Open Access Library Journal
2024, Volume 11, e11900
ISSN Online: 2333-9721
ISSN Print: 2333-9705

Simultaneous Analysis of the Impact of Vaccination and Compliance with Prevention Measures on the Transmission Dynamics of COVID-19

Abraham Sassou[1], Ezekiel Dangbé[2], Richard Guiem[3], Séraphin Djaoué[1], Irépran Damakoa[4], Antoine Perasso[5]

[1]Faculty of Sciences, University of Maroua, Maroua, Cameroon
[2]Institute of Technology, University of Ngaoundere, Ngaoundere, Cameroon
[3]National Advanced School of Engineering, University of Maroua, Maroua, Cameroon
[4]Faculty of Sciences, University of Ngaoundere, Ngaoundere, Cameroon
[5]UMR6249 Chrono-Environment Laboratory, University of Franche-Comté (France), Besancon, France
Email: abrahamsassou@gmail.com, dangbeezechiel@yahoo.fr, guiemrichard@yahoo.fr, sdjaoue@gmail.com, damakoa@yahoo.fr, antoine.perasso@univ-fcomte.fr

How to cite this paper: Sassou, A., Dangbé, E., Guiem, R., Djaoué, S., Damakoa, I. and Perasso, A. (2024) Simultaneous Analysis of the Impact of Vaccination and Compliance with Prevention Measures on the Transmission Dynamics of COVID-19. *Open Access Library Journal*, **11**: e11900.
https://doi.org/10.4236/oalib.1111900

Received: July 3, 2024
Accepted: August 23, 2024
Published: August 26, 2024

Copyright © 2024 by author(s) and Open Access Library Inc.
This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).
http://creativecommons.org/licenses/by/4.0/
Open Access

Abstract

The transmission dynamics of COVID-19 worldwide have not been controlled very effectively, and still represent a major threat to public health and even to sustainable global socio-economic development. A number of vaccines have been discovered and approved, and vaccination programs have been implemented in many countries in Africa, Asia, Europe and America. Some countries have even imposed a health pass to enter their country of residence. Other countries have opted to comply with prevention measures. Our study demonstrates and underlines the essential role of vaccination and respect for prevention measures during the spreading of this infection. Using a mathematical model with seven (07) compartments, which takes into account factors such as the prevention measures decreed by the World Health Organization and the vaccination coverage of the population studied, we have demonstrated the need for a given population to be vaccinated while scrupulously observing the so-called prevention measures. This model integrates the virus concentration in the environment studied and a compartment of vaccinated individuals. It highlights the effect of vaccination and prevention measures in controlling the coronavirus epidemic. These parameters enabled us to assess the impact on disease transmission. Indeed, after studying the biological feasibility of the model, we calculated the basic reproductive number R_0 and showed that the disease disappears at $\mathcal{R}_0 < 1$; is endemic at $\mathcal{R}_0 > 1$.

We also calculated other important parameters. We have also analyzed the asymptotic behaviour of the solutions, by establishing equilibrium conditions and the existence of endemic equilibrium points, and we performed numerical simulations to highlight the asymptotic behaviour of the solutions thanks to data collected by the European Centre for Disease Prevention and Control in 2019 as well as those used by Yang and Wang in 2020. In future studies, we will highlight the impact of vaccination, temperature and prevention measurements on a fractional model.

Subject Areas

Applied Mathematics

Keywords

COVID-19, Vaccination, Prevention Measures, Basic Reproductive Number, Simulations

1. Introduction

COVID-19 is an infectious disease caused by a virus, the coronavirus. Symptoms include a dry cough, headaches, aches and pains, fever, sudden loss of smell and taste, diarrhea and fatigue. In more severe forms, respiratory difficulties can lead to hospitalization in intensive care or even death. According to a review by the Faculty of Medicine and Dentistry at the Catholic University of Leuven, other signs of the disease may also appear, such as skin manifestations. Discovered in the 1960s, coronavirus is transmitted from a contaminated patient to a healthy person via salivary droplets released when talking, coughing, or sneezing. SARS-CoV-2 can also be transmitted through contact with an infected animal or an object soiled with the virus [1]-[3]. According to a study published in May 2020 in the American scientific journal PNAS (Proceedings of the National Academy of Sciences), speech and breathing, which emit microdroplets, could also be vectors for the transmission of COVID-19. They would be just as dangerous as coughing and sneezing. Contamination could also be airborne. Indeed, according to a study conducted by Pierre Bienvault in March 2020, the coronavirus could remain airborne for up to three hours; but specialists believe that this work does not allow us to conclude that COVID-19 is transmitted via the air. SARS-CoV-2 was identified in December 2019 at the Huanan seafood wholesale market in Wuhan, China, which sells live animals. While the specific animal source of the virus has not yet been determined, it is believed to be of animal origin. The pangolin, an endangered species, and the bat have been singled out by some scientists for their potential as animal reservoirs and intermediary hosts respectively. The epidemic has since spread to every continent around the globe. From the identified case in China until November 15, 2020, the World Health Organization (WHO) reported over 53.7 million cases of COVID-19 worldwide,

classifying the disease as a pandemic. The disease spread to other countries via travelers. After Asia, the disease spread to Europe and America, before reaching Africa in early March 2020. To limit the spread of this pandemic, most countries have taken measures such as border closures and containment [1]-[3]. To date, to protect themselves and control the spread of the pandemic, according to WHO recommendations, the Cameroon government recommended taking vaccines as well as respecting some prevention measures such as wearing masks in public places, regular hand-washing, and respecting physical distancing, etc.

Managing this pandemic is a public health emergency for the international community. The situation is most pressing in countries with limited resources, particularly in sub-Saharan Africa. Given the precarious conditions in which the populations of sub-Saharan African countries live, and the fragility of these countries' economies, it is difficult to envisage the measures initiated in the developing countries of sub-Saharan Africa to combat this pandemic [4]-[9].

To prevent these countries with limited resources from being devastated by this pandemic, on the one hand, and on the other, from becoming potential hotbeds for the emergence of the COVID-19 epidemic, the international community and developing countries will be obliged to support these countries. So it's vital to determine the main factors likely to impact on the spread of the virus, and to assess impact of measures to be proposed. Mathematical modeling and numerical simulations are ideally suited to these tasks.

Numerous ways of evaluating the spread of some transmissible diseases have been set out. For the particular case of COVID-19, each of the proposed mathematical models is analyzed according to the different hypotheses set and objectives to be reached. Some of the work carried out on mathematical modelling needs to be presented. We begin by looking at the work of Shigui Ruan of the University of Miami *et al.*, who in January 2006, in a paper entitled "The effect of global travel on the spread of SARS" a multiregional compartmental model using the theory of medical geography (central place theory) and considering each epidemic zone (such as Hong Kong SAR, Singapore, Toronto and Beijing) as a single region. To understand the global spread of SARS, they proposed a multiregional model for the effect of international travel on the geographic transmission of the disease. They first calculated the basic reproduction number ($\mathcal{R}_0$) using the techniques of Diekmann *et al.* (1990) and van den Driessche and Watmough (2002).

In this article, we will study the effects of preventions on the dynamic transmission of this disease, as well as the impact of vaccinations on the prevention and control of COVID-19 infection. To this end, we will describe a mathematical model of dynamic transmission of COVID-19, which takes into account the compliance of prevention measures according to the World Health Organization and the vaccination coverage rate of the population studied. We will establish some basic properties of the model to prove its biological feasibility, and deter-

mine the basic reproductive number and other important parameters. We will run numerical simulations to illustrate the impact of these factors on the evolution of this global pandemic.

2. Mathematical Modelling

2.1. Formulation of Mathematical Models

In order to describe the spreading dynamics of the disease, based on the contribution to the prevention measures and vaccination, the population has been partitioned in seven classes also called compartments (see Table 1): S a population consisting of susceptible individuals, V vaccinated individuals, (L) latent individuals, (I_A) asymptomatic infectious individuals, (I_s) symptomatic infectious individuals and (R) recovered individuals.

Some of the following model parameters and their biological justification can be found in [5] [8]-[10].

The population of susceptible is increasing with new birth at a rate Λ. It decreases when the susceptible come into contact with infectious individuals (I_A, I_S) or with the concentration of the virus in the nature C_v. The contact rate between susceptible individuals and, in one hand with asymptomatic infectious individuals is denoted by β_A, in the other hand with symptomatic infectious individuals is β_S. Natural death also contribute to the reduction of susceptible individuals with the rate μ. The natural death rate μ is the same for all compartments. Infected individuals increase in size with the arrival of newly infected individuals from the susceptible population. After the incubation period, the infected individuals begin to decline with the rate ε to become infectious.

Removed individuals increase with recovery in the population of asymptomatic and symptomatic infectious respectively. Assuming the presence of permanent immunity, the rate of removed individuals that become susceptible is denoted by φ and the vaccinated rate is α. The vaccinated individuals increase at a rate α of removed individuals and susceptible individuals and decrease with the rate ξ when the vaccine failed to immunise the vaccinated individuals. We defined p as the proportion of vaccinated individuals whose vaccine succeeded in immunising the patient.

It is assumed that after disinfection of the premises, the concentration of viruses decreases at a rate θ. The concentration of C_v virus produced by the asymptomatic infected individuals is noted χ_A and that produced by symptomatic infected individuals is noted χ_S. Once an outbreak is declared, consideration is given to the implementation of strategies to control and prevent the spread of the disease in the population.

Denoting m_b the set of prevention measures put in place, the force of infection governing the interaction between the susceptible and the infectious and the concentration of the virus in nature is defined by $\lambda = \beta_A I_A + (1-m_b)(\beta_S I_S + \beta_v C_v)$. By infectious, we distinguish two classes that is asymptomatic infectious and symptomatic infectious. The population of asymptomatic infectious increases

with the proportion of latent infected persons (q) at ε rate. Asymptomatic individuals recover at β_A rate while symptomatic individuals recover at a rate δ_S to become immune.

Table 1. Classification of the population.

Group	Symbol	Description
Susceptible	S	People who do not have antibodies and are easily infected by COVID-19
Vaccinated	V	People who have been vaccinated against COVID-19
Latent	L	People who have no apparent symptoms, the symptoms remain hidden and do not manifest
Asymptomatic	I_A	People who are infected but do not have any symptoms
Symptomatic	I_S	People who have obvious symptoms after being infected
Removed	R	People who have recovered from infection
Virus concentrate	C_v	The concentration of the virus in the environment

The mathematical modelling of the dynamic transmission of COVID-19 is shown in the diagram in Figure 1.

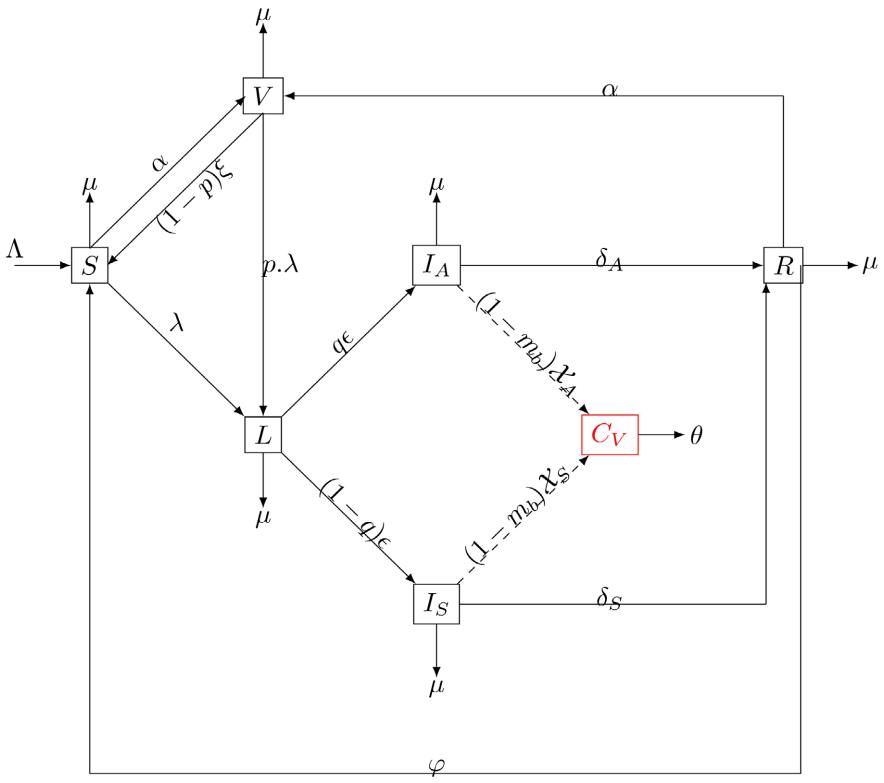

Figure 1. Diagram of dynamic transmission of COVID-19.

Transmission dynamics of COVID-19 are then modelled by the following system.

$$\begin{cases} \dot{S} = \Lambda + (1-p)\xi V + \varphi R - (\lambda + \mu + \alpha) S \\ \dot{V} = \alpha S + \alpha R - (p\lambda + (1-p)\xi + \mu) V \\ \dot{L} = \lambda S + p\lambda V - (\varepsilon + \mu) L \\ \dot{I}_A = q\varepsilon L - (\delta_A + \mu) I_A \\ \dot{I}_S = (1-q)\varepsilon L - (\delta_S + \mu) I_S \\ \dot{R} = \delta_A I_A + \delta_S I_S - (\varphi + \mu + \alpha) R \\ \dot{C}_v = (1-m_b)(\chi_A I_A + \chi_S I_S) - \theta(t) C_v \end{cases} \tag{1}$$

with

$$\lambda = \beta_A I_A + (1-m_b)(\beta_S I_S + \beta_v C_v).$$

2.2. Basic Properties

2.2.1. Local Existence and Positivity of Solution

Let $X = (S, V, L, I_A, I_S, R, C_v)$, $E = \mathbb{R}^7$, E_+ the positive cone of E and B_M is the ball of E centered at 0 and radius $M > 0$.

$$A(t) = \begin{pmatrix} -(\mu+\alpha) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -((1-p)\xi + \mu) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\varepsilon+\mu) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\delta_A+\mu) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\delta_S+\mu) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\varphi+\mu+\alpha) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\theta(t) \end{pmatrix}, t \geq 0$$

and

$$F(X) = \begin{pmatrix} \Lambda + (1-p)\xi V + \varphi R - \lambda S \\ \alpha S + \alpha R - p\lambda V \\ \lambda S + p\lambda V \\ q\varepsilon L \\ (1-q)\varepsilon L \\ \delta_A I_A + \delta_S I_S \\ (1-m_b)(\chi_A I_A + \chi_S I_S) \end{pmatrix}.$$

System (1) can be rewritten as follows:

$$\dot{X}(t) = A(t) X(t) + F(X(t)), \ t \geq 0. \tag{2}$$

Lemma 1.

1. F is lipschitz continuous on bounded of E;
2. For all $M > 0$, there is $\lambda_M > 0$ such that $X \in B_M \cap E_+ \Leftarrow F(X) + \lambda_M \in E_+$.

Proof.

1) Since the function $(t, X) \mapsto F(X(t))$ is C^∞ differentiable on E, then it

is lipschtiz continuous on bounded of E.

2) Let $M>0$ and $X \in B_M \cap E_+$.

We have,

$$F(X)+\lambda_M X = \begin{pmatrix} \Lambda+(1-p)\xi V+\varphi R-\lambda S+\lambda_M S \\ \alpha S+\alpha R-p\lambda V+\lambda_M V \\ \lambda S+p\xi V+\lambda_M L \\ q\varepsilon L+\lambda_M I_A \\ (1-q)\varepsilon L+\lambda_M I_S \\ \delta_A I_A+\delta_S I_S+\lambda_M R \\ (1-m_b)(\chi_A I_A+\chi_S I_S)+\lambda_M C_v \end{pmatrix}.$$

Since we have,

$$\lambda_M-\lambda \geq \lambda_M - M\left(\beta_A+(1-m_b)(\beta_S+\beta_v)\right)$$

and

$$\lambda_M-p\lambda \geq \lambda_M - M\left(\beta_A+(1-m_b)(\beta_S+\beta_v)\right).$$

then it follows that this property is satisfied once

$\lambda_M \geq M\left(\beta_A+(1-m_b)(\beta_S+\beta_v)\right)$.□

We obtain the following theorem.

Theorem 1. *Let* $X_0=(S_0,V_0,L_0,I_{A0},I_{S0},R_0,C_{v0})\in E_+$. *The Cauchy problem associated to system* (2) *for the initial condition* $(0,X_0)$ *has an unique maximal solution in* E_+.

Proof. From lemma 1 and according to the Cauchy-Lipschitz theorem [11], Equation (2) has a unique maximum solution for all related Cauchy problems, defined on an interval $[0,T_{\max}[$, $T_{\max}>0$.

To show the positivity of the solution, we rewrite the Equation (2):

$$\dot{X}(t)=\left(A(t)-\lambda_M I_E\right)X(t)+F_M\left(X(t)\right),\ t\geq 0. \tag{3}$$

where $F_M=F+\lambda_M I_E$ and λ_M choosen such that $F_M(X)\in E_+$ for all $X\in E_+$.

The solution of the Cauchy problem associated with the differential Equation (3) and initial condition $(0,X_0)$, using Duhamel's formula, is given by

$$X(t)=\mathrm{e}^{\int_0^t (A(s)-\lambda_M I_E)\mathrm{d}s}X_0+\int_0^t \mathrm{e}^{-\int_s^t (A(s)-\lambda_M I_E)\mathrm{d}s}F_M\left(X(s)\right)\mathrm{d}s,\ t\geq 0.$$

This shows that the maximal solution to the Cauchy problem associated to (3) (*i.e.* Equation (2)) is positive. Prove that the result is valuable.□

2.2.2. Boundedness of Solutions

Taking into account (1) model, we have divided it into two sub-sections: the human population and the viral population. And so, the dynamics of the total human population satisfies the following conditions

$$\dot{N}=\Lambda-\mu N \tag{4}$$

Integrating the above differential equality yield

$$0 \le N(t) = \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t} \le \max\left(\frac{\Lambda}{\mu}, N(0) \right), \ t \ge 0.$$

where $N(0)$ is the initial value of $N(t)$.

Let's note $\Delta = \max\left(\frac{\Lambda}{\mu}, N(0) \right)$.

Using $\chi_A I_A(t) + \chi_S I_S(t) \le (\chi_A + \chi_S)(I_A(t) + I_S(t)) \le (\chi_A + \chi_S)\Delta$, the dynamics of viral population satisfies

$$\dot{C}_v \le (1 - m_b)(\chi_A + \chi_S)\Delta - \theta(t) C_v \le (1 - m_b)(\chi_A + \chi_S)\Delta - \underline{\theta} C_v, \tag{5}$$

where $\underline{\theta} = \inf_{t \ge 0} \theta(t)$.

Integrating (5) gives

$$0 \le C_v \le \frac{(1 - m_b)(\chi_A + \chi_S)\Delta}{\underline{\theta}} + \left(C_v(0) - \frac{(1 - m_b)(\chi_A + \chi_S)\Delta}{\underline{\theta}} \right) e^{-\underline{\theta} t}, \ t \ge 0$$

where $C_v(0)$ is the initial value of $C_v(t)$.

This implies that $0 \le C_v(t) \le \max\left(C_v(0), \frac{(1 - m_b)(\chi_A + \chi_S)\Delta}{\underline{\theta}} \right) \equiv \tilde{\Delta}$ for all $t \ge 0$.

It then follows that

$$N(t) + C_v(t) \le \Delta + \tilde{\Delta}, \ \forall t \ge 0.$$

We have the following proposition.

Proposition 1. *Every maximal solution of the system* (1) *is global and the region*

$$\Omega = \left\{ (S, V, L, I_A, I_S, R, C_v) \in E_+ : N \le \Delta \text{ and } C_v \le \tilde{\Delta} \right\}$$

is positively invariant and attractive for the model (1).

2.3. Asymptotic Properties: Autonomous Case

2.3.1. Disease-Free Equilibrium and Basic Reproductive Number

Disease-free equilibrium (DFE) is obtained when there are no infected individuals in the population. For the system (1), the equilibrium DFE is given by

$$x_0 = \left(S^0, V^0, 0, 0, 0, 0, 0 \right)$$

where $S^0 = \frac{\Lambda(\mu + (1-p)\xi)}{\mu(\alpha + \mu + (1-p)\xi)}$ and $V^0 = \frac{\Lambda\alpha}{\mu(\alpha + \mu + (1-p)\xi)}$.

In mathematical biology, the basic reproductive number, which is highly essential for qualitative analysis of a model, is defined using the next-generation matrix method employed in [12]. Following the notations of [12], the F and V matrices for the new infection terms and the remaining transfer terms are, respectively, given by

$$F = \begin{pmatrix} 0 & \beta_A(S^0 + pV^0) & (1 - m_b)\beta_S(S^0 + pV^0) & (1 - m_b)\beta_v(S^0 + pV^0) \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$-V=\begin{pmatrix}\mu+\varepsilon & 0 & 0 & 0\\ -q\varepsilon & \mu+\delta_A & 0 & 0\\ -(1-q)\varepsilon & 0 & \mu+\delta_S & 0\\ 0 & -(1-m_b)\chi_A & -(1-m_b)\chi_S & \theta\end{pmatrix}$$

The inverse matrix of V is given by:

$$-V^{-1}=\begin{pmatrix}\frac{1}{\mu+\varepsilon} & 0 & 0 & 0\\ \frac{q\varepsilon}{(\mu+\varepsilon)(\mu+\delta_A)} & \frac{1}{\mu+\delta_A} & 0 & 0\\ \frac{(1-q)\varepsilon}{(\mu+\varepsilon)(\mu+\delta_S)} & 0 & \frac{1}{\mu+\delta_S} & 0\\ \frac{\varepsilon(1-m_b)}{\theta(\mu+\varepsilon)}\left(\frac{\chi_A q}{\mu+\delta_A}+\frac{\chi_S(1-q)}{\mu+\delta_S}\right) & \frac{\chi_A(1-m_b)}{\theta(\mu+\delta_A)} & \frac{\chi_S(1-m_b)}{\theta(\mu+\delta_S)} & \frac{1}{\theta}\end{pmatrix}$$

Based on the technique of the next-generation matrix, the basic reproductive number is defined as the spectral radius of matrix $-FV^{-1}$ noted $\mathcal{R}_0=\rho\left(-FV^{-1}\right)$ (see [12] [13]).

Thus

$$\mathcal{R}_0=\frac{\Lambda\varepsilon\left[\mu+(1-p)\xi+\alpha p\right]\left(\frac{q\beta_A}{\mu+\delta_A}+\frac{(1-q)\beta_S}{\mu+\delta_S}+\frac{(1-m_b)\chi_A\beta_v}{\theta}\left(\frac{q}{\mu+\delta_A}+\frac{1-q}{\mu+\delta_S}\right)\right)}{\mu(\mu+\varepsilon)(\mu+\alpha+(1-p)\xi)}.$$

we assert the following result based on Theorem 2 by Van Den Driessche and Watmough in [12].

Theorem 2. *If* $\mathcal{R}_0<1$ *then the disease-free equilibrium of* (1) *is locally asymptotically stable, it is unstable if* $\mathcal{R}_0\geq 1$.

2.3.2. Global Stability of Disease-Free Equilibrium

The stability of the disease-free equilibrium is not just local, it is also global. To establish the global stability of the disease-free equilibrium, we use the result of Kamgang and Sallet [14].

Using [14] notations, the (1) model system is rephrased as:

$$\begin{cases}\dot{Y}_S=B_1(X)\left(Y_S-Y_S^0\right)+B_{12}(X)Y_I\\ \dot{Y}_I=B_2(X)Y_I\end{cases}\tag{6}$$

where Y_S is class of non infected individuals (S,V,R) and the vector Y_I is class of infected individuals (L,I_A,I_S,C_v). We have $Y_S=(S,V,R)^{\mathrm{T}}$, $Y_I=(L,I_A,I_S,C_v)^{\mathrm{T}}$, $X=(Y_S,Y_I)^{\mathrm{T}}$ and $Y_S^0=\left(S^0,V^0,R^0\right)^{\mathrm{T}}$, with

$$B_1(X)=\begin{pmatrix}-(\alpha+\mu) & (1-p)\xi & \phi\\ \alpha & -\left((1-p)\xi+\mu\right) & \alpha\\ 0 & 0 & -(\alpha+\phi+\mu)\end{pmatrix},$$

$$B_{12}(X)=\begin{pmatrix}0 & -\beta_A S & -(1-m_b)\beta_S S & -(1-m_b)\beta_v S\\ 0 & -p\beta_A V & -p(1-m_b)\beta_S V & -p(1-m_b)\beta_v V\\ 0 & \delta_A & \delta_S & 0\end{pmatrix}$$

and

$$B_2(X)=\begin{pmatrix}-(\varepsilon+\mu) & \beta_A(S+pV) & (1-m_b)\beta_S(S+pV) & (1-m_b)\beta_v(S+pV)\\ q\varepsilon & -(\delta_A+\mu) & 0 & 0\\ (1-q)\varepsilon & 0 & -(\delta_S+\mu) & 0\\ 0 & (1-m_b)\chi_A & (1-m_b)\chi_S & -\theta\end{pmatrix}$$

A direct calculation shows that all the eigenvalues of $B_1(X)$ are real and negative. Thus, the system $\dot{Y}_S = B_1(X)\left(Y_S - Y_S^0\right)$ is globally asymptotically stable at equilibrium $Y_S^0 = \left(S^0, V^0, R^0\right)^{\mathrm{T}}$. Let's note that $A_2(X)$ is a Metzler matrix, *i.e.* a matrix such that the off-diagonal terms are non-negative [15] [16].

Consider the bounded set $\mathcal{D}$:

$$\mathcal{D} = \left\{\left(Y_S, Y_I\right) \in \Omega, Y_S \neq 0\right\}$$

Recall the following theorem in [14].

Theorem 3. *The system* (1) *is of class* C^1*, defined E. If*

(1) $\mathcal{D}$ is positively invariant relative to (1).

(2) The system $\dot{Y}_S = B_1(X)\left(Y_S - Y_S^0\right)$ is globally asymptotically stable at equilibrium $Y_S^0 = \left(S^0, V^0, R^0\right)$ on the canonical projection of $\mathcal{D}$ onto $\left(\mathbb{R}_+\right)^3$.

(3) Considering any $x \in \mathcal{D}$, the matrix $B_2(X)$ is Metzler irreducible.

(4) There exists a matrix $\bar{B}_2$ which is an upper bound of the set $\mathbb{M} = \left\{B_2(x) \in \mathcal{M}_4(\mathbb{R}) : x \in \mathcal{D}\right\}$ with the property that if $\bar{B}_2 \in \mathbb{M}$, for any $\bar{x} \in \mathcal{D}$, such that $B_2(\bar{x}) = \bar{B}_2$, therefore $\bar{x} \in \mathbb{R}^3 \times \left\{0_{\mathbb{R}^4}\right\}$.

(5) The stability modulus of $\bar{B}_2$, $\alpha\left(\bar{B}_2\right) = \max_{\lambda \in \rho\left(\bar{B}_2\right)} Re(\lambda)$ satisfied $\alpha\left(\bar{B}_2\right) \leq 0$.

Then the DFE is GAS in $\mathcal{D}$.

Now let's check the assumptions of the previous theorem: it's obvious that conditions (1-3) of the theorem are satisfied. An upper bound on the set of matrices B_2, which is the matrix $\bar{B}_2$, is given by

$$\bar{B}_2=\begin{pmatrix}-(\varepsilon+\mu) & \beta_A\left(S^0+pV^0\right) & (1-m_b)\beta_S\left(S^0+pV^0\right) & (1-m_b)\beta_v\left(S^0+pV^0\right)\\ q\varepsilon & -(\delta_A+\mu) & 0 & 0\\ (1-q)\varepsilon & 0 & -(\delta_S+\mu) & 0\\ 0 & (1-m_b)\chi_A & (1-m_b)\chi_S & -\theta\end{pmatrix}.$$

To check condition (5) in theorem 3, we will use the following useful lemma which is the characterization of Metzler stable matrices:

Lemma 2. *Let M be a square Metzler matrix written in block form* $\begin{pmatrix}A & B\\ C & D\end{pmatrix}$ *with A and D square matrices. M is Metzler stable if and only if matrices D and* $A - BD^{-1}C$ *are Metzler stable.*

Matrix $\bar{B}_2$ can be express in the form of the matrix M in lemma 2, with:

$$A=\begin{pmatrix} -(\varepsilon+\mu) & \beta_A\left(S^0+pV^0\right) \\ q\varepsilon & -(\delta_A+\mu) \end{pmatrix},$$

$$B=\begin{pmatrix} (1-m_b)\beta_S\left(S^0+pV^0\right) & (1-m_b)\beta_v\left(S^0+pV^0\right) \\ 0 & 0 \end{pmatrix},$$

$$C=\begin{pmatrix} (1-q)\varepsilon & 0 \\ 0 & (1-m_b)\chi_A \end{pmatrix} \text{ and } D=\begin{pmatrix} -(\delta_S+\mu) & 0 \\ (1-m_b)\chi_S & -\theta \end{pmatrix}$$

Clearly, D is a stable Metzler matrix. Then, after some computations, we obtain $A-BD^{-1}C$ is a stable Metzler matrix if and only if $\mathcal{R}_0 \le 1$.

So we have the following result:

Theorem 4. *If* $\mathcal{R}_0 \le 1$ *then the disease-free equilibrium of* (1) *is globally asymptotically stable.*

2.3.3. Existence of Endemic Equilibrium and Stability

The equilibria of system (1) are the elements $\left(S^*, V^*, L^*, I_A^*, I_S^*, R^*, C_v^*\right)$ which verify the following system:

$$\begin{cases} \Lambda+(1-p)\xi V^*+\varphi R^*-\left(\lambda^*+\mu+\alpha\right)S^*=0 \\ \alpha S^*+\alpha R^*-\left(p\lambda^*+(1-p)\xi+\mu\right)V^*=0 \\ \lambda^* S^*+p\lambda^* V^*-(\varepsilon+\mu)L^*=0 \\ q\varepsilon L^*-(\delta_A+\mu)I_A^*=0 \\ (1-q)\varepsilon L^*-(\delta_S+\mu)I_S^*=0 \\ \delta_A I_A^*+\delta_S I_S^*-(\varphi+\mu+\alpha)R^*=0 \\ (1-m_b)\left(\chi_A I_A^*+\chi_S I_S^*\right)-\theta C_v^*=0 \end{cases} \tag{7}$$

where $\lambda^*=\beta_A I_A^*+(1-m_b)\left(\beta_S I_S^*+\beta_v C_v^*\right)$.

After a few calculations, we get

$$\begin{cases} S^*=\dfrac{\Lambda\left(p\lambda^*+(1-p)\xi+\mu\right)+a_6 f\left(\lambda^*\right)L^*}{h\left(\lambda^*\right)} \\ V^*=\dfrac{\alpha\Lambda+a_6 g\left(\lambda^*\right)L^*}{h\left(\lambda^*\right)} \\ L^*=\dfrac{\lambda^*\Lambda\left(p\alpha+p\lambda^*+(1-p)\xi+\mu\right)}{(\varepsilon+\mu)h\left(\lambda^*\right)-a_6\lambda^*\left(f\left(\lambda^*\right)+pg\left(\lambda^*\right)\right)} \\ I_A^*=a_4 L^* \\ I_S^*=a_5 L^* \\ R^*=a_6 L^* \\ C_v^*=a_7 L^*, \end{cases} \tag{8}$$

where

$$\begin{cases} a_4 = \dfrac{q\varepsilon}{\delta_A + \mu} \\ a_5 = \dfrac{(1-q)\varepsilon}{\delta_S + \mu} \\ a_6 = \dfrac{\varepsilon}{\varphi + \mu + \alpha}\left(\dfrac{q\delta_A}{\delta_A + \mu} + \dfrac{(1-q)\delta_S}{\delta_S + \mu}\right) \\ a_7 = \dfrac{\varepsilon(1-m_b)}{\theta}\left(\dfrac{q\chi_A}{\delta_A + \mu} + \dfrac{(1-q)\chi_S}{\delta_S + \mu}\right) \end{cases}$$

with the real functions f, g and h defined respectively by

$$f(\lambda^*) = \varphi\left(p\lambda^* + (1-p)\xi + \mu\right) + \alpha(1-p)\xi,$$

$$g(\lambda^*) = \alpha\left(\lambda^* + \mu + \alpha + \varphi\right),$$

$$h(\lambda^*) = \left(\lambda^* + \mu + \alpha\right)\left(p\lambda^* + (1-p)\xi + \mu\right) - \alpha(1-p)\xi$$

and the real λ^* verifies the following equation

$$\lambda^* \frac{\Lambda\left[p\alpha + p\lambda^* + (1-p)\xi + \mu\right]\left[\beta_A a_4 + (1-m_b)(\beta_S a_5 + \beta_v a_7)\right]}{(\varepsilon+\mu)h(\lambda^*) - a_6\lambda^*\left(f(\lambda^*) + pg(\lambda^*)\right)} = \lambda^*. \quad (9)$$

From Equation (9), we have $\lambda^* = 0$ or

$$\frac{\Lambda\left[p\alpha + p\lambda^* + (1-p)\xi + \mu\right]\left[\beta_A a_4 + (1-m_b)(\beta_S a_5 + \beta_v a_7)\right]}{(\varepsilon+\mu)h(\lambda^*) - a_6\lambda^*\left(f(\lambda^*) + pg(\lambda^*)\right)} = 1. \quad (10)$$

From inequality $\mu > 0$, it is easy to check that $(\varepsilon+\mu)h(\lambda^*) - a_6\lambda^*\left(f(\lambda^*) + pg(\lambda^*)\right) > 0$ for $\lambda^* \geq 0$. Therefore, we deduce that (10) is equivalent to following equation

$$a_1\lambda^{*2} + a_2\lambda^* + a_3 = 0, \quad (11)$$

where $a_1 = p\left[1 - \dfrac{a_6(\varphi+\alpha)}{\varepsilon+\mu}\right]$, $a_3 = \mu\left(\mu + \alpha + (1-p)\xi\right)(1-\mathcal{R}_0)$ and

$$a_2 = \frac{(\varepsilon+\mu)\left(p\mu + p\alpha + (1-p)\xi + \mu\right) - a_6\left[\varphi\left((1-p)\xi + \mu\right) + \alpha\left(p\mu + p\alpha + p\varphi + (1-p)\xi\right)\right]}{\varepsilon+\mu}$$

$$- p\mathcal{R}_0\frac{\mu\left(\alpha + (1-p)\xi + \mu\right)}{\left[\alpha p + (1-p)\xi + \mu\right]}.$$

From all the above, we can state the following theorem.

Theorem 5. *If* $\mathcal{R}_0 > 1$, *system* (1) *has two equilibria, the disease-free equilibrium and an endemic equilibrium.*

Proof. We have $a_1 > 0$ and $a_3 < 0$, because $\mathcal{R}_0 > 1$. Then the second-degree Equation (11) has a unique positive solution λ^* which corresponds to the endemic equilibrium $\left(S^*, V^*, L^*, I_A^*, I_S^*, R^*, C_v^*\right)$ given by system (8). □

2.4. Asymptotic Properties: Non-Autonomous Case

Let $\mathcal{S}$ be the threshold defined by:

$$\underline{\mathcal{S}} = \frac{\min\{\mu+\beta_A, \mu+\delta_S, \mu+\varepsilon\}}{\max\{\mu+\beta_A, \mu+\delta_S, \mu+\varepsilon\}}\mathcal{L}, \tag{12}$$

where

$$\mathcal{L} = \min\left(\frac{p\Lambda}{\frac{\Lambda\beta_{\max}}{\mu}\left(1+\frac{\chi_{\max}}{\mu_{\max}+\mu}(1-m_b)\right)}, \varepsilon Q\right)$$

with

$$Q = \frac{q}{\mu+\beta_A} + \frac{1-q}{\mu+\delta_S},$$

$$\beta_{\max} = \max(\beta_A, \beta_S, \beta_v), \quad \chi_{\max} = \max(\chi_A, \chi_S),$$

and

$$\bar{\mathcal{S}} = \left(\frac{\beta_0(1+\mathcal{C}_0)(1+Q\cdot\varepsilon)}{\mu+\varepsilon}\right)\frac{\bar{\eta}}{\underline{\eta}}$$

with

$$\bar{\eta} = \max\{\mu+\beta_A, \mu+\delta_S, \mu+\varepsilon\}, \quad \underline{\eta} = \min\{\mu+\beta_A, \mu+\delta_S, \mu+\varepsilon\},$$

$$\beta_0 = \frac{\beta_{\max}\Lambda}{\mu}, \quad \mathcal{C}_0 = \frac{\beta_v C_v \chi_{\max}(1-m_b)(r_0+r_1)}{\beta_{\max}}$$

Let's also consider the function

$$f(t) = \frac{I_A(t)}{\mu+\beta_A} + \frac{I_S(t)}{\mu+\delta_S} + \frac{L(t)}{\mu+\varepsilon}$$

From all the above, we assume that $x(0) \in \Omega$

2.4.1. Disease Extinction

Theorem 6. *If* $\bar{\mathcal{S}} < 1$, *then the disease dies out, i.e* $I_A(t) \to 0$ *and* $I_S(t) \to 0$

Proof.

$$\begin{aligned}
&\frac{\mathrm{d}f(t)}{\mathrm{d}t} \le \frac{\beta_{\max}}{\mu+\varepsilon}\left(I_A + I_S + \frac{\beta_v C_v \cdot C}{\beta_{\max}}\right)(S + p\cdot V) + Q\cdot\varepsilon L - \underline{\eta}\cdot f \\
&\frac{C_v(t)}{\mathrm{d}t} \le (1-m_b)\chi_{\max}(I_A + I_S) - \frac{1}{r_0+r_1}\cdot C_v
\end{aligned} \tag{13}$$

then

$$\begin{aligned}
C_v(t) &\le \chi_{\max}(1-m_b)(r_0+r_1)\bar{\eta}\,\bar{f} \\
&+\left(C_v(0) - \chi_{\max}(1-m_b)(r_0+r_1)\bar{\eta}\,\bar{f}\right)\exp\left(-\frac{t}{r_0+r_1}\right)
\end{aligned} \tag{14}$$

thus

$$C_v(t) \le \chi_{\max}(1-m_b)(r_0+r_1)\bar{\eta}\,\bar{f} \tag{15}$$

we deduce then

$$C_v\left(S+p\cdot V\right)\le\frac{\Lambda\chi_{\max}\left(1-m_b\right)\left(r_0+r_1\right)\bar{\eta}\,\bar{f}}{\mu} \tag{16}$$

Introducing inequality (16) in following inequality

$$\frac{\mathrm{d}L}{\mathrm{d}t}=\beta_A\cdot I_A+\left(1-m_b\right)\cdot\left(\beta_S\cdot I_S+\beta_v\cdot C_v\right)\left(S+p\cdot V\right)-\left(\mu+\varepsilon\right)L \tag{17}$$

Noting

$$\beta_0=\frac{\beta_{\max}\Lambda}{\mu},\ \ \mathcal{C}_0=\frac{\beta_v C_v\chi_{\max}\left(1-m_b\right)\left(r_0+r_1\right)}{\beta_{\max}}$$

we obtain:

$$\frac{\mathrm{d}L}{\mathrm{d}t}\le\beta_0\left(1+\mathcal{C}_0\right)\bar{\eta}\,\bar{f}-\left(\mu+\varepsilon\right)L \tag{18}$$

then

$$L\left(t\right)\le\frac{\beta_0\left(1+\mathcal{C}_0\right)\bar{\eta}\,\bar{f}}{\mu+\varepsilon}+\left(L\left(0\right)-\frac{\beta_0\left(1+\mathcal{C}_0\right)\bar{\eta}\,\bar{f}}{\mu+\varepsilon}\right)\exp\left(-\left(\mu+\varepsilon\right)t\right) \tag{19}$$

We deduce that

$$L\le\frac{\beta_0\left(1+\mathcal{C}_0\right)\bar{\eta}\,\bar{f}}{\mu+\varepsilon} \tag{20}$$

introducing inequalities (16) and (20) in inequality (13), we deduce the following inequality

$$\frac{\mathrm{d}f}{\mathrm{d}t}\le\frac{\beta_0\left(1+\mathcal{C}_0\right)\left(1+Q\cdot\varepsilon\right)}{\mu+\varepsilon}\bar{\eta}\,\bar{f}-\underline{\eta}\,f \tag{21}$$

From Equation (21) Using a fluctuation method (see [10] [17]) one conludes that

$$0\le\frac{\beta_0\left(1+\mathcal{C}_0\right)\left(1+Q\cdot\varepsilon\right)}{\mu+\varepsilon}\bar{\eta}\,\bar{f}-\underline{\eta}\,\bar{f} \tag{22}$$

we deduce then

$$0\le\left(\frac{\beta_0\left(1+\mathcal{C}_0\right)\left(1+Q\varepsilon\right)\bar{\eta}}{\left(\mu+\varepsilon\right)\underline{\eta}}-1\right)\underline{\eta}\,\bar{f} \tag{23}$$

Thus we obtain

$$\left(\bar{\mathcal{S}}-1\right)\underline{\eta}\,\bar{f}\ge 0 \tag{24}$$

Then $\bar{\mathcal{S}}<1$ implies that $\bar{f}=0$. We therefore conclude that the disease is extinguished when $\bar{\mathcal{S}}<1$, *i.e.* $I_A\left(t\right)\to 0$, $I_S\left(t\right)\to 0$ and $L\left(t\right)\to 0$ as $t\to\infty$.□

2.4.2. Disease Persistence

This part is devoted to formulate the disease persistence property. To establish the conditions that guarantee the persistence of the disease in the population, we use the mathematical properties cited in [18]-[23] are used.

Theorem 7. *Suppose that there exists* $t_0 \geq 0$ *, such that*

$$L(t_0) + A(t_0) + I(t_0) > 0 \tag{25}$$

If $\underline{\mathcal{S}} > 1$, the disease described by the (2) system is persistent.

Proof. Suppose by contradiction that the disease is not uniformly weakly persistent. Suppose there exists $\sigma > 0$, such that.

$$\limsup_{t \to +\infty} \left(L(t) + I_A(t) + I_S(t) \right) \leq \sigma \quad \text{as} \quad t \to +\infty$$

The sum of the first two equations of system (2) gives us the following equation:

$$\dot{S} + \dot{V} \geq \Lambda - \left(\frac{\Lambda \beta_{\max}}{\mu} \left(1 + \frac{\chi_{\max}}{\mu_{\max}} (1 - m_b) \right) + \mu \right) (S + V) \tag{26}$$

Solving the differential inequation (26), we obtain the following inequality:

$$S + V \geq \frac{\Lambda}{\frac{\Lambda \beta_{\max}}{\mu} \left(1 + \frac{\chi_{\max}}{\mu_{\max}} (1 - m_b) \right) + \mu} \tag{27}$$

One deduces that:

$$S + p \cdot V \geq \frac{p \cdot \Lambda}{\frac{\Lambda \beta_{\max}}{\mu} \left(1 + \frac{\chi_{\max}}{\mu_{\max}} (1 - m_b) \right) + \mu} \tag{28}$$

Introducing the inequality (28) in system (2), the following is obtained:

$$\frac{\mathrm{d}f}{\mathrm{d}t} \geq \frac{\beta_0 \Lambda \cdot p}{\mu + \beta_{\max} \frac{\Lambda}{\mu} \left(1 + \frac{\chi_{\max}}{\mu_{\min}} (1 - m_b) \right)} \left(I_A + I_S + \frac{\beta_v \cdot C_v}{\beta_{\min}} \right) + \varepsilon \cdot Q \cdot L - \overline{\eta} f \tag{29}$$

From where:

$$\frac{\dot{f}}{f} \geq \frac{p\Lambda}{\frac{\Lambda \beta_{\max}}{\mu} \left(1 + \frac{\chi_{\max}}{\mu_{\max}} (1 - m_b) \right) + \mu} \left(\frac{I_A + I_S}{f} \right) + \frac{\varepsilon Q L}{f} - \overline{\eta} \tag{30}$$

Note

$$\mathcal{L} = \min \left(\frac{p\Lambda}{\frac{\Lambda \beta_{\max}}{\mu} \left(1 + \frac{\chi_{\max}}{\mu_{\max}} (1 - m_b) \right) + \mu}, \varepsilon \cdot Q \right)$$

Equation (30) implies the following:

$$\frac{\mathrm{d}}{\mathrm{d}t} \left(\ln \left(f(t) \right) \right) \geq \left(\frac{\eta \mathcal{L}}{\overline{\eta}} - 1 \right) \overline{\eta} \tag{31}$$

one deduces then:

$$\ln \left(f(t) \right) - \ln \left(f(t_0) \right) \geq \left(\frac{\eta \mathcal{L}}{\overline{\eta}} - 1 \right) \overline{\eta} (t - t_0) \tag{32}$$

Finally one gets

$$f(t) \geq f(t_0)\exp(\underline{\mathcal{S}}-1)\overline{\eta}(t-t_0) \tag{33}$$

We find that:

$\limsup_{t\to+\infty} f(t) = +\infty$ since $\underline{\mathcal{S}} > 1$ contradicts the fact that $f(t)$ is bounded. so, if $\underline{\mathcal{S}} > 1$, then the disease described by the system (2) is persistence.□

3. Simulations

Here, we focus on the simulation considering a population of 10 thousand inhabitants. These simulations lay emphasis on asymptomatic behaviour of result of task 1 describing the transmission method of the new version of the coronavirus in the given population. The different representations of the diagram were obtained by diversifying the parameters in such a way that m_b represents the prevention measures as proposed by the World Health Organization and certain patners in charge of health. We equally vary α who is rate of increase in vaccinated people who recover. The different values used in simulation are given in Table 2. We have divided the simulations into two sub-sections: in the first sub-section, we run several simulations taking into account the impact of prevention measures on the spread dynamics of coronavirus, but without vaccination, and in the second section, we run different simulations on the effect of

Table 2. Biological description, values and units of the parameters of model.

Parameter	description	Value	Reference
Λ	Human birth rate	0.05 per day	[6]
β_{I_A}	contact rate of spread between S and A	0.1 per day	[6]
β_{I_S}	contact rate of spread between S and I	0.15 per day	[24]
β_{C_v}	contact rate of spread between S and C_v	0.2 per day	[6]
φ	Immune loss rate	0.0027 per day	[6]
ε	duration of incubation period	0.14	[24]
θ	rate where the concentration of the virus decreases	5×10^{-2} per day	[24]
α	rate of increase in vaccinated people who recover	0 per day	[24]
δ_A	recovery rate of asymptomatic infected	0.1 per day	[6] [24]
δ_S	recovery rate of symptomatic infected	0.07 per day	[6] [24]
μ	Natural death rate	5×10^{-5} per day	[25]
ξ	vaccination failure rate	0.01 per day	[25]
	Virus shedding rate by asymptomatic infected	2.3	[25]
χ_S	Virus shedding rate by symptomatic infected	0.3	[25]
p	proportion of the people vaccinated	0.4	
q	proportion of the	0.65	

vaccination on this pandemic. Simulations are therefore carried out to test the model's behavior over time, and the following information can be drawn from the model's parameters.

3.1. Effect of Prevention Measures on the Dynamics Spreading of Coronavirus without Vaccination

In this sub-section, we show curves obtained after simulations of our system taking into account the effect of prevention measures taken by the World Health Organization and international partners in the health sector, but without taking vaccination or the health pass into account. Here we consider $\alpha = 0$, but we vary the values of the parameter m_b, which is in fact the set of prevention measures. Thus we obtain the black curve ($m_b = 0$), the red curve ($m_b = 0.15$), the blue curve ($m_b = 0.30$), the green curve ($m_b = 0.45$), and finally the pink curve ($m_b = 0.6$).

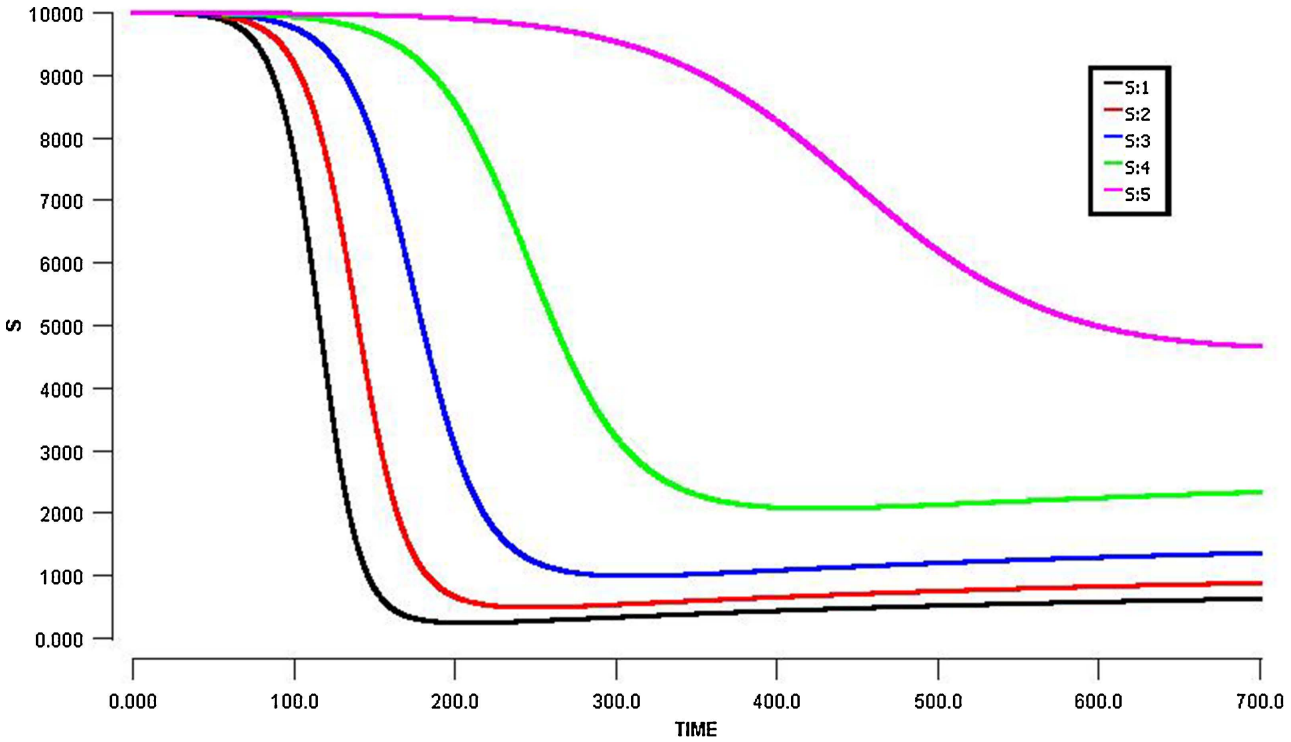

Figure 2. Evolution of number cases of COVID-19 for $\alpha = 0$, $m_b = 0$, $m_b = 0.15$, $m_b = 0.30$, $m_b = 0.45$, $m_b = 0.6$ and $\beta_{I_A} = 0.1$, $\beta_{I_S} = 0.15$, $\beta_{C_v} = 0.2$.

Figure 2 illustrates the effect of the people complying with prevention measures on changes in number of new infections among susceptible people. The simulation was carried out by varying the values of the prevention compliance parameter. We can see that the proportion of susceptible people decreases as compliance with prevention measures decreases over time. This shows the positive impact of compliance with prevention measures by susceptible individuals.

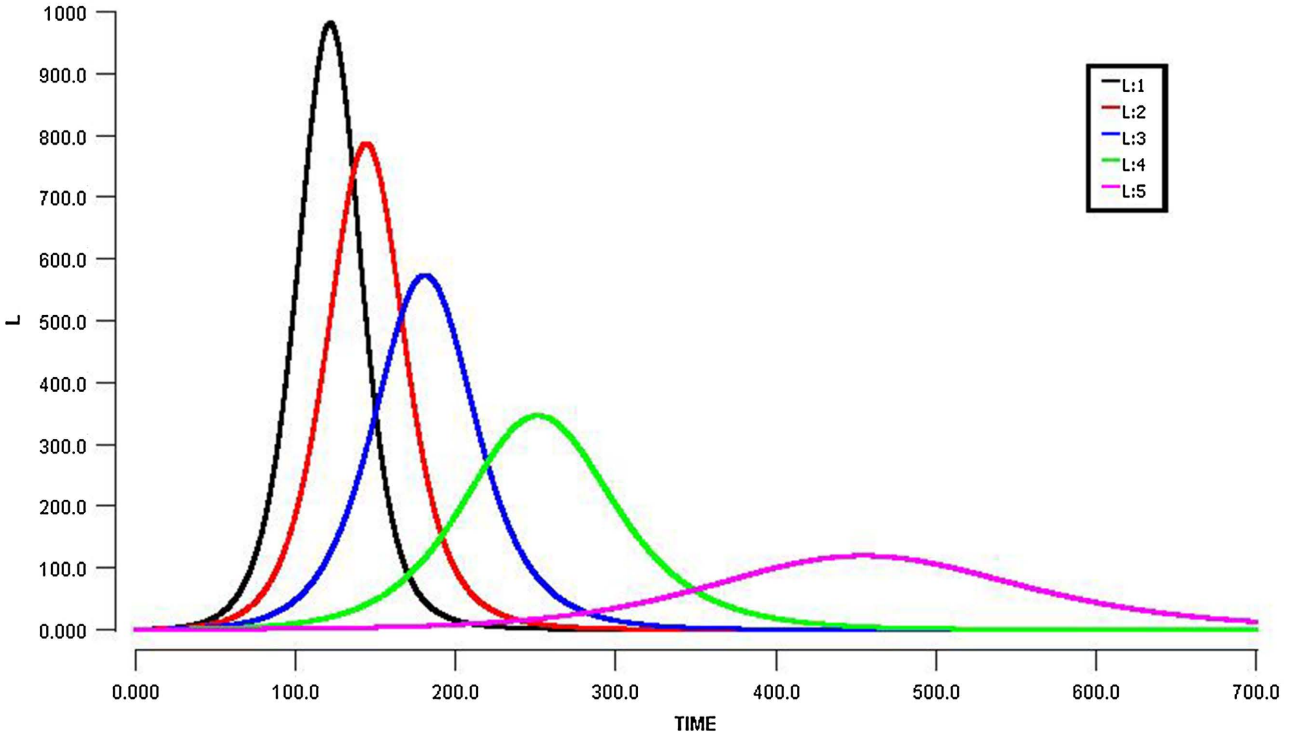

Figure 3. Evolution of number cases of COVID-19 for $\alpha = 0$, $m_b = 0$, $m_b = 0.15$, $m_b = 0.30$, $m_b = 0.45$, $m_b = 0.6$ and $\beta_{I_A} = 0.1$, $\beta_{I_S} = 0.15$, $\beta_{C_v} = 0.2$.

Figure 3 illustrates the effect of people complying with prevention measures on evolution of new cases of latent individuals. By varying the values of the prevention measures parameter, we can see that: the more the latent population decreases, the faster it reaches its maximum before stabilizing. This shows that compliance with prevention measures by latent individuals prevents the disease from manifesting itself more rapidly.

Figure 4 illustrates the effect of the people complying with prevention measures on evolution of number of new cases of asymptomatic infected. The case where of people complying with prevention measures is zero ($m_b = 0$), the disease evolves very rapidly in the population, reaching a peak of almost 40 new cases per day by day 100^e. By increasing of people complying with the prevention measures, the disease progresses more slowly, and the number of new cases is reduced to a low peak. We can see that for 60 per cent of people complying with the prevention measures ($m_b = 0.6$), the number of new cases per day is only 10 when the peak is reached. This peak is reached around 500 days after the onset of the disease.

Figure 5 illustrates the effect of the proportion of people complying with prevention measures on changes in the number of new cases among symptomatically infected people. The behavior of the curves is identical to that observed in **Figure 4**, although it should be noted that the number of cases is higher for the symptomatically infected than for the asymptomatically infected.

Figure 6 shows the effect of people complying with prevention measures on

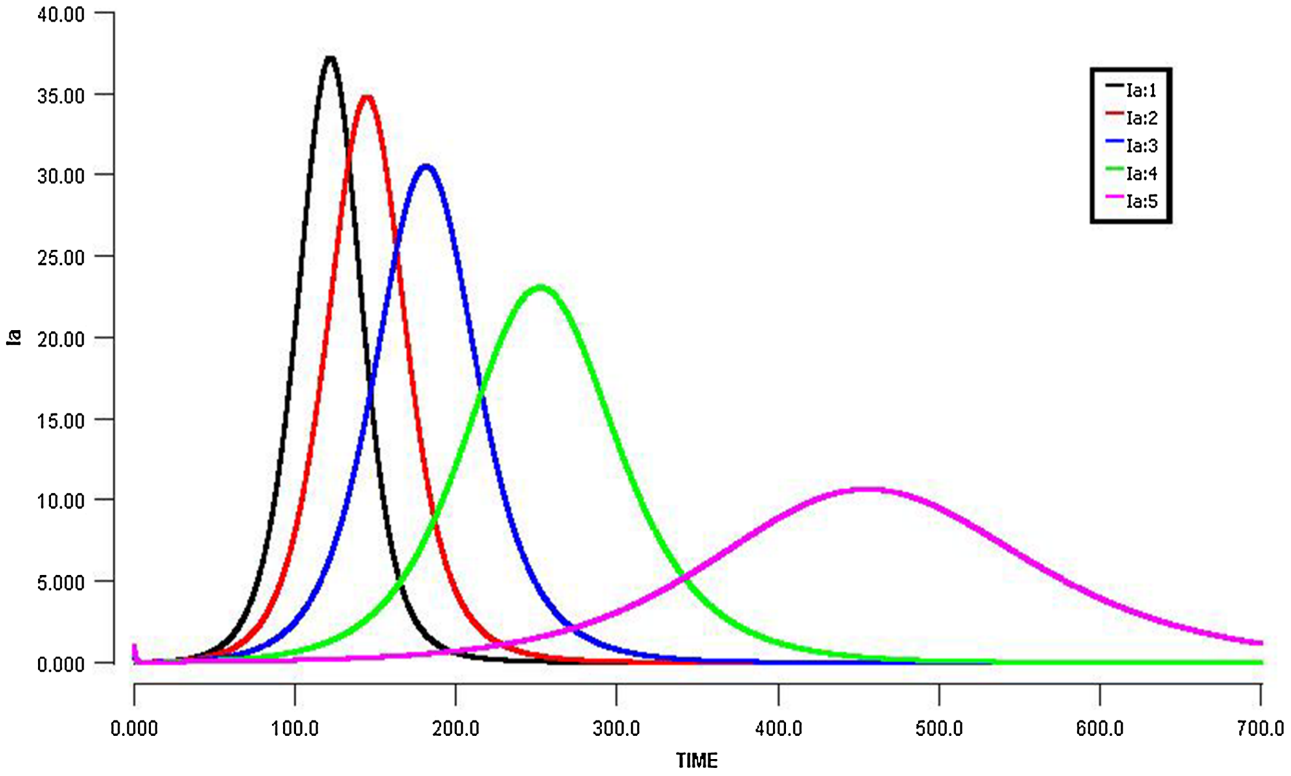

Figure 4. Evolution of number cases of COVID-19 for $\alpha = 0$, $m_b = 0$, $m_b = 0.15$, $m_b = 0.30$, $m_b = 0.45$, $m_b = 0.6$ and $\beta_{I_A} = 0.1$, $\beta_{I_S} = 0.15$, $\beta_{C_v} = 0.2$.

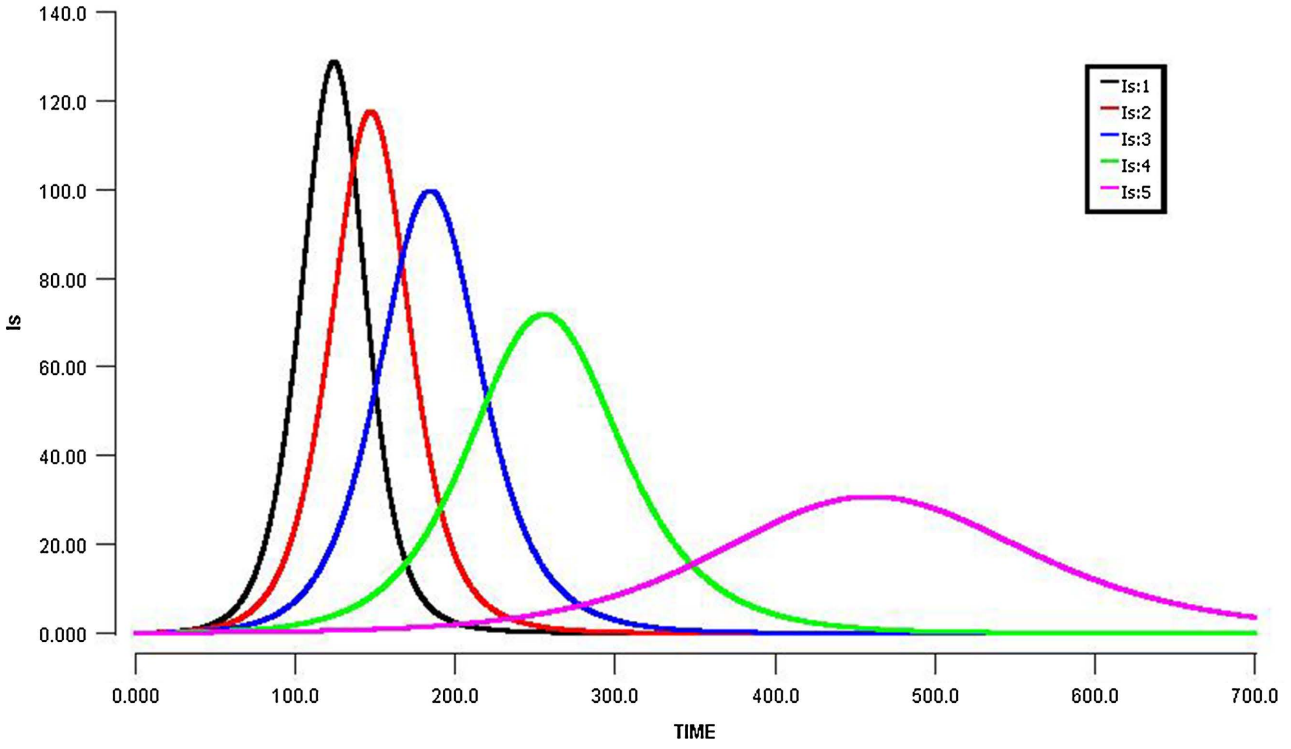

Figure 5. Evolution of number cases of COVID-19 for $\alpha = 0$, $m_b = 0$, $m_b = 0.15$, $m_b = 0.30$, $m_b = 0.45$, $m_b = 0.6$ and $\beta_{I_A} = 0.1$, $\beta_{I_S} = 0.15$, $\beta_{C_v} = 0.2$.

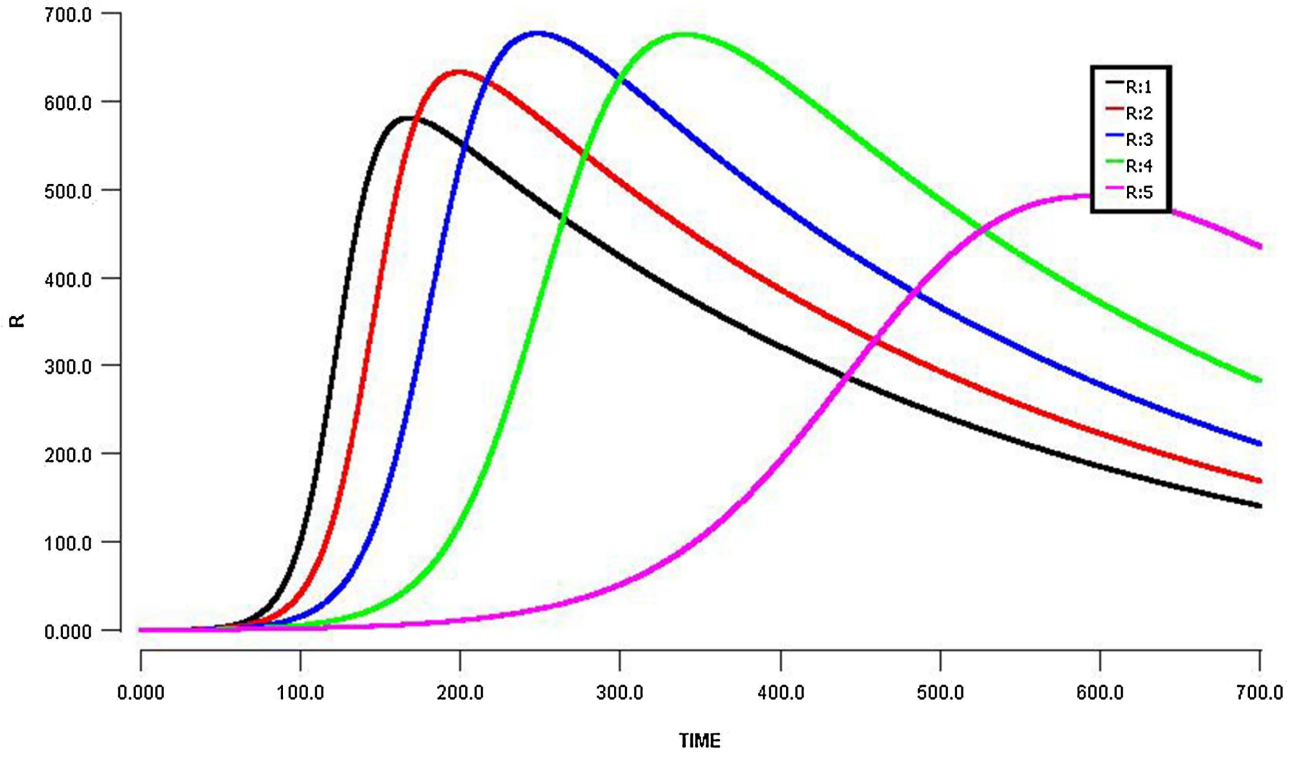

Figure 6. Evolution of number cases of COVID-19 for $\alpha = 0$, $m_b = 0$, $m_b = 0.15$, $m_b = 0.30$, $m_b = 0.45$, $m_b = 0.6$ and $\beta_{I_A} = 0.1$, $\beta_{I_S} = 0.15$, $\beta_{C_v} = 0.2$.

changes in a number of new cases among those who have been cured. The simulation shows that the more prevention measures are observed, the higher the proportion of cured cases.

Figure 7 illustrates the impact of compliance with prevention measures on virus concentration in the environment. This simulation shows that: when the population respects the prevention measures, the virus concentration in the environment decreases.

Partial conclusion: Compliance with prevention measures in the various compartments studied had a positive impact on the evolution of the disease.

3.2. Vaccination's Effect on Coronavirus Disease

In this part, vaccination is taken into account in the control of the disease. Thus, we set the value of the prevention measures at $m_b = 0.4$ and we vary the values of the parameter α. We thus obtain curves of black ($\alpha = 0$), red curve ($\alpha = 0.001$), blue curve ($\alpha = 0.002$), green curve ($\alpha = 0.003$), pink curve ($\alpha = 0.004$). With $p = 0.4$ fixed.

Figure 8 shows the evolution of cases in susceptible individuals under the influence of vaccination. This simulation shows a descending curve, demonstrating that as the proportion or rate of vaccinated individuals increases in a given population, the population of susceptible individuals decreases and becomes constant at the end of the vaccination period.

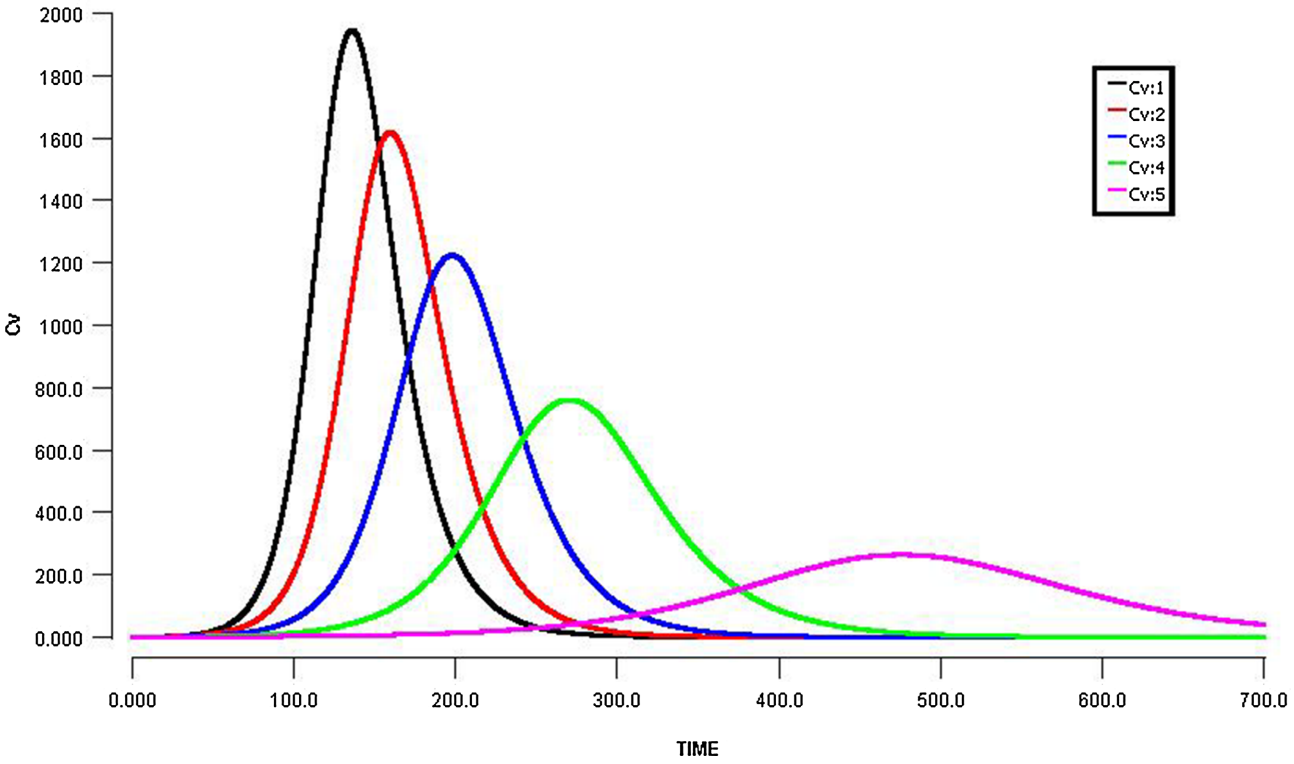

Figure 7. Evolution of number cases of COVID-19 for $\alpha = 0$, $m_b = 0$, $m_b = 0.15$, $m_b = 0.30$, $m_b = 0.45$, $m_b = 0.6$ and $\beta_{I_A} = 0.1$, $\beta_{I_S} = 0.15$, $\beta_{C_v} = 0.2$.

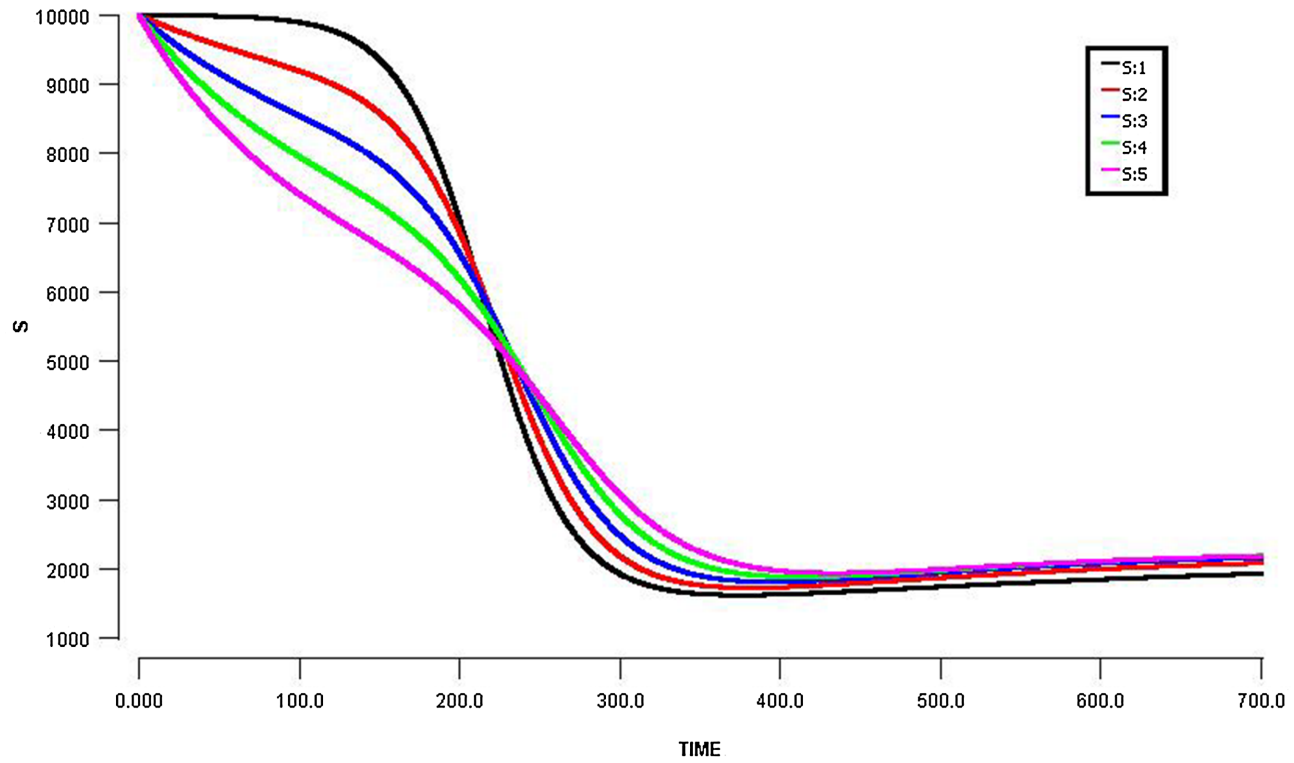

Figure 8. Evolution of number cases of COVID-19 for $m_b = 0.4$, $p = 0.4$ and $\alpha = 0$, $\alpha = 0.001$, $\alpha = 0.002$, $\alpha = 0.003$, $\alpha = 0.004$; $\beta_{I_A} = 0.1$, $\beta_{I_S} = 0.15$, $\beta_{C_v} = 0.2$.

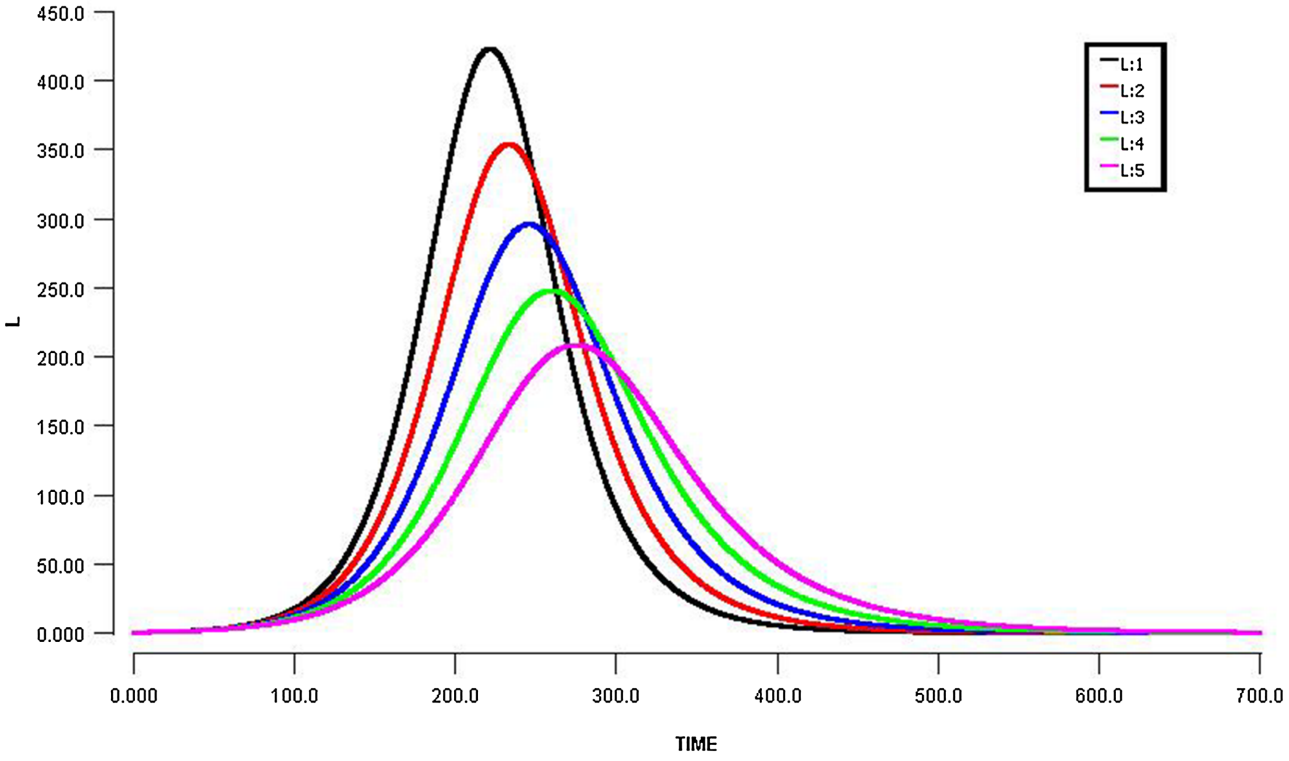

Figure 9. Evolution of number cases of COVID-19 for $m_b = 0.4$, $p = 0.4$ and $\alpha = 0$, $\alpha = 0.001$, $\alpha = 0.002$, $\alpha = 0.003$, $\alpha = 0.004$; $\beta_{I_A} = 0.1$, $\beta_{I_S} = 0.15$, $\beta_{C_v} = 0.2$.

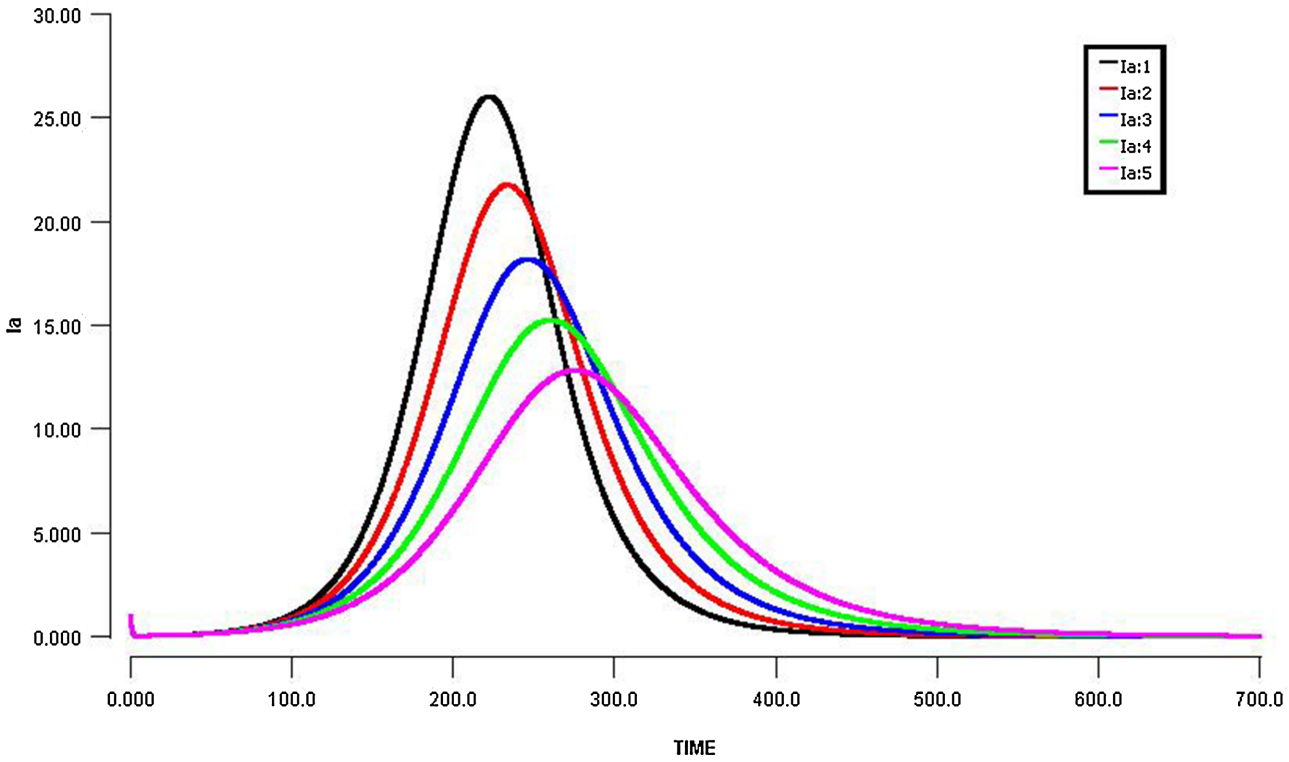

Figure 10. Evolution of number cases of COVID-19 for $m_b = 0.4$, $p = 0.4$ and $\alpha = 0$, $\alpha = 0.001$, $\alpha = 0.002$, $\alpha = 0.003$, $\alpha = 0.004$; $\beta_{I_A} = 0.1$, $\beta_{I_S} = 0.15$, $\beta_{C_v} = 0.2$.

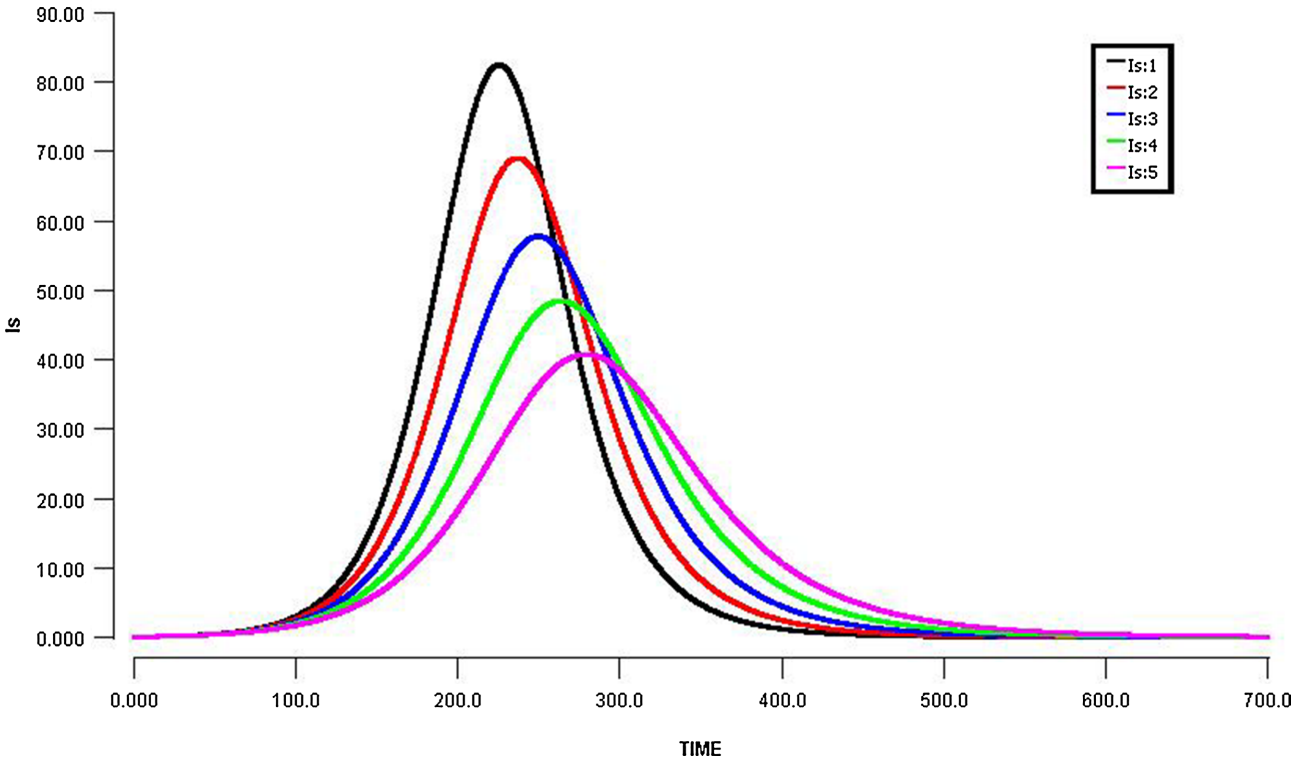

Figure 11. Evolution of number cases of COVID-19 for $m_b = 0.4$, $p = 0.4$ and $\alpha = 0$, $\alpha = 0.001$, $\alpha = 0.002$, $\alpha = 0.003$, $\alpha = 0.004$; $\beta_{I_A} = 0.1$, $\beta_{I_S} = 0.15$, $\beta_{C_v} = 0.2$.

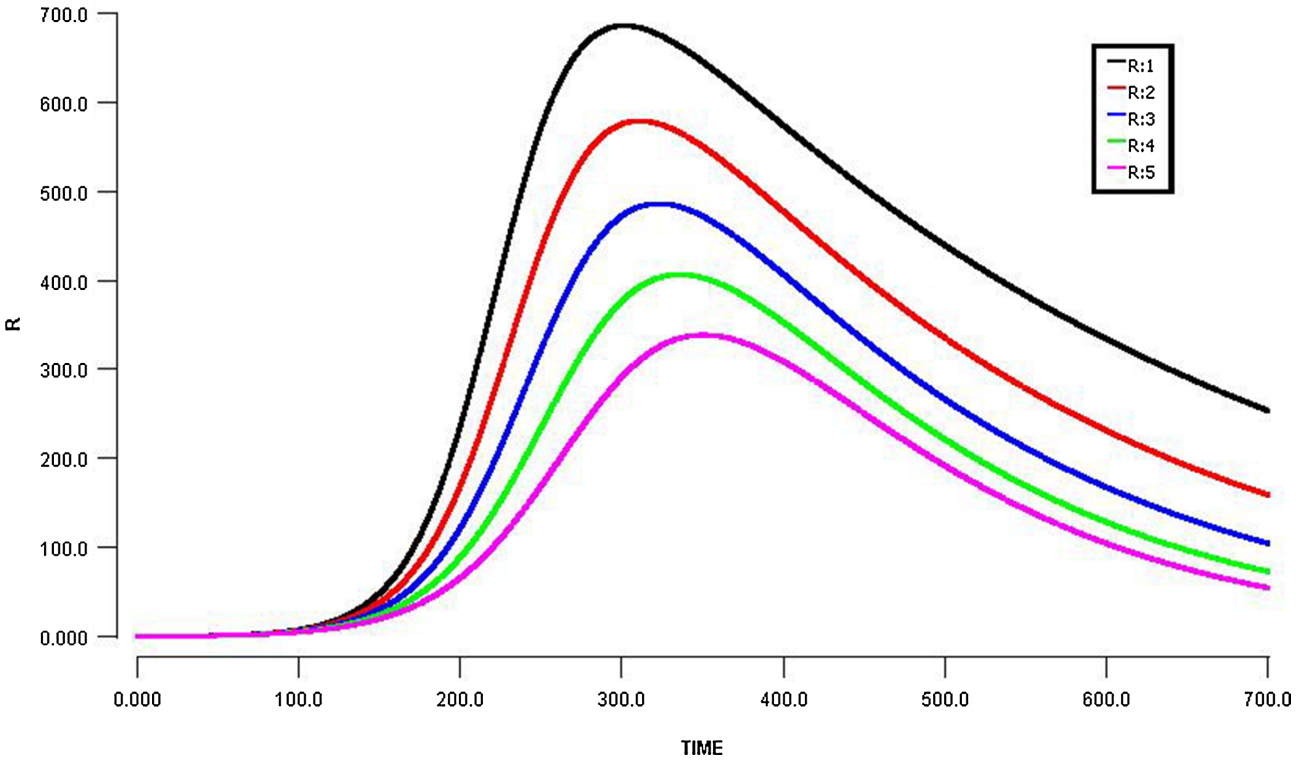

Figure 12. Evolution of number cases of COVID-19 for $m_b = 0.4$, $p = 0.4$ and $\alpha = 0$, $\alpha = 0.001$, $\alpha = 0.002$, $\alpha = 0.003$, $\alpha = 0.004$; $\beta_{I_A} = 0.1$, $\beta_{I_S} = 0.15$, $\beta_{C_v} = 0.2$.

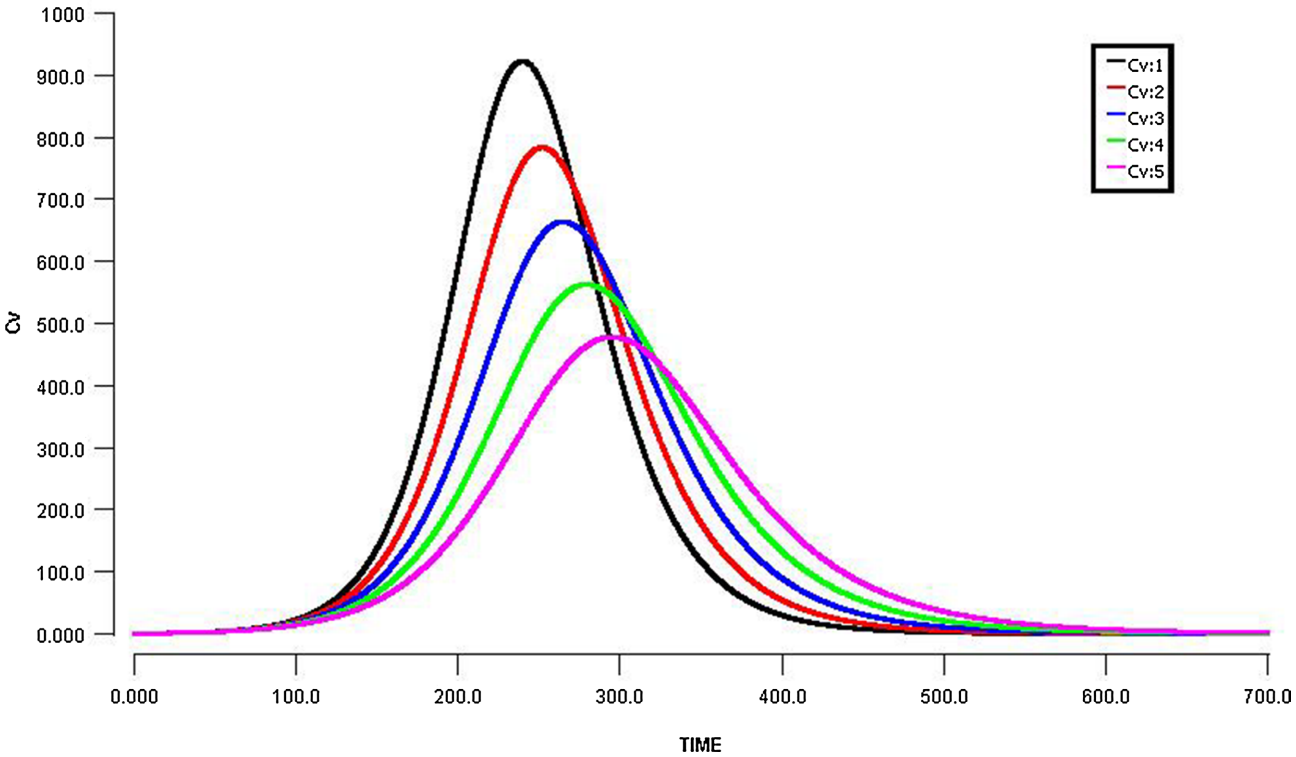

Figure 13. Evolution of number cases of COVID-19 for $m_b = 0.4$, $p = 0.4$ and $\alpha = 0$, $\alpha = 0.001$, $\alpha = 0.002$, $\alpha = 0.003$, $\alpha = 0.004$; $\beta_{I_A} = 0.1$, $\beta_{I_S} = 0.15$, $\beta_{C_v} = 0.2$.

Figures 9-11 illustrate the influence of vaccination on the evolution of the number of new symptomatic and asymptomatic cases. The peak of each curve is reached around 300 days after the onset of infection. The number of new cases decreases as the proportion of people vaccinated increases. Hence the positive impact of vaccination

Figure 12 and Figure 13 illustrate the same phenomena as the previous figures.

Partial conclusion: The curves obtained from these simulations show that the proportion of individuals complying with prevention measures reduces in number the occurrence of new cases, as well as cases obtained when the peak is reached. The proportion of vaccinated individuals reduces the number of new cases. Finally, we observe that optimal combination can be achieved to eradicate the disease. These results prove that our proposed mathematical model is valid.

4. Conclusion

COVID-19 has been spreading worldwide for over five years, and has yet to be rigorously managed. Multiple, much more infectious variants of the SARS-CoV-2 virus have been identified, representing a major scientific challenge for the management of the SARS epidemic. The prevention and control of coronavirus diseases remains a task not to be neglected. Several vaccines have been invented and implemented, but relying solely on vaccination to manage the spread of this in-

fectious disease will only lead to a partial resolution. We show that, when the basic reproductive number $\mathcal{R}_0 < 1$, there exists a locally asymptotically stable endemic equilibrium in addition to a locally asymptotically stable disease-free equilibrium, proving that even if the basic reproductive number is less than 1, the disease cannot be completely eliminated. To achieve optimal and rapid control of this pandemic, in addition to the sanitary pass or vaccination, it is essential to rigorously implement certain so-called "prevention" measures. In this article, we have formulated a seven-compartment (class) ordinary differential equation model to understand the spread dynamics of this infectious disease. Emphasis was placed on compliance with prevention measures and the effect of vaccination on a defined population. Numerical simulations were carried out to give a clearer illustration of its temporal evolution. In particular, we examined the impact of prevention measures and vaccination on slowing the spread of coronavirus infection. The model is fully analyzed and strategies to control the spread of the disease are proposed. Using the method developed by (van den Driessche and Watmough, 2002), we obtained the basic reproduction number R0 of the model. It has been shown that the rigorous application of prevention and vaccination measures can have a positive impact on the management of this epidemic. We suggest that scrupulous compliance with prevention measures combined with vaccination are rigorous strategies for controlling coronavirus infection. Our paper aims to provide decision-makers with a clearer reading and vision of the dynamics of the spread of the infectious disease known as coronavirus.

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Ferreira, T.B., Ferreira, A.L., Carrondo, M.J.T. and Alves, P.M. (2005) Two Different Serum-Free Media and Osmolality Effect upon Human 293 Cell Growth and Adenovirus Production. *Biotechnology Letters*, **27**, 1809-1813. https://doi.org/10.1007/s10529-005-3558-z

[2] Ruan, S., Wang, W. and A. Levin, S. (2006) The Effect of Global Travel on the Spread of Sars. *Mathematical Biosciences and Engineering*, **3**, 205-218. https://doi.org/10.3934/mbe.2006.3.205

[3] Wu, J.T., Leung, K. and Leung, G.M. (2020) Nowcasting and Forecasting the Potential Domestic and International Spread of the 2019-nCOV Outbreak in Wuhan, China: A Modelling Study. *The Lancet*, **395**, 689-697. https://doi.org/10.1016/s0140-6736(20)30260-9

[4] Casanova, L.M., Jeon, S., Rutala, W.A., Weber, D.J. and Sobsey, M.D. (2010) Effects of Air Temperature and Relative Humidity on Coronavirus Survival on Surfaces. *Applied and Environmental Microbiology*, **76**, 2712-2717. https://doi.org/10.1128/aem.02291-09

[5] Chen, T., Rui, J., Wang, Q., Zhao, Z., Cui, J. and Yin, L. (2020) A Mathematical Model for Simulating the Phase-Based Transmissibility of a Novel Coronavirus. *Infectious Diseases of Poverty*, **9**, Article No. 24. https://doi.org/10.1186/s40249-020-00640-3

[6] European Centre for Disease Prevention and Control (2020) Novel Coronavirus Disease 2019 (COVID-19) Pandemic: Increased Transmission in the EU/EEA and the UK-Seventh Update. *Stockholm*, **7**, 1-31.

[7] Kampf, G., Todt, D., Pfaender, S. and Steinmann, E. (2020) Persistence of Coronaviruses on Inanimate Surfaces and Their Inactivation with Biocidal Agents. *Journal of Hospital Infection*, **104**, 246-251. https://doi.org/10.1016/j.jhin.2020.01.022

[8] Meng, L. Hua, F. and Bian, Z. (2020) Coronavirus Disease 2019 (COVID-19): Emerging and Future Challenges for Dental and Oral Medicine. *Journal of Dental Research*, **99**, 481-487. https://doi.org/10.1177/0022034520914246

[9] CDC Weekly, C. (2020) The Epidemiological Characteristics of an Outbreak of 2019 Novel Coronavirus Diseases (COVID-19)—China, 2020. *China CDC Weekly*, **2**, 113-122. https://doi.org/10.46234/ccdcw2020.032

[10] Thieme, H.R. (2003) Mathematics in Population Biology. Princeton Series in Theoretical and Computational Biology. Princeton.

[11] Coddington, E.A. and Levinson, N. (1955) Theory of Ordinary Differential Equations. McGraw-Hill Book Company.

[12] van den Driessche, P. and Watmough, J. (2002) Reproduction Numbers and Sub-Threshold Endemic Equilibria for Compartmental Models of Disease Transmission. *Mathematical Biosciences*, **180**, 29-48. https://doi.org/10.1016/s0025-5564(02)00108-6

[13] Heesterbeek, J.A.P. (2002) A Brief History of R_0 and a Recipe for Its Calculation. *Acta Biotheoretica*, **50**, 189-204. https://doi.org/10.1023/a:1016599411804

[14] Kamgang, J.C. and Sallet, G. (2008) Computation of Threshold Conditions for Epidemiological Models and Global Stability of the Disease-Free Equilibrium (DFE). *Mathematical Biosciences*, **213**, 1-12. https://doi.org/10.1016/j.mbs.2008.02.005

[15] Fischer, A., Hinrichsen, A. and Son, N.K.D. (1998) Stability Radii of Metzler Operators. *Vietnam Journal of Mathematics*, **26**, 147-163.

[16] Narendra, K.S. and Shorten, R. (2010) Hurwitz Stability of Metzler Matrices. *IEEE Transactions on Automatic Control*, **55**, 1484-1487. https://doi.org/10.1109/tac.2010.2045694

[17] Hirsch, W.M., Hanisch, H. and Gabriel, J. (1985) Differential Equation Models of Some Parasitic Infections: Methods for the Study of Asymptotic Behavior. *Communications on Pure and Applied Mathematics*, **38**, 733-753. https://doi.org/10.1002/cpa.3160380607

[18] Freedman, H.I. and Moson, P. (1990) Persistence Definitions and Their Connections. *Proceedings of the American Mathematical Society*, **109**, 1025-1033. https://doi.org/10.1090/s0002-9939-1990-1012928-6

[19] Freedman, H.I., Ruan, S. and Tang, M. (1994) Uniform Persistence and Flows near a Closed Positively Invariant Set. *Journal of Dynamics and Differential Equations*, **6**, 583-600. https://doi.org/10.1007/bf02218848

[20] Magal, P. (2008) Perturbation of a Globally Stable Steady State and Uniform Persistence. *Journal of Dynamics and Differential Equations*, **21**, 1-20. https://doi.org/10.1007/s10884-008-9127-0

[21] Perasso, A. and Razafison, U. (2014) Asymptotic Behavior and Numerical Simulations for an Infection Load-Structured Epidemiological Model: Application to the Transmission of Prion Pathologies. *SIAM Journal on Applied Mathematics*, **74**, 1571-1597. https://doi.org/10.1137/130946058

[22] Thieme, H.R. (2000) Uniform Persistence and Permanence for Non-Autonomous

Semiflows in Population Biology. *Mathematical Biosciences*, 1**66**, 173-201. https://doi.org/10.1016/s0025-5564(00)00018-3

[23] Thieme, H.R. (2011) Global Stability of the Endemic Equilibrium in Infinite Dimension: Lyapunov Functions and Positive Operators. *Journal of Differential Equations*, **250**, 3772-3801. https://doi.org/10.1016/j.jde.2011.01.007

[24] European Centre for Disease Prevention and Control (2020) Novel Coronavirus Disease 2019 (COVID-19) Pandemic: Increased Transmission in the EU/EEA and the UK-Sixth Update. *Stockholm*, **6**, 1-28.

[25] Yang, C. and Wang, J. (2020) A Mathematical Model for the Novel Coronavirus Epidemic in Wuhan, China. *Chinese Center for Disease Control and Prevention*, **17**, 2708-2724.