . Using the method and notations of Van den Driessche [27], we have

$$F = \begin{bmatrix} 0 & \beta q & \beta r \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \mu + \kappa & 0 & 0 \\ -p\kappa & \gamma + \mu & 0 \\ (p-1)\kappa & 0 & \eta + \mu \end{bmatrix}. \quad (2)$$

Based on The Model (1), the basic reproduction number is defined as the spectral radius of the next generation matrix FV^{-1} , i.e.

$$R_0 = \frac{\beta}{(\kappa + \mu)} \left(\frac{r\kappa(1-p)}{\eta + \mu} + \frac{\kappa pq}{\gamma + \mu} \right). \quad (3)$$

In general, R_0 depends on the infectious period, recovery rate, and transmission rate. There are two equilibria of System (1), which we called as a disease-free equilibrium $E_0 = (S^0 = 1, E^0 = 0, I^0 = 0, A^0 = 0)$ and an endemic equilibrium such as

$$E_1 = \left(S^* = \frac{1}{R_0}, E^* = \frac{\eta + \mu}{\kappa(1-p)} A^*, I^* = \frac{\eta + \mu}{\gamma + \mu} \frac{p}{1-p} A^*, A^* = \frac{(1-p)(\gamma + \mu)\kappa}{\eta\kappa p + \kappa\mu + \gamma\kappa(1-p) + (\eta + \mu)(\gamma + \mu)} \frac{R_0 - 1}{R_0} \right)$$

Next, the stability analysis of each equilibrium is presented here. We linearize the System (1) around the disease-free equilibrium E_0 . The Jacobian at E_0 is given by

$$J(E_0) = \begin{bmatrix} -\mu & 0 & -\beta q + \gamma & -\beta r + \eta \\ 0 & -\kappa - \mu & \beta q & \beta r \\ 0 & p\kappa & -a - \mu & 0 \\ 0 & (1-p)\kappa & 0 & -\eta - \mu \end{bmatrix}$$

One of the eigenvalues of $J(E_0)$ is $-\mu < 0$. The remaining three roots of (4) will be analyzed by the cubic equation given as

$$\lambda^3 + k_2\lambda^2 + k_1\lambda + k_0 = 0 \quad (4)$$

where

$$\begin{aligned} k_2 &= \gamma + \eta + \kappa + 3\mu, \\ k_1 &= -\beta(p\kappa q + r\kappa(1-p)) + \gamma\eta + \gamma\kappa + 2\mu\gamma + \eta\kappa + 2\mu\eta + 2\kappa\mu + 3\mu^2, \\ k_0 &= (1-R_0)\mu(\kappa + \mu)(\eta + \mu)(\gamma + \mu). \end{aligned}$$

If $R_0 < 1$, then $k_2 > 0, k_1 > 0, k_0 > 0$ and $k_2 k_1 > k_0$. Thus, according to the Routh-Hurwitz criterion [28], all of the eigenvalues of the characteristic equation (4) have negative real parts. Hence, the disease-free equilibrium E_0 is locally asymptotically stable.

For stability E_1 , linearizing System (1) around endemic equilibrium E_1 give us Jacobian as

$$J(E_1) = \begin{bmatrix} -\frac{(\kappa+\mu)(\eta+\mu)(\gamma+\mu)}{P_2} (1-R_0) - \mu & 0 & -\beta q S^* + \gamma & -\beta r S^* + \eta \\ \beta(rA^* + qI^*) & -\frac{\kappa\beta\mu}{R_0\mu} \left(\frac{pq}{\gamma+\mu} + \frac{r(1-p)}{\eta+\mu} \right) & \beta q S^* & \beta r S^* \\ 0 & p\kappa & -(\gamma + \mu) & 0 \\ 0 & (1-p)\kappa & 0 & -(\eta + \mu) \end{bmatrix}$$

The characteristic polynomial of the above matrix is given by

$$(\lambda + \mu)(K_3\lambda^3 + K_2\lambda^2 + K_1\lambda + K_0) = 0$$

where

$$\begin{aligned} K_3 &= P_1 P_2 \mu \\ K_2 &= \mu P_1^2 \beta + \mu \kappa ((\gamma + 3\mu + \kappa) \gamma \kappa (1-p) + (\eta p + \mu) \kappa^2 + (\eta^2 p + 3\mu \eta p + \gamma \eta + \mu \gamma + \mu \eta + 3\mu^2) \kappa + (\eta + \mu)(\gamma + \mu)(\gamma + \eta + 2\mu)) \\ &\quad ((1-p)(\gamma + \mu)r + (\eta + \mu)pq) \\ K_1 &= \mu(\gamma + \kappa + 2\mu + \eta) P_1^2 \beta + \mu \kappa (1-p)(\gamma + \mu)^2 r ((\eta + \kappa + 2\mu)(-\eta + \gamma) \kappa (1-p) + \gamma \eta^2 + 2\gamma \eta \mu + \gamma \mu^2) + \mu(\eta + \mu)^2 \\ &\quad ((\gamma + \kappa + 2\mu) \kappa^2 p^2 q (\eta - \gamma) - \kappa(\gamma + \mu)^2 (-\eta q) p) \\ K_0 &= P_1 P_2 \mu (1-R_0)(\kappa + \mu)(\eta + \mu)(\gamma + \mu) \\ P_1 &= \frac{\mu}{\beta \mu} R_0 (\kappa + \mu)(\eta + \mu)(\gamma + \mu) \\ P_2 &= \eta \kappa p + \kappa \mu + \gamma \kappa (1-p) + (\eta + \mu)(\gamma + \mu) \end{aligned} \tag{5}$$

It clear that, when $R_0 > 1$ then $\det J(E_1) = \frac{K_0}{K_3} < 0$ and all of diagonal element of matrix $J(E_1)$ are negative. This means all of the eigenvalues have a negative part. Furthermore, by using Routh-Hurwitz criterion, E_1 is locally stable when $R_0 > 1$. So, the endemic equilibrium E_1 is locally asymptotically stable.

Table 1: Biological meaning of parameters

Parameter	Definitions	Value	Source
μ	Natural death and birth rate (per day)	$\frac{1}{365 \times 70}$	[29]
β	Transmission rate of an infected person (per day)	[0, 1]	Assumed
r	Infectivity reduction factor for asymptomatic compartment	[0, 1]	Assumed
q	Infectivity reduction factor for infectious compartment	[0, 1]	Assumed
κ	Progression rate to infectious (per day)	[0, 1]	Assumed
p	Fraction of exposed developing symptoms	[0, 1]	Assumed
γ	Recovery rate for infectious compartment (per day)	$\frac{1}{10}$	[30]
η	Recovery rate for asymptomatic compartment (per day)	$\frac{1}{10}$	[30]

4 Sensitivity Analysis and Numerical Simulation

The concept of sensitivity analysis in [31] is used to investigate the effect of parameters on the solution of Equation 1. In this section, we focus on investigating the effect of the transmission rate probability of an infected person (β), the infectivity reduction factor for the asymptomatic compartment (r), the infectivity reduction factor for the infectious compartment (q), progression rate to infectious (κ), and fraction of exposed developing symptoms (p). A more general approach to sensitivity functions is introduced and computed in [32].

Rewrite System (1) as follows

$$\dot{X} = F(X, P)$$

where $F = \left\{ \frac{dS}{dt}, \frac{dE}{dt}, \frac{dI}{dt}, \frac{dA}{dt} \right\}$, $X = \{S, E, I, A\}$, and $P = \{\beta, q, r, \kappa, p\}$. In this section, we want to analyze the influence of parameter P on variable X .

Let

$$Y = \frac{\partial X}{\partial P}$$

with assumption, X is the solution of System (1) continuous and we obtain the following ODE for the first-order solution sensitivity

$$\frac{dY}{dt} = JY + \frac{\partial F}{\partial P} \quad (6)$$

where J is Jacobian Matrix. In equilibrium point Equation (6) can be found as follows

$$\lim_{t \rightarrow \infty} \dot{Y} = 0 \quad (7)$$

with assumption J matrix non singular, the solution of (7) is

$$Y = -J^{-1} \frac{\partial F}{\partial P}. \quad (8)$$

Furthermore, this matrix Y represents the local change of the states with respect to all parameters at the endemic equilibrium.

In this numerical simulation, we assume the population is constant by normalizing and the rate of birth is equal to the rate of death. Figure 2 shows the numerical solution of System (1) when it is stable in E_1 . This means that the presence of the COVID-19 disease exists with a non-zero proportion of infected asymptomatic and symptomatic compartments when time tends to infinity. Numerical simulations of sensitivity analysis using the set of parameters in Table 1.

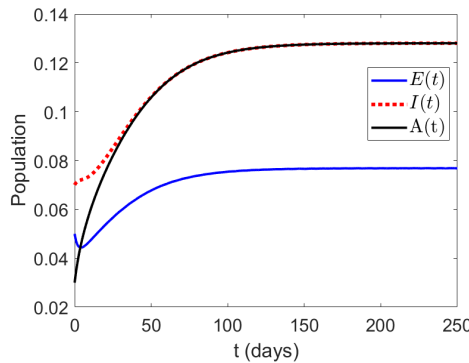


Figure 2: Solution behaviour of $A(t)$, $I(t)$ and $E(t)$ when $R_0 > 1$ with $\mu = \frac{1}{365*70}$, $\beta = 0.3$, $p = 0.5$, $q = 0.5$, $r = 0.5$, $\kappa = \frac{1}{3}$, $\eta = \frac{1}{10}$, $\gamma = \frac{1}{10}$.

Next, sensitivity analysis is done to show the dynamics of the change effect of S, E, I, A with respect to parameter β, κ, q, r , and p in Equation 6. Using parameter $\mu = \frac{1}{365*70}$, $p = 0.5$, $q = 0.5$, $r = 0.5$, $\kappa = \frac{1}{3}$, $\eta = \frac{1}{10}$, $\gamma = \frac{1}{10}$, $\beta = 0.3$ and initial condition as $S(0) = 0.85$, $E(0) = 0.05$, $I(0) = 0.07$, $A(0) = 0.03$, and $Y(0) = \mathbf{0}$ the sensitivity analysis can be seen in Figure 3. Furthermore, using Equation (8), we have the sensitivity

matrix that evaluates at endemic equilibrium as follows

$$\begin{bmatrix} \frac{\partial S}{\partial \beta} & \frac{\partial S}{\partial q} & \frac{\partial S}{\partial r} & \frac{\partial S}{\partial \kappa} & \frac{\partial S}{\partial p} \\ \frac{\partial E}{\partial \beta} & \frac{\partial E}{\partial q} & \frac{\partial E}{\partial r} & \frac{\partial E}{\partial \kappa} & \frac{\partial E}{\partial p} \\ \frac{\partial I}{\partial \beta} & \frac{\partial I}{\partial q} & \frac{\partial I}{\partial r} & \frac{\partial I}{\partial \kappa} & \frac{\partial I}{\partial p} \\ \frac{\partial A}{\partial \beta} & \frac{\partial A}{\partial q} & \frac{\partial A}{\partial r} & \frac{\partial A}{\partial \kappa} & \frac{\partial A}{\partial p} \end{bmatrix} = \begin{bmatrix} -2.22 & -0.67 & -0.67 & -0.0002 & -0 \\ 0.51 & 0.15 & 0.15 & -0.18 & -0 \\ 0.86 & 0.26 & 0.26 & 0.09 & 0.26 \\ 0.86 & 0.26 & 0.26 & 0.09 & -0.26 \end{bmatrix} \quad (9)$$

Figures 3(a), (b), (c), and (d) are the results of simulations on Equation (8) using the data in the Figure Caption (3). The results of the sensitivity analysis when the equilibrium is stable are shown in Figure (3)(a) (d). In Figure (3)(a), the parameter that most dominates the change in behavior of the susceptible compartment is the transmission rate, with a negative influence. This can be seen in the blue curve in Figure (3)(a), which has a negative value over time. This means that the change in the susceptible population decreases as the transmission rate increases. Then, for a very long time, the change in the number of susceptibles to the transmission rate parameter will remain stable at -2.22. On the other hand, the parameter β provides a significant added effect to exposed, symptomatic, infectious, and asymptomatic infectious (see Figure 3(b), (c), and (d).)

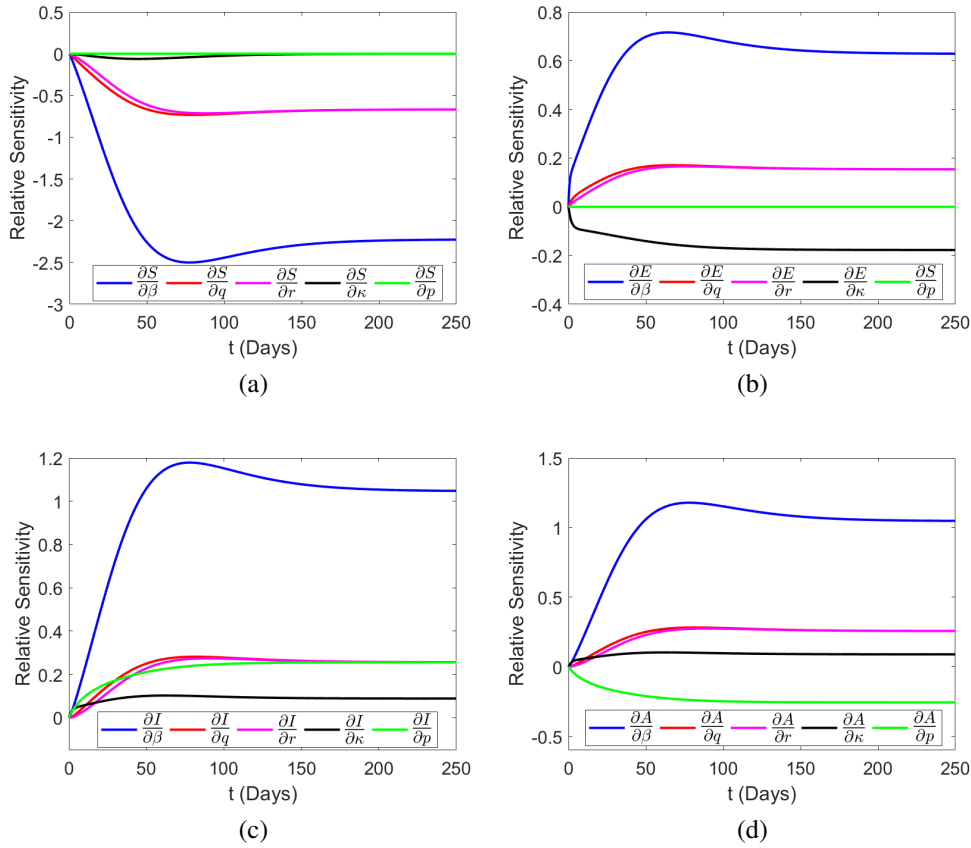


Figure 3: Numerical approximates for the time-dependent sensitivity states (a) S to β, q, r, κ, p , (b) E to β, q, r, κ, p , (c) I to β, q, r, κ, p , and (d) A to β, q, r, κ, p around $\beta = 0.3, p = 0.5, q = 0.5, r = 0.5, \kappa = \frac{1}{3}$.

5 Spatio-Temporal SEIA Model

In this session, we developed a deterministic Model (1) including human movement effects. Assume that the habitat $\Omega \subset \mathbb{R}^2$ is a bounded domain with smooth boundary $\partial\Omega$, and ν is the outward unit normal

vector on $\partial\Omega$ and $\frac{\partial}{\partial\nu}$ means the normal derivative along ν on $\partial\Omega$. Let $t \in [0, T]$ be the time variable and $\mathbf{x} = (x, y) \in \Omega$ is the location of an individual in domain Ω . Subdividing domain Ω into smaller subregions (e.g small square cells), the within one-time step (Δt), an individual located at $(x, y) \in \Omega$ remains with probability $(1 - p)$ at this place. Assuming that no preferences of the four neighboring cells, this individual moves to one of the four neighboring cells with probability p that we call **random walk**. If number of individuals in domain Ω in time $t > 0$ notated by $N(x, t)$ then

$$\partial_t N(x, t) = D\Delta N \text{ with } D = \frac{p}{4} \frac{\Delta t}{(\Delta x)^2}.$$

The number of individuals at time t in location $\mathbf{x}$, $N(x, t)$ is divided into four compartments: susceptible $S(x, t)$, exposed $E(x, t)$, symptomatic infectious $I(x, t)$, and asymptomatic infectious $A(x, t)$. Further, assuming that the mobility of human are not influenced by individual health condition, then the mathematical modeling of for spatio-temporal SEIA model can be presented as a coupled parabolic system as follows:

$$\begin{aligned} \frac{\partial S}{\partial t} &= D_S \Delta S + \mu - \beta S(rA + qI) + \gamma I + \eta A - \mu S, \\ \frac{\partial E}{\partial t} &= D_E \Delta E + \beta S(rA + qI) - (\kappa + \mu)E, \\ \frac{\partial I}{\partial t} &= D_I \Delta I + p\kappa E - (\gamma + \mu)I, \\ \frac{\partial A}{\partial t} &= D_A \Delta A + (1 - p)\kappa E - (\eta + \mu)A, \end{aligned} \quad (10)$$

where D_S, D_E, D_I, D_A denote the corresponding diffusion rates for the susceptible, exposed, symptomatic infected, and asymptomatic infected individual, respectively and Δ is the Laplace operator. Since the mobility of individuals is not affected by infection or disease, we assume that $D_S = D_E = D_I = D_A = D$. The total population at the time t and location x is $N(x, t) = S(x, t) + E(x, t) + I(x, t) + A(x, t)$. The initial value is given by

$$S(x, 0) = S_0(x), E(x, 0) = E_0(x), I(x, 0) = I_0(x), A(x, 0) = A_0(x),$$

with non-negative function $E_0(x), I_0(x), A_0(x)$ and positive function $S_0(x)$. The homogenous Neumann condition, which can be written as

$$\frac{\partial S(x, t)}{\partial n} = \frac{\partial E(x, t)}{\partial n} = \frac{\partial I(x, t)}{\partial n} = \frac{\partial A(x, t)}{\partial n} = 0, \text{ with } x \in \partial\Omega$$

mean there is no population flux across the boundary (no migration occurs) $\partial\Omega$ and all of compartment population live in the self-contained environment (isolated habitat).

Lemma 5.1. *The total population $N(t) = \int_{\Omega} N(t, x) d\Omega$ is constant in time.*

Proof. From system (10) and $N(x, t) = S(x, t) + E(x, t) + I(x, t) + A(x, t)$ then we can get

$$\begin{aligned} \frac{\partial}{\partial t} \int_{\Omega} N(x, t) dx &= \int_{\Omega} \frac{\partial N(x, t)}{\partial t} dx = \int_{\Omega} \frac{\partial (S + E + I + A)}{\partial t} dx = \int_{\Omega} \Delta D(S + E + I + A) dx \\ &= D \int_{\Omega} \Delta N(x, t) d\Omega = D \int_{\partial\Omega} \frac{\partial N}{\partial n} = 0 \end{aligned}$$

This means that total population in domain Ω will be constant, or $\int_{\Omega} N(x, t) = N$ for $t \geq 0$. $\square$

The author in [22] was interested mainly in the equilibrium solution of (10), that is the solutions of the following semi-linear elliptic systems :

$$\begin{aligned} -D_S \Delta \tilde{S} &= \mu - \beta \tilde{S}(r\tilde{A} + q\tilde{I}) + \gamma \tilde{I} + \eta \tilde{A} - \mu \tilde{S}, \\ -D_E \Delta \tilde{E} &= \beta \tilde{S}(r\tilde{A} + q\tilde{I}) - (\kappa + \mu) \tilde{E}, \\ -D_I \Delta \tilde{I} &= p\kappa \tilde{E} - (\gamma + \mu) \tilde{I}, \\ -D_A \Delta \tilde{A} &= (1 - p)\kappa \tilde{E} - (\eta + \mu) \tilde{A}, \end{aligned} \quad (11)$$

Here, $\tilde{S}$, $\tilde{E}$, $\tilde{I}$, and $\tilde{A}$, which is called the equilibrium solution of (10), denote the density of susceptible, exposed, symptomatic infectious, and asymptomatic infectious, respectively, at $x \in \Omega$. In view of Lemma 5.1, it is reasonable to impose the additional hypothesis

$$\int_{\Omega} (\tilde{S}(x) + \tilde{E}(x) + \tilde{I}(x) + \tilde{A}(x)) dx = N$$

Throughout this paper, it is always assumed that N is a fixed positive constant.

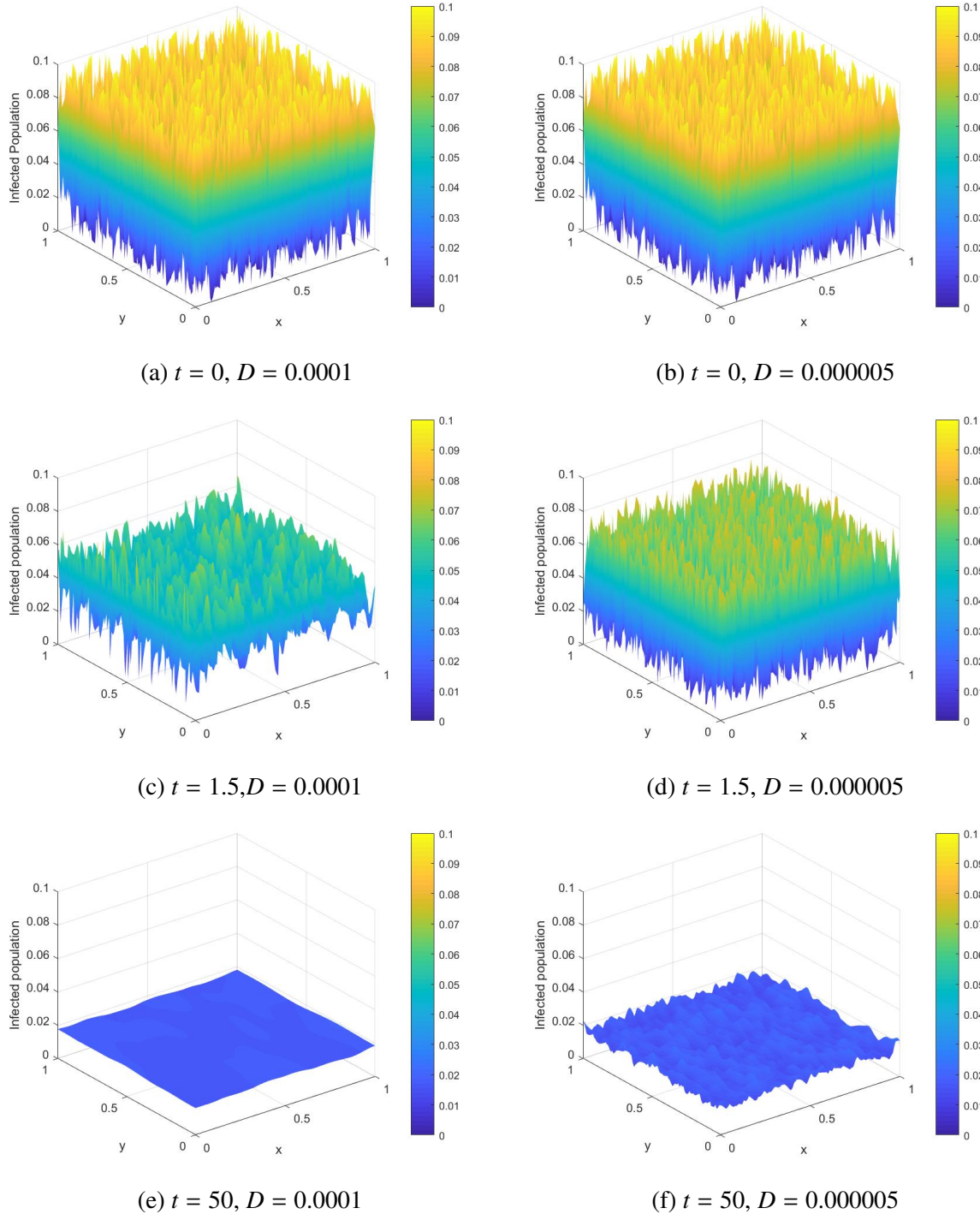


Figure 4: Solution of ppulation I in System (10) with $D = 0.0001$ and $D = 0.000005$ with parameter in Table 1 and $b = 0.4$. The color describes the proportion of I

Lemma 5.2. Define R_0 as

$$R_0 = \frac{\beta\mu}{\mu(\kappa + \mu)} \left(\frac{r\kappa(1-p)}{\eta + \mu} + \frac{\kappa pq}{\gamma + \mu} \right) \quad (12)$$

System (10) has two positive steady states, which can be formulated as

1. $DFE = \{S^0 = 1, E^0 = 0, I^0 = 0, A^0 = 0\}$
2. $END = \{S^*, E^*, I^*, A^*\}$

When $R_0 < 1$ then DFE stable, but while $R_0 > 1$ then END stable.

Using the parameters in Table 1, we simulate the System (10) with a homogeneous initial spatial condition in Figure 4. In this numerical simulation, we display and compare numerical simulations in certain infected populations with symptoms of I in System (11) with two different diffusion rates, namely $D = 0.0001$ and $D = 0.000005$ (see in Figure 5). At the beginning of time t , infected population exist in each location (x, y) randomly about 0.1. At time $t = 1.5$, the proportion of infected people starts to decrease, but the proportion of I with $D = 0.0001$ is smaller than the proportion of I with $D = 0.000005$. The results show that the diffusion rate of $D = 0.0001$ spreads faster than $D = 0.000005$ and reaches an endemic equilibrium point (with $I = 0.008416821167$) for each region at the time of $t = 200$ (see in Figure 5). From The Figure 5, it is shown that the spread of the disease is faster with a diffusion coefficient of 0.0001 compared to 0.000005.

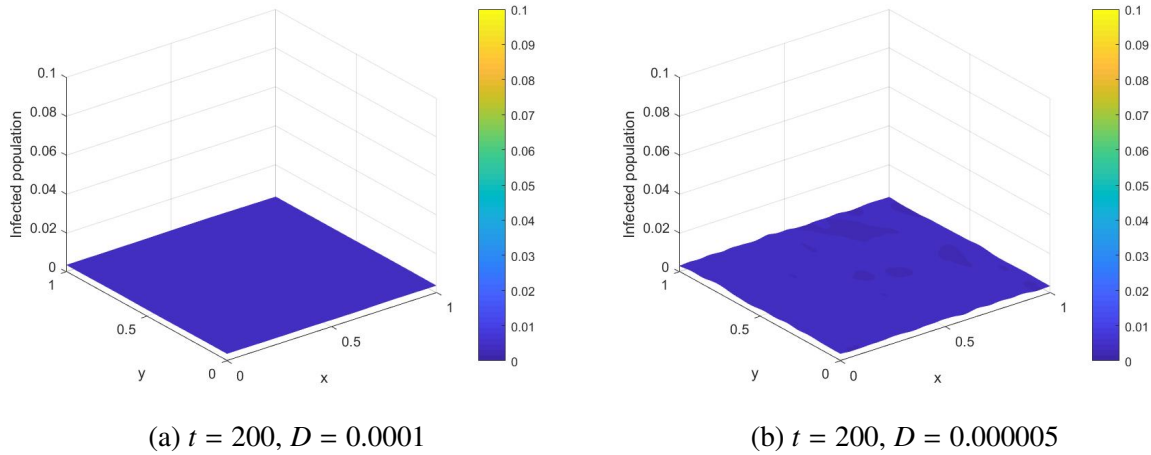


Figure 5: Solution of population I in System (10) with $D = 0.0001$ and $D = 0.000005$ and stable in endemic equilibrium

6 Conclusion

In this paper, first, we built a deterministic model of susceptible, exposed, symptomatic infectious, and asymptomatic infectious (SEIA) with Ordinary differential equations (ODE). This model has two equilibria, i.e., the disease-free equilibrium and the endemic equilibrium. The disease will become extinct when basic reproduction $R_0 < 1$ and will occur endemically at $R_0 > 1$. Sensitivity analysis shows that the transmission rate β provides a significant magnitude of the chance of all solutions for susceptible, exposed, symptomatic infectious, and asymptomatic infectious. Then, SEIA ODE is modified by adding the effect of mobility to partial differential equations with Neumann homogeneous conditions. The mobility effect is displayed at the diffusion rate. In this numerical simulation, we show the transmission of infected populations with symptoms. This numerical simulation on the Spatio-temporal SEIA model shows that the diffusion rate influences the speed of the transmission of populations infected with symptoms to other areas. The diffusion rate increases, and then the transmission to other regions of infected populations with symptoms also increases. In other words, the rate of mobility of infected people increases, so the faster the infected population spreads to other areas that were not initially infected.

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