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ON THE IMPACT OF VACCINATION CAMPAIGNS AND SOCIAL DISTANCING IN THE CONTROL OF THE NOVEL CORONAVIRUS OUTBREAKS

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Abstract: The continuous spread of "SARS-COV-2" around the globe, push the governments to start vaccination campaigns by approving several vaccines under the emergency procedure. However, the shortage of vaccines supplies slowed down the vaccination campaigns in most countries. In this paper we performed a 'SELIAAvHRD' model containing nine stages (Susceptible, Exposed, Latent, Symptomatic Infected, Asymptomatic Infected, Asymptomatic Vaccinated, Hospitalized, Recovered and Dead) for analyzing and modelling the effect of social distancing measures and vaccination campaigns. The simulation using the proposed model in different scenarios, shows the importance of respecting the social distancing measure to cover the shortage of vaccine supplies and avert deaths from "SARS-COV-2" across the world.

Keywords: asymptomatic; basic reproduction number; coronavirus; vaccine.

2010 AMS Subject Classification: 92C60, 93A30.

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1. INTRODUCTION

In March 2020 the world health organization (WHO) declares a global pandemic due to the global outbreak of the coronavirus, named "SARS-COV-2" [1, 2, 3]. In order to develop effective response strategies to anticipate the development of the pandemic, throughout the year 2020 researchers from different countries have developed several mathematical models. In [4] Peirlinck et al. proposed a SEIIR model, which breaks down infected individuals into symptomatic and asymptomatic groups with the same rate of transmission. Pribylova et al. in their work [5], they describe a SEIAR model to analyze the " SARS-COV-2 " and they showed the importance of barrier gestures and the effectiveness of the curfew applied by most governments around the world. However, as countries face the harsh economic realities of lockdown, many are choosing to reopen their economies. At the end of May 31, 2020, most countries began to ease lockdown measures even though the coronavirus is far from over. As a result, the number of new cases detected began to increase, putting the health care systems under great pressure. Developing and producing an effective vaccine in just 12 months has become a global challenge and priority.

The World Health Organization (WHO) approved a Covid-19 vaccine for the first time on December 31, 2020 under the emergency procedure [6]. Governments of different countries around the world have started to present their strategies for immunizing the majority of the population in order to achieve herd immunity.

With a view to assisting public decision-making, this work anticipates vaccination scenarios based on the possible evolution of virus circulation and on the characteristics of the vaccines developed which will constitute in addition to essential barrier measures and possible treatments, the best tool for preventing and combating the pandemic.

In our previous work, we proposed a SELIAHRD model (Susceptible, Exposed, Latent, Symptomatic, Asymptomatic, Hospitalized, Recovered and Death) with a simulation of the barrier gesture implementation scenarios showing that if the populations apply the health instructions and respect social distinction the health care system would not be saturated [7]. Based on this work we improve the proposed model taking into account the vaccinated persons. The remainder of the paper is organized as follows. Section 2. gives a brief description of the model of interest. Then, in section 3. we discuss the well-posedness and equilibria of the proposed model. Section 4. gives

the proof of the global stability of the disease-free equilibrium. The results and discussion are presented in the last section.

2. PROPOSED EPIDEMIC MODEL

Initial vaccine trials focused on vaccine safety. These trials were designed to gather data quickly on how effectively the vaccines prevented people from progressing to hospitalization and death with Covid-19. Now, as new, SARS-CoV-2 variants from the United Kingdom, South Africa and Brazil spread at the World level, understanding transmission as it relates to vaccine rollout efforts is essential. However, until now there is no evidence that any of the current Covid-19 vaccines can completely stop people from being infected (viral transmission from vaccinated individuals to unvaccinated individuals).

To investigate the effect of vaccinated people, we propose the SELIAA_vHRD model including nine compartments shown in figure 1 below, where E, L, I, A, H, R and D have same meaning as in [7] which are respectively, the exposed, latent, symptomatic, asymptomatic, hospitalized, recovered and death compartments. On the other hand, A_v denotes the asymptomatic vaccinated people and S the susceptible people (the total population is considered as susceptible people “vaccinated and non-vaccinated”).

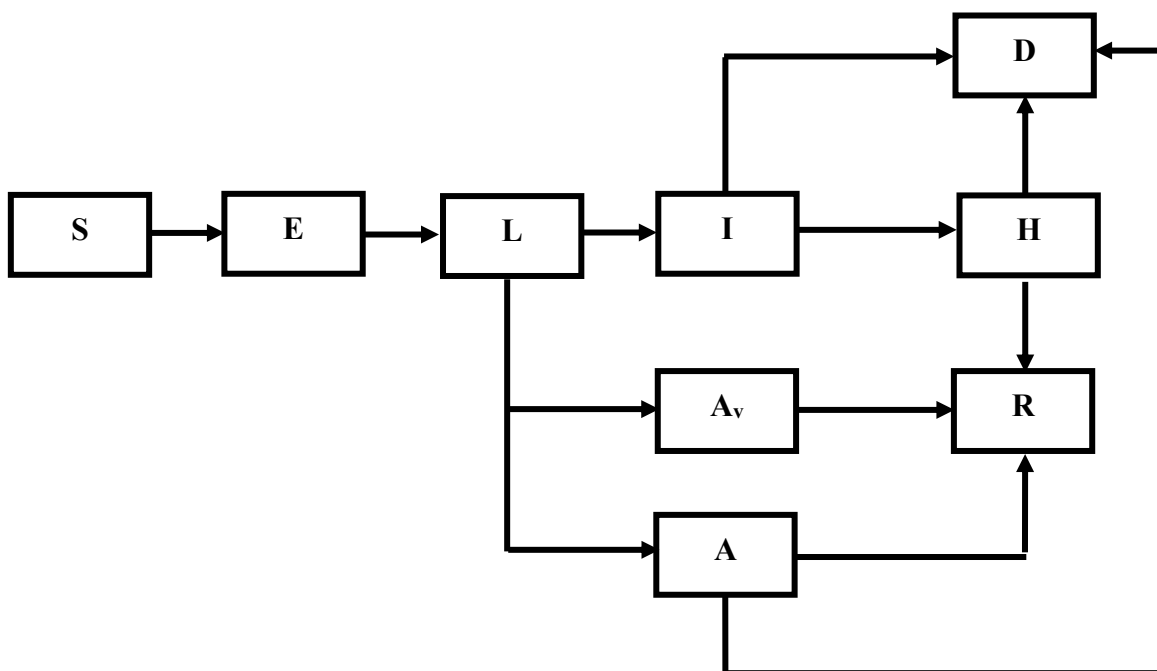


Figure 1: SELIAA_vHRD model.

Based on our previous model [7] we proposed the following model (system 1):

$$\begin{aligned}
\frac{dS(t)}{dt} &= \frac{-\beta S(t)[L(t) + A(t) + I(t)] - \beta_v S(t)A_v(t)}{N}, \\
\frac{dE(t)}{dt} &= \frac{\beta S(t)[L(t) + A(t) + I(t)] + \beta_v S(t)A_v(t)}{N} - \mu E(t), \\
\frac{dL(t)}{dt} &= \mu E(t) - [\mu_1 \alpha_1 + \mu_2 \alpha_2 + (1 - \alpha_2)]L(t), \\
\frac{dI(t)}{dt} &= \mu_1 \alpha_1 L(t) - [\mu_3 \sigma_1 + (1 - \sigma_1)]I(t), \\
\frac{dA(t)}{dt} &= \mu_2 \alpha_2 L(t) - [\mu_4 \sigma_2 + (1 - \sigma_2)]A(t), \\
\frac{dA_v(t)}{dt} &= (1 - \alpha_2)L(t) - \sigma_3 A_v(t), \\
\frac{dH(t)}{dt} &= \mu_3 \sigma_1 I(t) - [\mu_5 \gamma + (1 - \gamma)]H(t), \\
\frac{dR(t)}{dt} &= \mu_4 \sigma_2 A(t) + \mu_5 \gamma H(t) + \sigma_3 A_v(t), \\
\frac{dD(t)}{dt} &= (1 - \sigma_1)I(t) + (1 - \sigma_2)A(t) + (1 - \gamma)H(t) - \delta D(t).
\end{aligned} \tag{1}$$

Table 1 below show the symbol definition for the proposed model.

Symbol	Definition
S	Number of susceptible person
E	Number of exposed person
L	Number of latent person
I	Number of symptomatic person
A	Number of asymptomatic person
A_v	Number of vaccinated asymptomatic person
H	Number of hospitalized person
R	Number of recovered person
D	Number of death person
N	Total number of person
β	Transmission rate
β_v	Transmission rate of vaccinated people
M	Exposed rate

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α_1	Symptomatic infection rate
α_2	Asymptomatic infection rate
σ_1	Symptomatic hospitalized rate
σ_2	Asymptomatic recovery rate
σ_3	Vaccinated Asymptomatic recovery rate
μ_1	The velocity of latent person become symptomatic person
μ_2	The velocity of latent person become asymptomatic person
μ_3	The velocity of symptomatic person become hospitalized person
μ_4	The velocity of asymptomatic person become recovered person
μ_5	The velocity of hospitalized person become recovered person
γ	The recovery rate
δ	The normal death rate

Table 1: *Symbol definition.***3. POSITIVITY, BOUNDEDNESS AND EQUILIBRIA**

In this paragraph, we ensure that the model has a mathematical and biological meaning, by proofing the existence, the positivity and the boundedness of solutions of the proposed system (1), that can be rewritten as follows:

$$dZ = P(Z(t)),$$

where

$$Z(t) = \begin{pmatrix} S(t) \\ E(t) \\ L(t) \\ I(t) \\ A(t) \\ A_v(t) \\ H(t) \\ R(t) \\ D(t) \end{pmatrix},$$

and P is a C1 function mapping \mathbb{R}^9 into it self defined by:

$$P(Z) = \begin{pmatrix} p_1(S, E, L, I, A, A_v, H, R, D) \\ p_2(S, E, L, I, A, A_v, H, R, D) \\ p_3(S, E, L, I, A, A_v, H, R, D) \\ p_4(S, E, L, I, A, A_v, H, R, D) \\ p_5(S, E, L, I, A, A_v, H, R, D) \\ p_6(S, E, L, I, A, A_v, H, R, D) \\ p_7(S, E, L, I, A, A_v, H, R, D) \\ p_8(S, E, L, I, A, A_v, H, R, D) \\ p_9(S, E, L, I, A, A_v, H, R, D) \end{pmatrix} = \begin{pmatrix} \frac{-\beta S[L + A + I] - \beta_v S A_v}{N} \\ \frac{\beta S[L + A + I] + \beta_v S A}{N} - \mu E \\ \mu E - [\mu_1 \alpha_1 + \mu_2 \alpha_2 + (1 - \alpha_2)]L \\ \mu_1 \alpha_1 L - [\mu_3 \sigma_1 + (1 - \sigma_1)]I \\ \mu_2 \alpha_2 L - [\mu_4 \sigma_2 + (1 - \sigma_2)]A \\ (1 - \alpha_2)L - \sigma_3 A_v \\ \mu_3 \sigma_1 I - [\mu_5 \gamma + (1 - \gamma)]H \\ \mu_4 \sigma_2 A + \mu_5 \gamma H + \sigma_3 A_v \\ (1 - \sigma_1)I + (1 - \sigma_2)A + (1 - \gamma)H - \delta D \end{pmatrix}.$$

According to the fundamental theory of functional differential equations [8], the system (1) has a unique solution $(S(t), E(t), L(t), I(t), A(t), A_v(t), H(t), R(t), D(t))$ with respect to the initial data Z_0 such that :

$$S(t) \geq 0, E(t) \geq 0, L(t) \geq 0, I(t) \geq 0, A(t) \geq 0, A_v(t) \geq 0, H(t) \geq 0, R(t) \geq 0, D(t) \geq 0.$$

If we put $S + E + L + I + A + A_v + R + H + D \leq N$, then we have the following theorems:

Theorem 1. (Invariant Region) *The following biological feasible region of the system (1) $B = \{(S, E, L, I, A, A_v, H, R, D) \in \mathbb{R}_+^9; S + E + L + I + A + A_v + R + H + D \leq N\}$ is positively invariant and attracting.*

Theorem 2. *Let $t_0 > 0$ and the initial conditions satisfied $S(t_0) > 0, E(t_0) > 0, L(t_0) > 0, I(t_0) > 0, A(t_0) > 0, A_v(t_0) \geq 0, H(t_0) > 0, R(t_0) > 0, D(t_0) > 0$ then the solution $S(t), E(t), L(t), I(t), A(t), A_v(t), H(t), R(t), D(t)$ of the system (1) are positive for all $t \geq 0$.*

Proof:

From the first equation of system (1) we have:

$$S(t) = s(0) \left[e^{\frac{-\beta}{N} \int_0^t [L(u) + A(u) + I(u)] du} + e^{\frac{-\beta_v}{N} \int_0^t A_v(u) du} \right].$$

We know that $S(t)$ is non-negative for all $t \geq 0$. From the others equations of system (1) we set:

$$E(t) = E(0)e^{-\mu t} + \int_0^t e^{(u-t)\mu} \frac{\beta}{N} S(u)[L(u) + A(u) + I(u)] du + \frac{\beta_v}{N} \int_0^t e^{(u-t)\mu} S(u) A_v(u) du,$$

$$E(t) = [E(0) + \frac{\beta}{N} \int_0^t e^{u\mu} S(u)[L(u) + A(u) + I(u)] du + \frac{\beta_v}{N} \int_0^t e^{u\mu} S(u) A_v(u) du] e^{-\mu t},$$

$$L(t) = [L(0) + \mu \int_0^t e^{u(\mu_1\alpha_1 + \mu_2\alpha_2 + (1-\alpha_2))} E(u) du] e^{-(\mu_1\alpha_1 + \mu_2\alpha_2 + (1-\alpha_2))t},$$

$$I(t) = [I(0) + \mu_1\alpha_1 \int_0^t e^{u(\mu_3\sigma_1 + (1-\sigma_1))} L(u) du] e^{-(\mu_3\sigma_1 + (1-\sigma_1))t},$$

$$A(t) = [A(0) + \mu_2\alpha_2 \int_0^t e^{u(\mu_4\sigma_2 + (1-\sigma_2))} L(u) du] e^{-(\mu_4\sigma_2 + (1-\sigma_2))t},$$

$$A_v(t) = \left[A_v(0) + (1 - \alpha_2) \int_0^t e^{u\sigma_3} L(u) du \right] e^{-t\sigma_3},$$

$$H(t) = [H(0) + \mu_3\sigma_1 \int_0^t e^{u(\mu_5\gamma_1 + (1-\gamma))} I(u) du] e^{-(\mu_5\gamma_1 + (1-\gamma))t},$$

$$D(t) = [D(0) + \int_0^t e^{u\delta} [(1 - \sigma_1)I(u) + (1 - \sigma_2)A(u) + (1 - \gamma)H(u)] du] e^{-\delta t}.$$

Therefore $E(t)$, $L(t)$, $I(t)$, $A(t)$, $A_v(t)$, $H(t)$ and $D(t)$ are all non-negative for all $t \geq 0$.

From the seventh equation of the system (1), clearly we can deduce the positivity of $R(t)$ for all $t \geq 0$. Hence, the model is mathematically and epidemiologically well posed.

4. STABILITY ANALYSIS

In this paragraph, we prove the stability of the diseases-free equilibrium U_0 of the model (1).

The equilibrium of the model (1) is obtained by setting $\frac{dS(t)}{dt} = \frac{dE(t)}{dt} = \frac{dL(t)}{dt} = \frac{dI(t)}{dt} = \frac{dA(t)}{dt} =$

$$\frac{dA_v(t)}{dt} = \frac{dH(t)}{dt} = \frac{dR(t)}{dt} = \frac{dD(t)}{dt} = 0.$$

The system disease free equilibrium (DFE) is given as, $U_0 = (N, 0, 0, 0, 0, 0, 0, 0, 0)$. With the existence of different types of infected individuals, the use of the next generation operator is mandatory to define the effective basic reproduction number (R_0) [9], in order to investigate the stability of the disease free equilibrium.

Let Y be vector of infected classes, such as infectious, exposed, hospitalized, etc and X be vector of uninfected classes, such as susceptible, recovered, etc.

$$\frac{dX}{dt} = W(X, Y),$$

$$\frac{dY}{dt} = f(X, Y) = F(X, Y) - V(X, Y).$$

Let $U_0 = (X^*, 0) \in \mathbb{R}_+^9$ denote the disease-free equilibrium, that is $f(X^*, 0) = W(X^*, 0) = 0$, where F_i is the rate of new infection in compartment 'i' and $V(X, Y)$ is the vector of all others rates (Not new infection). For each compartment, in flow in V is negative and out flow in V is positive.

The spectral radius (ρ) of the next generation matrix $\mathcal{F}\mathcal{V}^{-1}$, define the basic reproductive number $R_0 = \rho(\mathcal{F}\mathcal{V}^{-1})$, where

$$\mathcal{F} = \left(\frac{\partial F}{\partial Y} \right)_{(X^*, 0)} = \begin{pmatrix} 0 & \beta & \beta & \beta & \beta_v & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$\mathcal{V}^{-1} = \left(\frac{\partial V}{\partial Y} \right)_{(X^*, 0)} = \begin{pmatrix} \frac{1}{\mu} & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{1}{J} & \frac{1}{J} & 0 & 0 & 0 & 0 & 0 \\ \frac{\mu_1 \alpha_1}{J d} & \frac{\mu_1 \alpha_1}{J d} & \frac{1}{d} & 0 & 0 & 0 & 0 \\ \frac{\mu_2 \alpha_2}{J e} & \frac{\mu_2 \alpha_2}{J e} & 0 & \frac{1}{e} & 0 & 0 & 0 \\ \frac{1 - \alpha_2}{J \sigma_3} & \frac{1 - \alpha_2}{J \sigma_3} & 0 & 0 & \frac{1}{\sigma_3} & 0 & 0 \\ \frac{(\mu_1 \alpha_1)(\mu_3 \sigma_1)}{J d m} & \frac{(\mu_1 \alpha_1)(\mu_3 \sigma_1)}{J d m} & \frac{\mu_3 \sigma_1}{d m} & 0 & 0 & \frac{1}{m} & 0 \\ C & C & T & \frac{1 - \sigma_2}{e \delta} & 0 & \frac{1 - \gamma}{m \delta} & \frac{1}{\delta} \end{pmatrix},$$

where

$$\begin{aligned} J &= \mu_1 \alpha_1 + \mu_2 \alpha_2 + (1 - \alpha_2), \\ d &= \mu_3 \sigma_1 + (1 - \sigma_1), \\ e &= \mu_4 \sigma_2 + (1 - \sigma_2), \\ m &= \mu_5 \gamma + (1 - \gamma), \\ C &= \frac{(\mu_1 \alpha_1)(1 - \sigma_1)}{J d \delta} + \frac{(\mu_2 \alpha_2)(1 - \sigma_2)}{J e \delta} + \frac{(1 - \gamma)(\mu_1 \alpha_1)(\mu_3 \sigma_1)}{J d m \delta}, \end{aligned}$$

$$T = \frac{(1-\gamma)(\mu_3\sigma_1)}{m d \delta} + \frac{1-\sigma_1}{d \delta}.$$

Multiplying \mathcal{F} and \mathcal{V}^{-1} together and calculating the spectral radius, we obtained the basic reproductive number:

$$R_0 = \frac{\beta}{J} + \frac{\beta\mu_1\alpha_1}{Jd} + \frac{\beta\mu_2\alpha_2}{Je} + \frac{\beta_v(1-\alpha_2)}{J\sigma_3},$$

$$R_0 = \frac{\beta}{\mu_1\alpha_1 + \mu_2\alpha_2 + (1-\alpha_2)} + \frac{\beta\mu_1\alpha_1}{(\mu_1\alpha_1 + \mu_2\alpha_2 + (1-\alpha_2))(\mu_3\sigma_1 + (1-\sigma_1))}$$

$$+ \frac{\beta\mu_2\alpha_2}{(\mu_1\alpha_1 + \mu_2\alpha_2 + (1-\alpha_2))(\mu_4\sigma_2 + (1-\sigma_2))} + \frac{\beta_v(1-\alpha_2)}{(\mu_1\alpha_1 + \mu_2\alpha_2 + (1-\alpha_2))\sigma_3}.$$

Theorem 3. *If $R_0 < 1$, then DFE (disease-free equilibrium) U_0 is locally asymptotically stable (L.A.S). If $R_0 > 1$ then DFE is unstable.*

4.1. Global stability of disease-free equilibrium

In order to obtain the global stability for the disease-free equilibrium the bellow conditions must be satisfied.

First, the system (1) must be written in the form:

$$\frac{dX}{dt} = W(X, Y),$$

$$(2)$$

$$\frac{dY}{dt} = G(X, Y), \quad G(X, 0) = 0.$$

(C₁) for $\frac{dX}{dt} = W(X, 0)$, X^* is Globally Asymptotic Stable (GAS),

(C₂) for $G(X, Y) = QY - \hat{G}(X, Y)$, $\hat{G}(X, Y) \geq 0$ for $(X, Y) \in B$.

Where $Q = \left(\frac{\partial G}{\partial Y}\right)_{U_0}$ is a Metzler matrix (M-matrix, the off diagonal elements of Q are non-negative). The satisfaction of this two conditions give us the following lemma.

Lemma 4. *The disease-free equilibrium $U_0 = (X^*, 0)$ of the system (2) is globally asymptotically stable (G.A.S) provided that $R_0 < 1$ and assumption (C₁) and (C₂) are satisfied.*

Now we announce the following theorem:

Theorem 5. *The disease-free equilibrium of system (1) is globally asymptotically stable if $R_0 < 1$.*

Proof:

Let $X = (S, R)$, and $Y = (E, L, I, A, A_v, H, D)$. We will have:

$$W(X, Y) = \left(\frac{-\beta S[L + I + A] - \beta_v S A_v}{N}, \mu_4 \sigma_2 A + \mu_5 \gamma H + \sigma_3 A_v \right),$$

at the point $(X, 0)$, $W(X, 0) = (0)$, $X^* = (N, 0)$ is globally asymptotically stable for $\frac{dX}{dt} = W(X, 0)$.

Next, we prove the second condition is satisfied, that is: $\hat{G}(X, Y) = QY - G(X, Y)$,

$$G(X, Y) = \begin{pmatrix} \frac{\beta S[L + A + I] + \beta_v S A_v}{N} - \mu E \\ \mu E - [\mu_1 \alpha_1 + \mu_2 \alpha_2 + (1 - \alpha_2)]L \\ \mu_1 \alpha_1 L - [\mu_3 \sigma_1 + (1 - \sigma_1)]I \\ \mu_2 \alpha_2 L - [\mu_4 \sigma_2 + (1 - \sigma_2)]A \\ (1 - \alpha_2)L - \sigma_3 A_v \\ \mu_3 \sigma_1 I - [\mu_5 \gamma + (1 - \gamma)]H \\ (1 - \sigma_1)I + (1 - \sigma_2)A + (1 - \gamma)H - \delta D \end{pmatrix}.$$

And this follows that $Q = \left(\frac{\partial G}{\partial Y} \right)_{U_0}$,

$Q =$

$$\begin{pmatrix} -\mu & \beta & \beta & \beta & \beta_v & 0 & 0 \\ \mu & -(\mu_1 \alpha_1 + \mu_2 \alpha_2 + (1 - \alpha_2)) & 0 & 0 & 0 & 0 & 0 \\ 0 & \mu_1 \alpha_1 & -(\mu_3 \sigma_1 + (1 - \sigma_1)) & 0 & 0 & 0 & 0 \\ 0 & \mu_2 \alpha_2 & 0 & -(\mu_4 \sigma_2 + (1 - \sigma_2)) & 0 & 0 & 0 \\ 0 & (1 - \alpha_2) & 0 & 0 & -\sigma_3 & 0 & 0 \\ 0 & 0 & \mu_3 \sigma_1 & 0 & 0 & -(\mu_5 \gamma + (1 - \gamma)) & 0 \\ 0 & 0 & 1 - \sigma_1 & 1 - \sigma_2 & 0 & 1 - \gamma & -\delta \end{pmatrix}.$$

The off-diagonal elements of Q are non-negative and $\hat{G}(X, Y) = QY - G(X, Y) =$

$$\begin{pmatrix} \beta[L + A + I] + \beta_v A_v - \mu E \\ \mu E - [\mu_1 \alpha_1 + \mu_2 \alpha_2 + (1 - \alpha_2)]L \\ \mu_1 \alpha_1 L - [\mu_3 \sigma_1 + (1 - \sigma_1)]I \\ \mu_2 \alpha_2 L - [\mu_4 \sigma_2 + (1 - \sigma_2)]A \\ (1 - \alpha_2)L - \sigma_3 A_v \\ \mu_3 \sigma_1 I - [\mu_5 \gamma + (1 - \gamma)]H \\ (1 - \sigma_1)I + (1 - \sigma_2)A + (1 - \gamma)H - \delta D \end{pmatrix} - \begin{pmatrix} \frac{\beta S[L + A + I] + \beta_v S A_v}{N} - \mu E \\ \mu E - [\mu_1 \alpha_1 + \mu_2 \alpha_2 + (1 - \alpha_2)]L \\ \mu_1 \alpha_1 L - [\mu_3 \sigma_1 + (1 - \sigma_1)]I \\ \mu_2 \alpha_2 L - [\mu_4 \sigma_2 + (1 - \sigma_2)]A \\ (1 - \alpha_2)L - \sigma_3 A_v \\ \mu_3 \sigma_1 I - [\mu_5 \gamma + (1 - \gamma)]H \\ (1 - \sigma_1)I + (1 - \sigma_2)A + (1 - \gamma)H - \delta D \end{pmatrix},$$

$$\hat{G}(X, Y) = \begin{pmatrix} (\beta[L + I + A] + \beta_v A_v) \left(1 - \frac{S}{N}\right) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

Since $S \leq N$, $1 - \frac{S}{N} \geq 0$ and thus $\hat{G}(X, Y) \geq 0$. So the two condition of the lemma 4 are satisfied, then this prove the global asymptotic stability of the disease free-equilibrium of the proposed system (1) for $R_0 < 1$.

5. NUMERICAL RESULTS AND DISCUSSION

In this part, we discuss the simulation of our SELIAAvHRD model with implication of different scenario of social distancing and different vaccine product, to show the possible impact of vaccination campaigns. The vaccines chosen for this study are Pfizer-BioNTech and AstraZeneca. Many possibilities can be studied, using the proposed model. A simplified model is used for simulating the impact of vaccination campaigns.

- a) Since there is no scientific evidence that the vaccines chosen in this study cannot completely protect people from infection. We set $\beta = \beta_v$.
- b) The mean incubation period was 5.2 days (95% confidence interval) [10]. Therefore $\mu_1 = 0.1923$.
- c) There is a mean 5-day delay from symptom onset to detection/hospitalization of a case (Symptoms may appear 2 to 14 days after exposure to the virus) [10]. So we set $\mu_3 = 0.1724$.
- d) Recovery rate $\gamma = 0.15$, is determined by the average duration of recovery from infection
- e) We simulated $\mu_1 = \mu_2$ and $\mu_4 = 0.08$.
- f) Symptomatic and asymptomatic infection rate α_1, α_2 are defined by :
 - $\alpha_1 = (\text{Symptomatic infected})/\text{Susceptible};$
 - $\alpha_2 = (\text{Asymptomatic infected})/\text{Susceptible}.$

5.1. Pfizer-BioNTech Vaccine

In this first simulation, we assume that there is no social distancing and a total population of 10 million.

The Pfizer-BioNTech (BNT162b2) vaccine is a COVID-19 vaccine administered as a 2-dose series, 3 weeks apart and may not protect everyone with an efficacy of 95% [11]. We denote P_e , which corresponds to value for the Pfizer-BioNTech vaccine efficacy.

$$P_e = \begin{cases} 0 & \text{if } 0 \leq t < 12 \text{ days,} \\ 0.524 & \text{if } 12 \leq t < 21 \text{ days,} \\ 0.905 & \text{if } 21 \leq t < 28 \text{ days,} \\ 0.948 & \text{if } 28 \text{ days} \leq t. \end{cases}$$

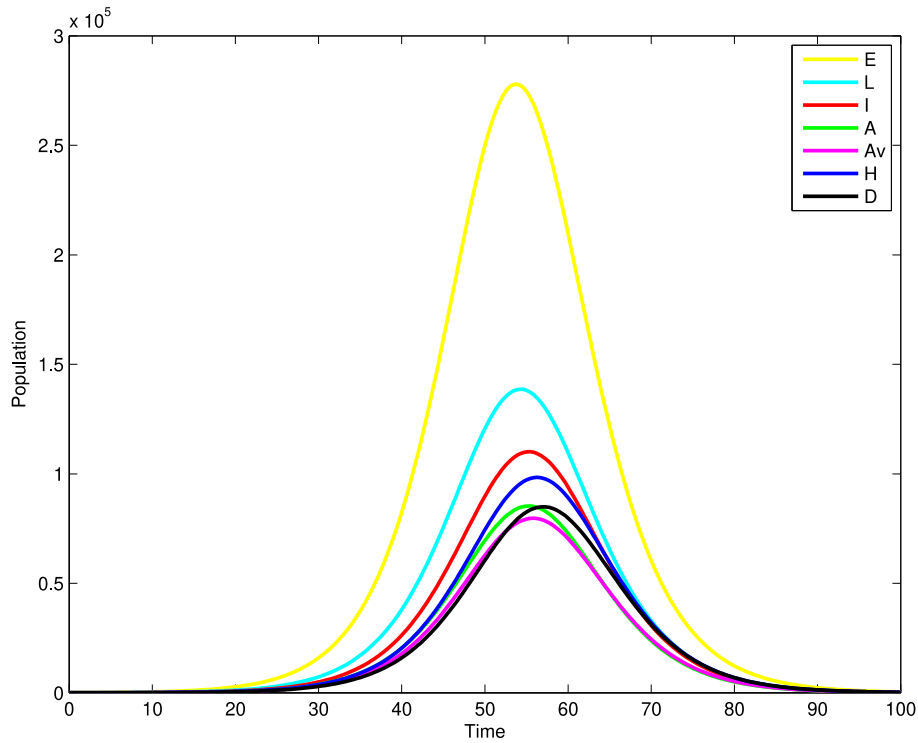


Figure 2: *Simulation for the number of cases with no social distancing.*

The basic reproductive number in the first scenario is $R_0 = 2.958$.

In the second simulation, we assume that 90% of population obey the rule of social distancing.

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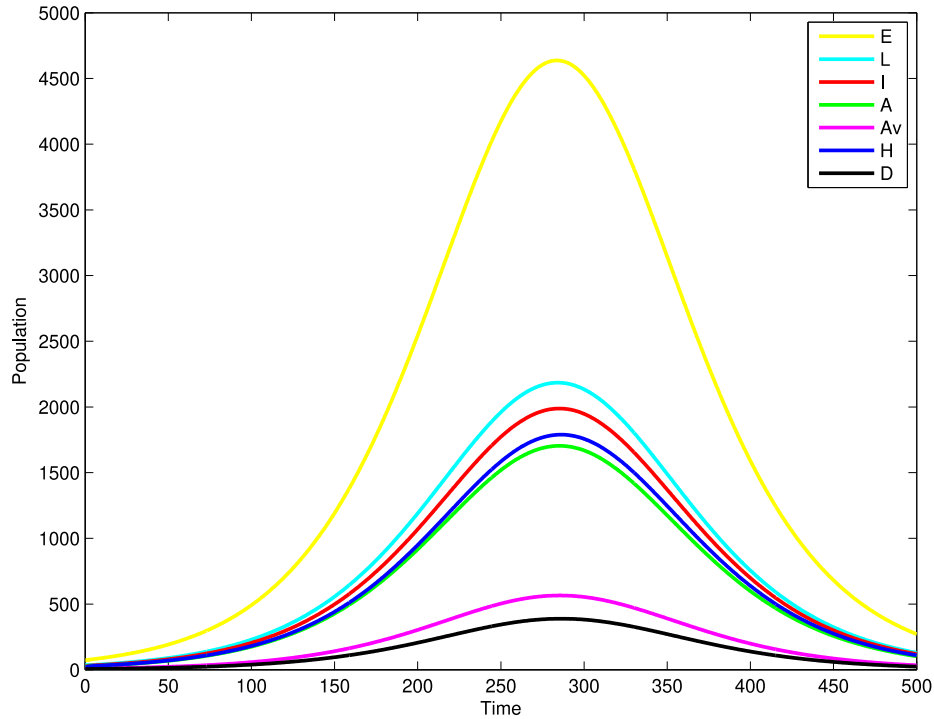


Figure 4: Simulation for the number of cases with 90% respect of social distancing.

The basic reproductive number in the second scenario is $R_0 = 0.842$.

5.2. AstraZeneca Vaccine

The Oxford AstraZeneca covid-19 vaccine ChAdOx1nCoV-19 (AZD1222) administered as a 2-dose series. A single dose of the vaccine provided 76% protection against symptomatic covid-19 in the first 90 days after vaccination [12]. We denote P_A , which corresponds to value for the AstraZeneca vaccine efficacy.

$$P_A = \begin{cases} 0 & \text{if } 0 \leq t < 12 \text{ days,} \\ 0.76 & \text{if } 12 \leq t < 90 \text{ days,} \\ 0.824 & \text{if } 90 \leq t \text{ days,} \end{cases}$$

We have simulated two different scenarios involving the vaccination campaign, with consideration of barrier gestures and social distancing measures.

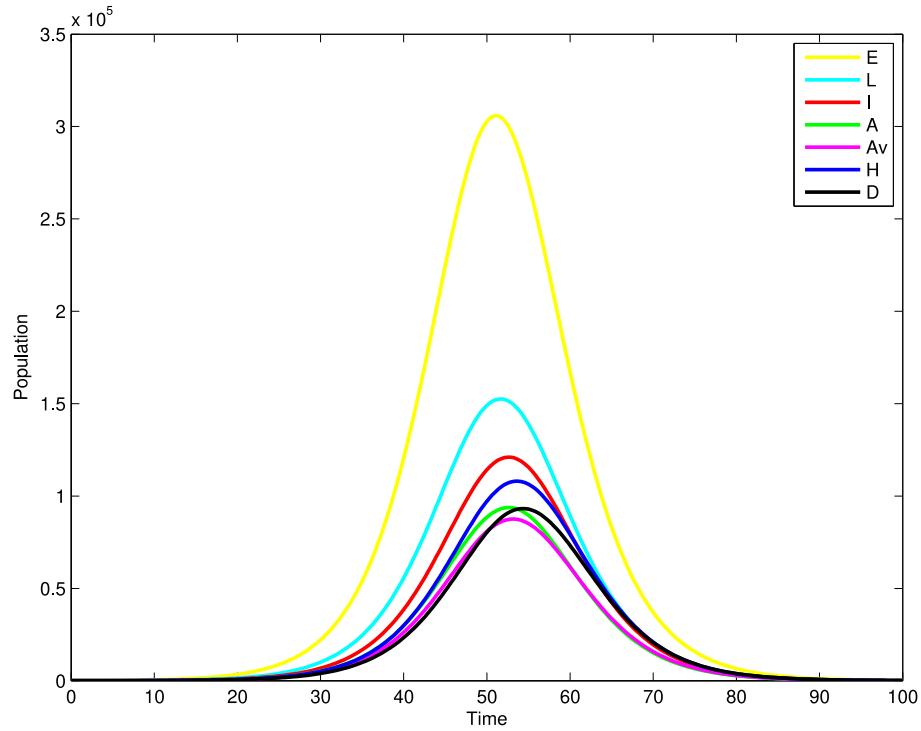


Figure 5: *Simulation for the number of cases with no social distancing.*

The basic reproductive number in the first scenario is $R_0 = 2.962$ and for the second scenario $R_0 = 0.875$.

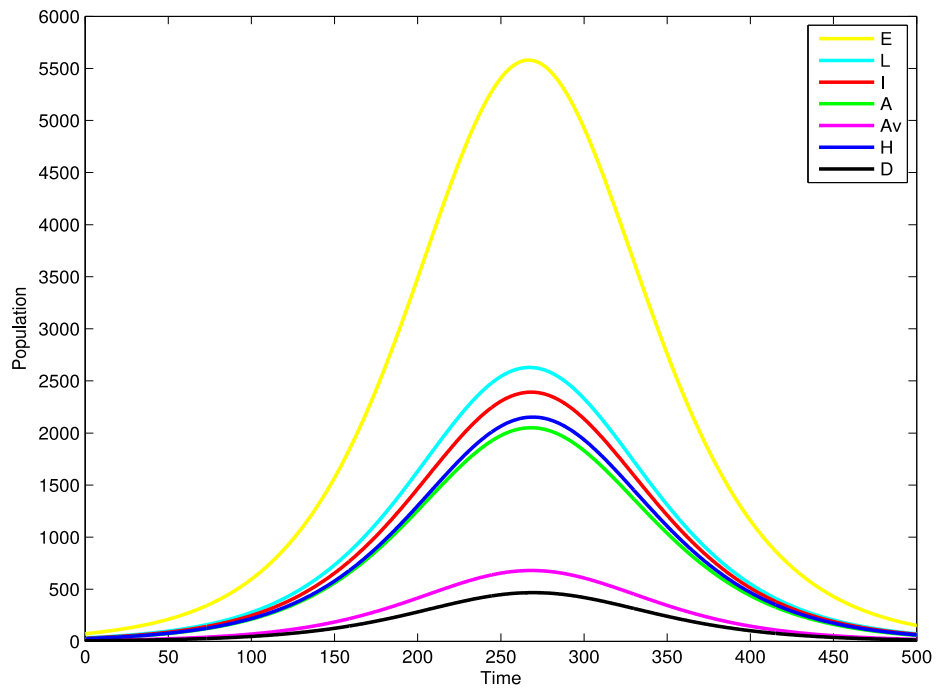


Figure 6: *Simulation for the number of cases with 90% respect of social distancing.*

As can be seen in figures 3 and 5, the number of death and hospitalized people still alerting even with the existence of effective vaccines. In the second application of the model we have introduced social distancing measures applied by some governments to slow down the Covid-19 spread and to cover the shortage of vaccine supplies. In figures 4 and 6 we show the results of the simulation, if we assume that the people respect the social distancing measures. We can see that the number of new deaths drop significantly.

From those results, and with the current rate of vaccination in most countries, we can say that strengthening of all measures of barrier gestures, social distancing and increasing the number of vaccinated person is mandatory.

6. CONCLUSIONS

In this paper we have proposed a mathematical model containing nine different stages: SELIAA_vHRD (Susceptible, Exposed, Latent, Symptomatic, Asymptomatic, Asymptomatic (vaccinated), Hospitalized, Recovered and Death). Using this proposed model, we were able to simulate different scenarios including the vaccination campaign and social distancing measures. We obtained a basic reproduction number $R_0 > 1$ in the absence of social distancing measures even with the existence of vaccinated people. On the other hand, if we maintained the control measures we obtained $R_0 < 1$, with the descent of the number of new cases and deaths. The limitation of vaccine production capacity slowed down the progress of vaccination campaigns all over the world. Since the targeted collective immunity will not be reached in the expected time planned by most countries, a return to normal life and the lifting of all restrictions will take time.

DATA AVAILABILITY

The data used to support the findings of this study are available from the corresponding author upon request.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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