



ANALYZING COVID-19 DYNAMICS IN TWO NON-INTERACTING REGIONS THROUGH A STRUCTURED COMPARTMENTAL MODEL

**Muhammad Afief Balya^{1,*}, Dipo Aldila², Yuni Yulida³,
Sila Rizqina⁴, Pardi Affandi⁵**

^{1,3,4,5}Departement of Mathematics, Lambung Mangkurat University, Banjarbaru

²Department of Mathematics, University of Indonesia, Depok

*email: afief.balya@ulm.ac.id

ABSTRACT

COVID-19 is an infectious disease caused by the coronavirus. Coronavirus or Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) is a virus that attacks the respiratory system. This paper will discuss the effect of the absence of human mobility on the spread of COVID-19. The proposed model consists of ten compartments, including susceptible, exposed, infected (symptomatic and asymptomatic), and recovered individuals in both regions. The model construction in this paper is quite simple, namely, it does not involve human mobility at all. This work is important because by understanding the characteristics of COVID-19 in a closed population, the spread of COVID-19 locally can be anticipated. Both analytical and numerical approaches are used. The numerical study involves elasticity analysis to identify key influential parameters and autonomous simulations to observe the long-term behavior of the system.

Keywords: COVID-19, SARS-CoV-2, Model Construction, Elasticity Analysis, Autonomous Simulation

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INTRODUCTION

Since December 2019, the global community has faced the COVID-19 pandemic. The virus is believed to have first come into contact with humans in Wuhan, China, on November 17, 2019, before spreading worldwide (Aldila et al., 2021). In Indonesia, the first confirmed case of COVID-19 was reported on March 2, 2020. The coronavirus, or Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), attacks the respiratory system. Droplets carrying the virus can settle on surrounding objects and surfaces. If someone touches these objects or surfaces and subsequently touches their eyes, nose, or mouth, they can become infected. Transmission can also occur if a person inhales droplets from the cough or breath of someone infected with COVID-19.

To gain a deeper understanding of COVID-19, integrating mathematics and public health becomes essential. (Ferguson et al., 2020) assessed the impact of non-pharmaceutical interventions, such as social distancing, on hospital burdens and mortality rates. A study by (Zlojutro et al., 2019) even designed a framework to optimize border control for mitigating global outbreaks. These studies demonstrate

how interdisciplinary approaches can provide more comprehensive solutions for addressing pandemics. For instance, mathematical modeling, such as the SIR (Susceptible-Infected-Recovered) model and its variations, has become a critical tool for studying the dynamics of infectious disease spread, including COVID-19. Research conducted by (He et al., 2020) developed the SEIR (Susceptible-Exposed-Infected-Recovered) model to study the impact of incubation periods and quarantine measures on the spread of COVID-19. Furthermore, (Yang & Wang, 2020) modified the SEIR model by incorporating non-pharmaceutical intervention factors to understand the spread patterns in Wuhan, China. (Jia et al., 2020) integrated inter-regional mobility elements to explore how population movement influences virus transmission on a global scale.

Most previous studies, such as those by (Pei & Shaman, 2020) and (Jia et al., 2020), included mobility elements in their models to describe the global or inter-regional spread of COVID-19. However, scenarios without mobility, such as during regional quarantine or total lockdown, require a different approach. In this context, models that consider only local interactions become highly relevant. By constructing a mathematical model that disregards inter-regional mobility, this study aims to analyze how factors such as transmission rates, recovery time, and local interventions affect the dynamics of COVID-19 spread. This approach is expected to illustrate transmission patterns under extreme conditions, such as total lockdown, and provide strategic recommendations for optimizing outbreak control at the local level. This paper discusses the development of a COVID-19 disease spread model considering more than one region, where the population in each region conducts activities only within its own boundaries, meaning there is no mobility between regions.

The study will derive the disease-free equilibrium point, the basic reproduction number, and the stability of the equilibrium point. The disease-free equilibrium point will be analyzed using the approach of Van den Driessche and Watmough (Driessche & Watmough, 2002). Subsequently, numerical simulations will be conducted. These simulations will include elasticity analysis of $\mathcal{R}_0$ and autonomous simulations.

METHOD

Below are the steps used in this study:

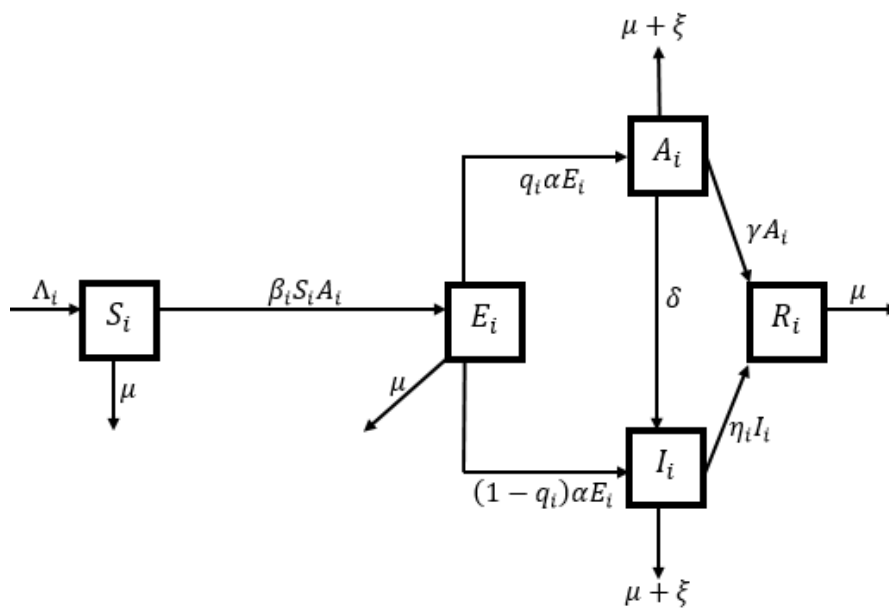
1. Model formulation. The model refers to the SIR model by Kermack and McKendrick, which consists of a system of nonlinear differential equations (Martcheva, 2015).
2. Determination of the disease-free equilibrium point. The equilibrium point is the solution of the constructed system or model
3. Derivation of the basic reproduction number. The method used is the Next Generation Matrix method (Diekmann et al., 2010), (Martcheva, 2015).

4. Stability analysis of the disease-free equilibrium point. The stability analysis is carried out using the method of Van den Driessche (Driessche & Watmough, 2002), which involves linearization and the construction of the Jacobian matrix (Boyce & DiPrima, 2009), followed by the computation of eigenvalues (Anton & Rorres, 2005)
5. Numerical simulation. The numerical simulation consists of elasticity analysis and autonomous simulation (Chitnis et al., 2008).

MODEL CONSTRUCTION

In this section, we will construct a COVID-19 disease transmission model with five compartments, consisting of: susceptible individuals in city i (S_i), exposed individuals in city i (E_i), infected individuals with symptoms in city i (A_i), infected individuals without symptoms in city i (I_i), and recovered individuals in city i (R_i). Several assumptions are necessary for constructing the model, as follows:

- 1) The human population in each city is closed and migration is ignored.
- 2) The human population in each city is homogeneous, meaning every individual has an equal chance of being infected by COVID-19.
- 3) All births are added to the susceptible population.
- 4) The infection process occurs through contact between compartment S_i and compartment A_i .
- 5) Compartment I_i is forced into quarantine, so I_i cannot infect other individuals.
- 6) Individuals in the A_i compartment represent undetected cases, meaning they are infected but remain unidentified and continue to interact freely with the susceptible group (S_i). In contrast, individuals in the I_i compartment are confirmed cases who have been detected and are therefore isolated, with no further contact with the susceptible group (S_i).
- 7) Recovery occurs due to natural recovery, and medication is ignored in this model.
- 8) Natural death occurs in every compartment.
- 9) The recovery rate, natural death rate, and COVID-19-related death rate are the same in each city.



where i denotes the city index. Since there are two cities, $i = 1, 2$.

Figure 1. Diagram of the infection process of COVID-19 disease spread in the city internally

Table 1. Description of model variables given in systems (1)

Variable	Description
S_1	The number of people in city 1 who are vulnerable to COVID-19
E_1	The number of people in city 1 who are infected with COVID-19 but have not yet transmitted the disease
A_1	The number of people in city 1 who are infected with COVID-19 but asymptomatic and undetected
I_1	The number of people in city 1 infected with COVID-19 showing symptoms and detected
R_1	The number of people in city 1 who have recovered from COVID-19
S_2	The number of people in city 2 who are vulnerable to COVID-19
E_2	The number of people in city 2 who are infected with COVID-19 but have not yet transmitted the disease
A_2	The number of people in city 2 who are infected with COVID-19 but asymptomatic and undetected
I_2	The number of people in city 2 infected with COVID-19 showing symptoms and detected
R_2	The number of people in city 2 who have recovered from COVID-19

Based on the diagram of infection process in figure 1 and the above assumptions, the COVID-19 model in isolated region can be expressed in the nonlinear system of differential equations stated below

$$\begin{aligned}
 \frac{dS_1}{dt} &= \Lambda_1 - \beta_1 S_1 A_1 - \mu S_1, \\
 \frac{dE_1}{dt} &= \beta_1 S_1 A_1 - q_1 \alpha E_1 - (1 - q_1) \alpha E_1 - \mu E_1, \\
 \frac{dA_1}{dt} &= q_1 \alpha E_1 - \delta A_1 - \gamma A_1 - \mu A_1 - \xi A_1, \\
 \frac{dI_1}{dt} &= (1 - q_1) \alpha E_1 + \delta A_1 - \eta_1 I_1 - \mu I_1 - \xi I_1, \\
 \frac{dR_1}{dt} &= \gamma A_1 + \eta_1 I_1 - \mu R_1,
 \end{aligned}$$

for city 1 and

(1)

$$\begin{aligned}
 \frac{dS_2}{dt} &= \Lambda_2 - \beta_2 S_2 A_2 - \mu S_2, \\
 \frac{dE_2}{dt} &= \beta_2 S_2 A_2 - q_2 \alpha E_2 - (1 - q_2) \alpha E_2 - \mu E_2, \\
 \frac{dA_2}{dt} &= q_2 \alpha E_2 - \delta A_2 - \gamma A_2 - \mu A_2 - \xi A_2, \\
 \frac{dI_2}{dt} &= (1 - q_2) \alpha E_2 + \delta A_2 - \eta_2 I_2 - \mu I_2 - \xi I_2, \\
 \frac{dR_2}{dt} &= \gamma A_2 + \eta_2 I_2 - \mu R_2,
 \end{aligned}$$

for city 2.

Equation system (1) is a five-dimensional nonlinear system of differential equations with initial conditions satisfying:

$$S_i(t) + E_i(t) + A_i(t) + I_i(t) + R_i(t) = N_i(t)$$

where i denotes the city index. Since there are two cities, $i = 1, 2$.

The table below presents the parameters in the model along with their descriptions and estimated values.

Table 2. Model parameters with their descriptions and estimated values

Variable	Description	Values	Source
Λ_i	Human recruitment rate in city 1 and 2	$10^7/(70 \times 365)$	(Aldila et al., 2020)
β_1	Rate of transmission of susceptible individuals in city 1	1.85×10^{-7}	fitted
β_2	Rate of transmission of susceptible individuals in city 2	8.3×10^{-8}	fitted
μ	Natural death rate	$1/(70 \times 365)$	(Feng & Velasco-Hernández, 1997)
q_i	The proportion of people in city i who are exposed to COVID-19 and then move to A_i	[0,1]	estimated
$1 - q_i$	The proportion of people in city i who are exposed to COVID-19 and then move to I_i	[0,1]	estimated
α	Incubation rate	1/5.1	(Li et al., 2020)
δ	The rate of human movement from A_i to I_i	$(0, \infty)$	fitted
ξ	Death rate due to COVID-19	0.05	(Aldila et al., 2021)
η_i	The Recovery rate of the I_i subpopulation in city i	$(0, \infty)$	fitted
γ	The Recovery rate of the A_i subpopulation	0.1	fitted

MODEL ANALYSIS

In this section, the analysis of the previously constructed mathematical model for the spread of COVID-19 will be continued. The model analysis begins with the determination of the disease-free equilibrium point, $\mathcal{R}_0$, and the stability of that point. The disease-free equilibrium point is analyzed using the approach of Van den Driessche and Watmough.

Disease Free-Equilibrium (DFE) and Basic Reproduction Number

The disease-free equilibrium (DFE) is a state where a disease no longer exists within a human population or will disappear from the population. Therefore, the disease-free equilibrium for this model is obtained as follows:

$$DFE = \left(\frac{\Lambda_1}{\mu}, 0, 0, 0, 0, \frac{\Lambda_2}{\mu}, 0, 0, 0, 0 \right). \quad (2)$$

In various mathematical models, the local stability of the DFE typically depends on the condition that the basic reproduction number ($\mathcal{R}_0$) is less than one (Aldila et al., 2021), (Balya et al., 2021). The value of $\mathcal{R}_0$ can be calculated using the Next Generation Matrix (NGM) approach. The first step in constructing the NGM is to linearize all subpopulations exposed to and infected by the disease (E_i, A_i, I_i) from the system of equations (1), resulting in the Jacobian matrix as shown in equation (3) below.

$$\mathcal{J} = \begin{bmatrix} j_{11} & \beta_1 S_1 & 0 & 0 & 0 & 0 \\ q_1 \alpha & j_{22} & 0 & 0 & 0 & 0 \\ (1-q_1)\alpha & \delta & -\eta_1 - \mu - \xi & 0 & 0 & 0 \\ 0 & 0 & 0 & j_{44} & \beta_2 S_2 & 0 \\ 0 & 0 & 0 & q_2 \alpha & j_{55} & 0 \\ 0 & 0 & 0 & (1-q_2)\alpha & \delta & -\eta_2 - \mu - \xi \end{bmatrix} \quad (3)$$

where

$$j_{11} = -q_1 \alpha - (1-q_1)\alpha - \mu,$$

$$j_{22} = j_{55} = -\delta - \gamma - \mu - \xi,$$

$$j_{44} = -q_2 \alpha - (1-q_2)\alpha - \mu.$$

Next, the decomposition of the Jacobian matrix in equation (3) is expressed as $\mathcal{J} = \mathcal{T} + \mathcal{\Sigma}$. Here, $\mathcal{T}$ represents the transmission matrix associated with the process of new infections, while $\mathcal{\Sigma}$ represents the transition matrix related to the movement of individuals between exposed and infected compartments. Then, by substituting the DFE into the transmission matrix ($\mathcal{T}$) and the transition matrix ($\mathcal{\Sigma}$), we obtain:

$$\mathcal{T}_{|DFE} = \begin{bmatrix} 0 & \frac{\Lambda_1 \beta_1}{\mu} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\Lambda_2 \beta_2}{\mu} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

and

$$\mathcal{\Sigma}_{|DFE} = \begin{bmatrix} j_{11} & 0 & 0 & 0 & 0 & 0 \\ q_1 \alpha & j_{22} & 0 & 0 & 0 & 0 \\ (1-q_1)\alpha & \delta & -\eta_1 - \mu - \xi & 0 & 0 & 0 \\ 0 & 0 & 0 & j_{44} & 0 & 0 \\ 0 & 0 & 0 & q_2 \alpha & j_{55} & 0 \\ 0 & 0 & 0 & (1-q_2)\alpha & \delta & -\eta_2 - \mu - \xi \end{bmatrix},$$

where

$$j_{11} = -q_1 \alpha - (1-q_1)\alpha - \mu,$$

$$j_{22} = j_{55} = -\delta - \gamma - \mu - \xi,$$

$$j_{44} = -q_2\alpha - (1 - q_2)\alpha - \mu .$$

Next, calculate the NGM matrix, denoted by $\mathcal{K}$, using the following calculation:

$$\mathcal{K} = -\mathbf{E}^T \cdot \mathcal{F}_{|DFE} \cdot \Sigma_{|DFE}^{-1} \cdot \mathbf{E} ,$$

with

$$\mathbf{E} = \begin{bmatrix} 1 & 0 \\ 0 & 0 \\ 0 & 0 \\ 0 & 1 \\ 0 & 0 \\ 0 & 0 \end{bmatrix} \text{ and } \mathbf{E}^T = \begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \end{bmatrix} .$$

Then, from the matrix $\mathcal{K}$ above, the characteristic polynomial equation is obtained as follows:

$$\lambda^2 - \frac{(\Lambda_1\beta_1q_1 + \Lambda_2\beta_2q_2)\alpha}{\mu(\delta + \gamma + \mu + \xi)(\alpha + \mu)} \lambda + \frac{\Lambda_1\beta_1q_1\Lambda_2\beta_2q_2\alpha^2}{\mu^2(\delta + \gamma + \mu + \xi)^2(\alpha + \mu)^2}$$

Thus, $\mathcal{R}_0$ is obtained as the spectral radius of $\mathcal{K}$, which is the largest eigenvalue of the matrix $\mathcal{K}$, as follows:

$$\mathcal{R}_0^{no \ mobility} = \frac{\Lambda_i\beta_iq_i\alpha}{\mu(\delta + \gamma + \mu + \xi)(\alpha + \mu)} \quad (4)$$

where $i = 1, 2$.

Theorem 1. The mathematical model in the system of equations (1) has a basic reproduction number ($\mathcal{R}_0$) given in equation (4).

Van den Driessche and Watmough Approach

Theorem 2. The disease-free equilibrium of the COVID-19 transmission model in the system of equations (1) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

Proof. The first step is to rewrite the system of equations (1) by ordering the first m compartments, which represent the infected compartments, followed by the uninfected compartments. Thus, the system can be rewritten as equation (5) as shown below.

$$\begin{aligned}
 f_1 &= \frac{dE_1}{dt} = \beta_1 S_1 A_1 - q_1 \alpha E_1 - (1 - q_1) \alpha E_1 - \mu E_1, \\
 f_2 &= \frac{dA_1}{dt} = q_1 \alpha E_1 - \delta A_1 - \gamma A_1 - \mu A_1 - \xi A_1, \\
 f_3 &= \frac{dI_1}{dt} = (1 - q_1) \alpha E_1 + \delta A_1 - \eta_1 I_1 - \mu I_1 - \xi I_1, \\
 f_4 &= \frac{dE_2}{dt} = \beta_2 S_2 A_2 - q_2 \alpha E_2 - (1 - q_2) \alpha E_2 - \mu E_2, \\
 f_5 &= \frac{dA_2}{dt} = q_2 \alpha E_2 - \delta A_2 - \gamma A_2 - \mu A_2 - \xi A_2, \\
 f_6 &= \frac{dI_2}{dt} = (1 - q_2) \alpha E_2 + \delta A_2 - \eta_2 I_2 - \mu I_2 - \xi I_2, \\
 f_7 &= \frac{dR_1}{dt} = \gamma A_1 + \eta_1 I_1 - \mu R_1, \\
 f_8 &= \frac{dR_2}{dt} = \gamma A_2 + \eta_2 I_2 - \mu R_2, \\
 f_9 &= \frac{dS_1}{dt} = \Lambda_1 - \beta_1 S_1 A_1 - \mu S_1, \\
 f_{10} &= \frac{dS_2}{dt} = \Lambda_2 - \beta_2 S_2 A_2 - \mu S_2.
 \end{aligned} \tag{5}$$

It is then defined that $\mathbf{X}_s$ is the set of all populations in the disease-free state, which can be written as follows:

$$\mathbf{X}_s = \left\{ x_1 = E_1 = 0, x_2 = A_1 = 0, x_3 = I_1 = 0, x_4 = E_2 = 0, x_5 = A_2 = 0, x_6 = I_2 = 0, x_7 = R_1 = 0, x_8 = R_2 = 0, x_9 = S_1 = \frac{\Lambda_1}{\mu}, x_{10} = S_2 = \frac{\Lambda_2}{\mu} \right\}.$$

In the disease transmission model, it must be ensured that the initial condition of each population (compartment) is non-negative, following the system of equations below, like in equation (11).

$$\dot{x}_k = f_i(x) = \mathcal{F}_k(x) - \mathcal{V}_k(x), k = 1, 2, \dots, n,$$

with n be the number of compartments in the model, and $\mathcal{V}_k = \mathcal{V}_k^- - \mathcal{V}_k^+$.

$\mathcal{F}_k$ represents the rate group that produces new infections in each k -th compartment, $\mathcal{V}_k^-$ represents the rate group of individuals leaving the k -th compartment, and $\mathcal{V}_k^+$ represents the rate group of individuals entering the k -th compartment. Therefore, $\mathcal{F}_k(x)$, $\mathcal{V}_k^-(x)$, and $\mathcal{V}_k^+(x)$ can be expressed as follows:

$$\mathcal{F}_k(x) = \begin{bmatrix} \beta_1 S_1 A_1 \\ 0 \\ 0 \\ \beta_2 S_2 A_2 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad (6)$$

$$\mathcal{V}_k^- = \begin{bmatrix} q_1 \alpha E_1 + (1 - q_1) \alpha E_1 + \mu E_1 \\ \delta A_1 + \gamma A_1 + \mu A_1 + \xi A_1 \\ \eta_1 I_1 + \mu I_1 + \xi I_1 \\ q_2 \alpha E_2 + (1 - q_2) \alpha E_2 + \mu E_2 \\ \delta A_2 + \gamma A_2 + \mu A_2 + \xi A_2 \\ \eta_2 I_2 + \mu I_2 + \xi I_2 \\ \mu R_1 \\ \mu R_2 \\ \beta_1 S_1 A_1 + \mu S_1 \\ \beta_2 S_2 A_2 + \mu S_2 \end{bmatrix}, \quad (7)$$

and

$$\mathcal{V}_k^+ = \begin{bmatrix} 0 \\ q_1 \alpha E_1 \\ (1 - q_1) \alpha E_1 + \delta A_1 \\ 0 \\ q_2 \alpha E_2 \\ (1 - q_2) \alpha E_2 + \delta A_2 \\ \gamma A_1 + \eta_1 I_1 \\ \gamma A_2 + \eta_2 I_2 \\ \Lambda_1 \\ \Lambda_2 \end{bmatrix}. \quad (8)$$

Then, the stability analysis of the Disease-Free Equilibrium (DFE) can be carried out by satisfying conditions (A1)–(A5) as stated below.

(A1): If $x \geq 0$, then $\mathcal{F}_k, \mathcal{V}_k^-, \mathcal{V}_k^+ \geq 0$ for $k = 1, 2, \dots, n$.

Based on equations (6), (7), and (8), we have $\mathcal{F}_k, \mathcal{V}_k^-, \mathcal{V}_k^+ \geq 0$ for $k = 1, 2, \dots, n$ because all parameters and variables are non-negative. Consequently, condition (A1) is satisfied.

Next, if a sub-population is empty, no individuals will move out of it due to death, infection, or any other reason. Therefore,

(A2): If $x_k = 0$, then $\mathcal{V}_k^- = 0$. In particular, if $\in \mathbf{X}_s$, we have $\mathcal{V}_k^- = 0$ for $k = 1, 2, \dots, m$, where m is the number of infected compartments. Substituting $x_k = 0$ into $\mathcal{V}_k^-(x)$ in equation (7) yields equation (9) as follows:

$$\mathcal{V}_k^- = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}. \quad (9)$$

Based on the result in equation (9) above, we see that every entry of $\mathcal{V}_k^-$ is zero for $k = 1, 2, 3, 4, 5, 6$ (considering only the infected compartments). Hence, condition (A2) is satisfied.

Next, condition (A3) is established on the simple fact that the incidence of infection in a disease-free sub-population is zero. Therefore,

(A3): $\mathcal{F}_k = 0$, when $k > m$.

To ensure that a disease-free sub-population remains free of infection, we assume the sub-population is closed—immigration is ignored when counting individuals. Hence, from equation (6) for $k = 7, 8, 9, 10$ (considering only the uninfected compartments), we obtain $\mathcal{F}_7(x) = 0$, $\mathcal{F}_8(x) = 0$, $\mathcal{F}_9(x) = 0$, $\mathcal{F}_{10}(x) = 0$. Therefore, condition (A3) is satisfied.

Next, condition (A4) is stated as follows:

(A4): If $x \in \mathbf{X}_s$, then $\mathcal{F}_k(x) = 0$ and $\mathcal{V}_k^+(x) = 0$ for $k = 1, 2, \dots, m$, where m is the number of infected compartments.

Substituting $x_k = 0$ into $\mathcal{F}_k(x)$ in equation (6) and into $\mathcal{V}_k^+(x)$ in equation (8) yields equation (10) as follows:

$$\mathcal{F}_k = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \text{ dan } \mathcal{V}_k^+ = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad (10)$$

Based on equation (10) above, every entry of $\mathcal{F}_k$ and $\mathcal{V}_k^+$ is zero for $k = 1, 2, 3, 4, 5, 6$ (considering only the infected compartments). Consequently, condition (A4) is satisfied.

In this approach, it is not necessary to assume that the model possesses a unique DFE. However, Van den Driessche and Watmough restrict their analysis to systems in which the DFE remains stable in the absence of new infections. Accordingly, condition (A5) is stated as follows:

(A5): If $\mathcal{F}(x) = 0$ is imposed, then every eigenvalue of $Df(x_0)$ has a negative real part, where x_0 is the disease-free equilibrium point. Setting $\mathcal{F}(x) = 0$ yields a new system of equations with no new infections, expressed as follows:

$$f_1 = \frac{dE_1}{dt} = -q_1 \alpha E_1 - (1 - q_1) \alpha E_1 - \mu E_1,$$

$$\begin{aligned}
 f_2 &= \frac{dA_1}{dt} = q_1\alpha E_1 - \delta A_1 - \gamma A_1 - \mu A_1 - \xi A_1, \\
 f_3 &= \frac{dI_1}{dt} = (1 - q_1)\alpha E_1 + \delta A_1 - \eta_1 I_1 - \mu I_1 - \xi I_1, \\
 f_4 &= \frac{dE_2}{dt} = -q_2\alpha E_2 - (1 - q_2)\alpha E_2 - \mu E_2, \\
 f_5 &= \frac{dA_2}{dt} = q_2\alpha E_2 - \delta A_2 - \gamma A_2 - \mu A_2 - \xi A_2, \\
 f_6 &= \frac{dI_2}{dt} = (1 - q_2)\alpha E_2 + \delta A_2 - \eta_2 I_2 - \mu I_2 - \xi I_2, \\
 f_7 &= \frac{dR_1}{dt} = \gamma A_1 + \eta_1 I_1 - \mu R_1, \\
 f_8 &= \frac{dR_2}{dt} = \gamma A_2 + \eta_2 I_2 - \mu R_2, \\
 f_9 &= \frac{dS_1}{dt} = \Lambda_1 - \beta_1 S_1 A_1 - \mu S_1, \\
 f_{10} &= \frac{dS_2}{dt} = \Lambda_2 - \beta_2 S_2 A_2 - \mu S_2.
 \end{aligned} \tag{11}$$

Next, linearize the system to obtain its Jacobian matrix

$$\begin{bmatrix}
 -\alpha - \mu & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 q_1\alpha & -\delta - \gamma - \mu - \xi & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 (1 - q_1)\alpha & \delta & -\eta_1 - \mu - \xi & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & -\alpha - \mu & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & q_2\alpha & -\delta - \gamma - \mu - \xi & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & (1 - q_2)\alpha & \delta & -\eta_2 - \mu - \xi & 0 & 0 & 0 & 0 \\
 0 & \gamma & \eta_1 & 0 & 0 & 0 & -\mu & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & \gamma & \eta_2 & 0 & -\mu & 0 & 0 \\
 0 & -\beta_1 \frac{\Lambda_1}{\mu} & 0 & 0 & 0 & 0 & 0 & 0 & -\mu & 0 \\
 0 & 0 & 0 & 0 & -\beta_2 \frac{\Lambda_2}{\mu} & 0 & 0 & 0 & 0 & -\mu
 \end{bmatrix}$$

After that, the characteristic polynomial equation is obtained, namely:

$$\begin{aligned}
 (\lambda + \mu)^4 (\lambda + \alpha + \mu)^2 (\lambda + \delta + \gamma + \mu + \xi)^2 (\lambda + \eta_1 + \mu + \xi) \\
 (\lambda + \eta_2 + \mu + \xi) = 0.
 \end{aligned} \tag{12}$$

From equation (12) above, 10 eigenvalues are obtained as follows:

$$\begin{aligned}
 \lambda_1 = \lambda_2 = \lambda_3 = \lambda_4 = -\mu, \lambda_5 = \lambda_6 = -\alpha - \mu, \\
 \lambda_7 = \lambda_8 = -\delta - \gamma - \mu - \xi, \lambda_9 = -\eta_1 - \mu - \xi, \\
 \lambda_{10} = -\eta_2 - \mu - \xi.
 \end{aligned} \tag{13}$$

Fom equation (13) above, it can be concluded that condition (A5) is satisfied. All conditions (A1)–(A5) have been satisfied. Thus, Theorem 2 is proven. ■

NUMERIC SIMULATION

In this section, we will discuss the numerical simulation of $\mathcal{R}_0$ for the model without mobility. The numerical simulation of $\mathcal{R}_0$ in the case of the model without

mobility consists of the elasticity analysis of $\mathcal{R}_0$ and the autonomous simulation of $\mathcal{R}_0$ for the model without mobility.

The Elasticity Analysis of $\mathcal{R}_0$ for the Model Without Mobility

$\mathcal{R}_0^{no\ mobility}$ has been obtained, as shown in Equation (4). Subsequently, the elasticity expressions of each parameter with respect to $\mathcal{R}_0^{no\ mobility}$ are analytically derived. After that, the parameter values are substituted into the elasticity equations to yield conditions where $\mathcal{R}_0^{no\ mobility} < 1$ and $\mathcal{R}_0^{no\ mobility} > 1$. The elasticity analysis with respect to the percentage change of all parameter values p is formulated as follows:

$$\varepsilon_{\partial \mathcal{R}_0}^p = \frac{\partial \mathcal{R}_0}{\partial p} \times \frac{p}{\mathcal{R}_0} \approx \frac{\% \Delta \mathcal{R}_0}{\% \Delta p} \quad (14)$$

where p is represents the parameters involved in $\mathcal{R}_0$. The resulting elasticity coefficients are presented in Table 3 below.

Table 3. Elasticity Table of $\mathcal{R}_0^{no\ mobility(i)}$

Parameter	$\mathcal{R}_0^{no\ mobility(i)} < 1$	$\mathcal{R}_0^{no\ mobility(i)} > 1$
Λ_i	1	1
β_1	1	1
q_i	1	1
μ	-1.000452373	-1.000452374
α	0.0001995688225	0.0001995688225
δ	-0.06459149732	-0.06459149732
ξ	-0.09688724598	-0.09688724598
γ	-0.8382684522	-0.8382684522

where i denotes the city index. Since there are two cities, $= 1, 2$.

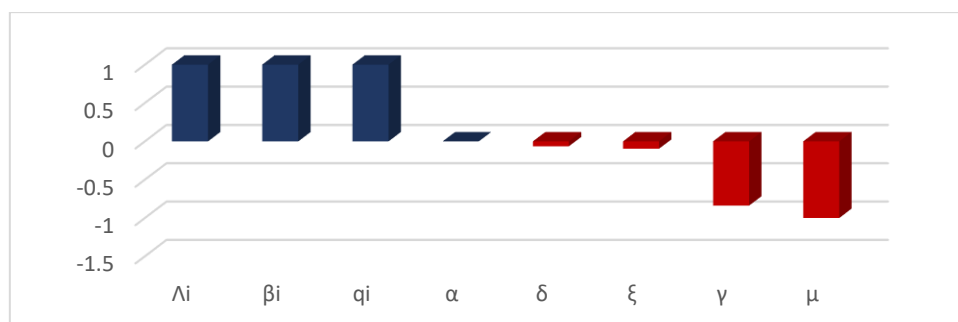


Figure 2. Elasticity Values of the Parameters with Respect to $\mathcal{R}_0^{no\ mobility}$

Based on Table 3 and Figure 2, the parameters Λ_i, β_i, q_i , and α (shown in blue) have a positive influence on $\mathcal{R}_0$, while the parameters δ, γ, ξ , and μ (shown in red) have a negative influence on $\mathcal{R}_0$. A parameter that has a positive influence on $\mathcal{R}_0$ means that increasing the value of this parameter will increase the value of $\mathcal{R}_0$.

Conversely, if a parameter has a negative influence on $\mathcal{R}_0$, increasing its value will decrease the value of $\mathcal{R}_0$. As an example, the elasticity value $\mathcal{E}_{\mathcal{R}_0}^\gamma = -0.06459149732$ means that a 1% increase in the value of γ will result in a decrease of approximately 0.06459149732% in $\mathcal{R}_0$. The same interpretation applies to the other parameters.

The Autonomous Simulation of $\mathcal{R}_0$ for the Model Without Mobility

In this section, a simulation is conducted to examine the relationship between δ and the infected population in each city ($E_1, E_2, A_1, A_2, I_1, I_2$). The parameter δ is chosen because, in this model, there is no mobility between cities. δ represents the rate at which individuals move from the asymptomatic infected population to the symptomatic infected population. This transition is typically triggered by rapid tests or swab tests, or by the worsening condition of asymptomatic individuals. The results of the autonomous simulation for this model are presented below.

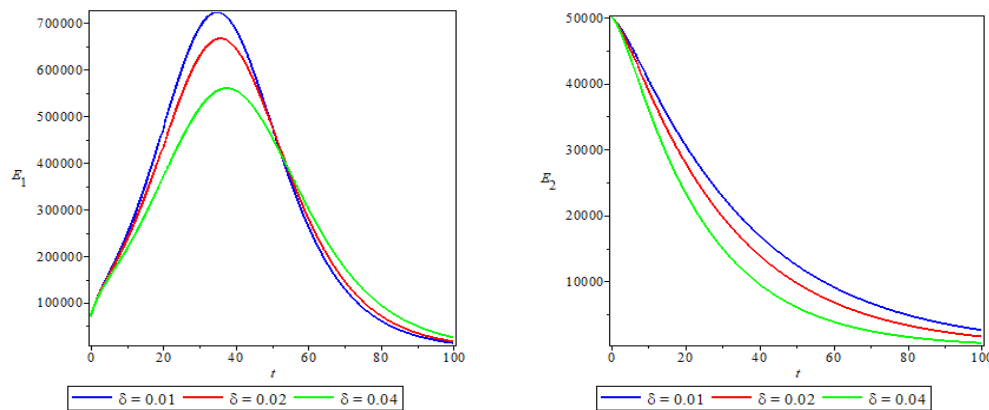


Figure 3. Autonomous Simulation of the Relationship Between the Latent Infected Population in Each City and Changes in the Value of δ in the Model Without Mobility

Based on Figure 3, the left graph shows that increasing the value of δ lowers and delays the peak of the latent infected population in City 1. The right graph indicates that in City 2, a higher δ accelerates the decline of the latent infected population toward zero. Overall, the greater the value of δ , the faster and lower the latent population decreases in both cities.

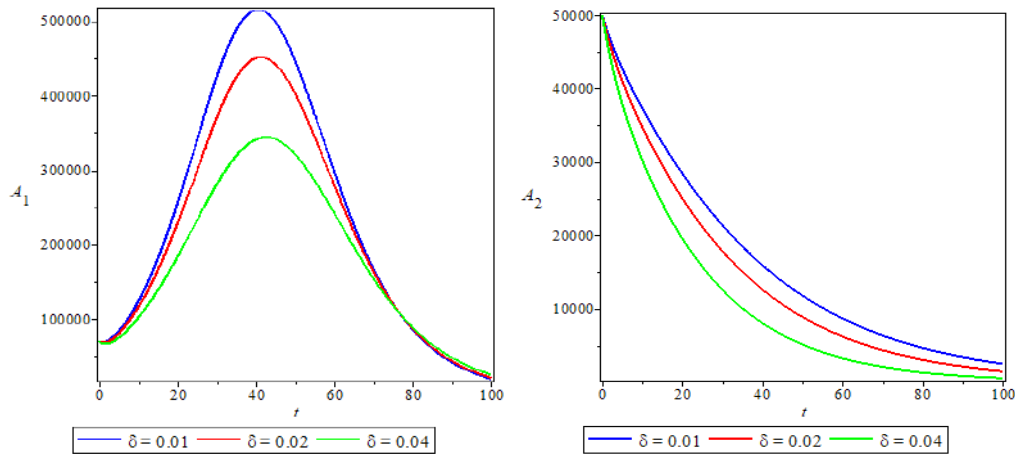


Figure 4. Autonomous Simulation of the Relationship Between the Asymptomatic Infected Population in Each City and Changes in the Value of δ in the Model Without Mobility

Figure 4 shows that in City 1 (left graph), increasing the value of δ reduces the peak height of the asymptomatic infected population, although the time to reach the peak remains relatively the same. In City 2 (right graph), a higher δ leads to a faster and steeper decline of the asymptomatic population toward zero. Overall, increasing δ accelerates the reduction of the asymptomatic infected population in both cities.

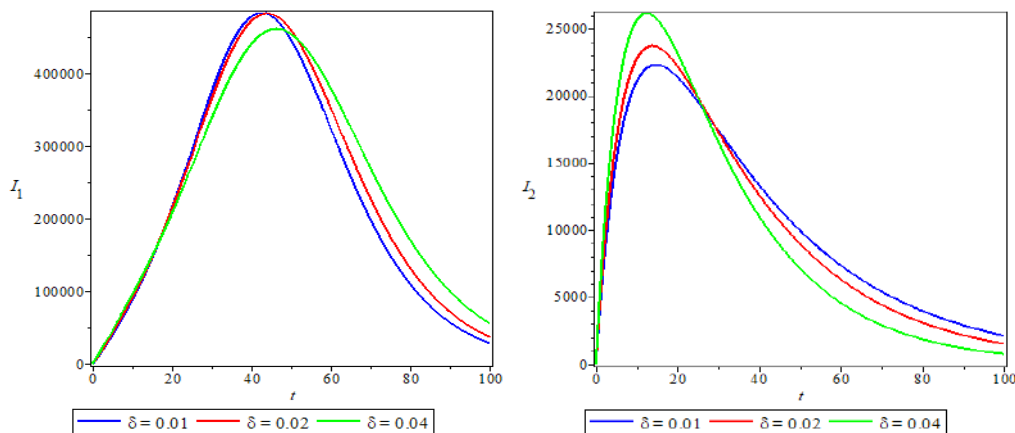


Figure 5. Autonomous Simulation of the Relationship Between the Symptomatic Infected Population in Each City and Changes in the Value of δ in the Model Without Mobility

Figure 5 shows that in City 1 (left graph), increasing the value of δ reduces the peak of the symptomatic infected population and slightly delays the time it is reached. A similar pattern is observed in City 2 (right graph), where a higher δ lowers the peak population and extends the time to reach it. In general, the larger the value of δ , the slower and lower the symptomatic infected population becomes in both cities.

The policy and practical implications of the parameter δ in controlling the spread of COVID-19.

The results indicate that the parameter δ , which governs the transition from asymptomatic to symptomatic infections, plays a significant role in the progression of the epidemic. A higher value of δ implies that more asymptomatic individuals are quickly identified as symptomatic, potentially due to increased testing capacity or earlier detection through surveillance. From a policy perspective, enhancing the value of δ through mass testing, active case finding, and prompt isolation of emerging symptomatic cases can significantly reduce the hidden spread of the virus. Therefore, improving early detection strategies not only shortens the duration of undetected transmission but also enables quicker interventions, thereby contributing to more effective control of COVID-19 outbreaks.

CONCLUSION

This paper presents both analytical and numerical studies of the developed model. The analytical study includes the determination of equilibrium points, the calculation of $\mathcal{R}_0$, and the stability analysis of the equilibrium points. The basic reproduction number $\mathcal{R}_0$ is calculated using the Next Generation Matrix (NGM) method. Furthermore, the stability of the disease-free equilibrium point is analyzed based on the value of $\mathcal{R}_0$ using the Van den Driessche method. This method shows that the disease-free equilibrium point is always stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$. This model, however, has certain limitations. It does not account for long-term immunity, vaccination effects, or social heterogeneity, which may influence the transmission dynamics in real-world scenarios. Future work will address these limitations by extending the model to incorporate one-way and two-way human mobility between regions, aiming to capture more realistic transmission patterns and improve the model's applicability to public health policy planning.

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