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QUANTIFYING THE IMPACT OF PRECAUTIONARY MEASURE: A DELAY DIFFERENTIAL EQUATION OF COVID-19 SPREAD

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Abstract

This research employs delay differential equations (DDEs) to investigate the impact of precautionary measures on the dynamics of COVID-19. A compartmental model $SELL_AQR$ is developed, considering a time delay parameter τ to account for delays caused by preventative measures. Stability analysis reveals the Disease-Free Equilibrium (DFE) stability for $R_0 < 1$ and Endemic Equilibrium (EE) stability for $R_0 > 1$ with $\tau > 0$. Sensitivity analysis highlights the significance of parameters β and Λ in controlling disease transmission. Numerical simulations illustrate the effects of increasing τ, showing initial decreases in susceptible individuals and increases in exposed and asymptomatic infected individuals. Oscillatory behaviour is observed during the initial phase. Understanding τ informs the optimal timing of interventions and response strategies for controlling COVID-19.

1. Introduction

The unprecedented global health crisis posed by the COVID-19 pandemic demands swift and effective strategies to mitigate its impact. Mathematical modelling has emerged as a powerful tool for understanding and predicting the dynamics of infectious diseases, offering valuable insights into

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transmission modes, vaccination effects, and the efficacy of intervention strategies. In India, a country with a sizable population and unique demographic characteristics, the use of mathematical modelling is crucial for guiding evidence-based decision-making to address COVID-19 effectively.

The inception of mathematical modelling in epidemiology dates back to Daniel Bernoulli's work on the effect of variolation against smallpox, which highlighted the potential to increase life expectancy [1]. Since then, pioneering work by Kermack and McKendrick laid the foundation for applying mathematical models to comprehend infectious disease dynamics [2]. In the context of COVID-19, numerous mathematical models have been developed to study transmission dynamics in India. Studies have extended classical models like SI [3], SIS [4], and SIR [5], introducing additional compartments to account for asymptomatic cases, isolated individuals, quarantine, protection, deaths, lockdowns, hospitalizations, and more [6-12]. The COVID-19 epidemic has damaged beyond repair society at large, prompting the development of a number of preventative measures to slow its rapid spread. While lockdowns, social isolation, and other treatments have all been successful, they also slow the coronavirus's ability to spread. For effective pandemic response measures to be developed, it is essential to comprehend the effects of these delays. In this research, we explore the field of mathematical modelling and use Delay Differential Equations (DDEs) to investigate the impact of delays brought on by preventative measures on the dynamics of COVID-19. Although several studies have used delay settings, the impact of this parameter on COVID-19 has not been extensively studied [13-17].

Delay Differential Equations (DDEs) are a powerful mathematical tool that has gained significant importance in various fields of science and engineering. Unlike ordinary differential equations (ODEs), which describe the rate of change of a system with respect to the current time, DDEs account for the influence of past states on the current state. This consideration of delays is particularly pertinent in epidemiology, where the time lag between exposure, infection, and transmission plays a crucial role in disease dynamics [18-21]. DDEs have been extensively used to model infectious diseases, providing valuable insights into the impact of time delays on disease spread and control strategies.

A Delay Differential Equation (DDE) is a type of differential equation where the current time derivatives of certain unknown functions depend on the values of these functions at previous points in time. In other words, the evolution of the system at the present moment is influenced not only by its current state but also by its past states.

The general form of a DDE can be represented as follows:

$$\frac{dx(t)}{dt} = f(t, x(t), x(t - \tau))$$

where

$$x(t - \tau) = x(\tau) : \tau \leq t$$

gives the trajectory of the solution in the past. Here, the function f is a functional operator from $\mathbb{R}X\mathbb{R}^n XC^1$ to $\mathbb{R}^n$ and $x(t) \in \mathbb{R}^n$ [22].

As a consequence of enforcing diverse precautionary measures, such as social distancing, self-isolation, practicing personal hygiene, mask-wearing, and widespread media awareness, a delay is anticipated in the time it takes for each susceptible individual to be exposed and potentially infected. In our study, we incorporate the notion of time delay by introducing the parameter τ, which represents the extent of this delay in the susceptibility of individuals to exposure and potential infection. This parameter accounts for the temporal gap resulting from precautionary measures, effectively postponing the transmission of the disease from infected to susceptible individuals.

2. Model Formulation

We create a deterministic compartmental model $SEII_AQR$ to describe the disease transmission mechanism. Let N be the total population of humans. The total population N is divided into six compartments: Susceptible (S), Exposed (E), Symptomatic Infection (I), Asymptomatic Infection (IA), Quarantine (Q) and individuals that are either recovered or die from COVID-19(R). We also include Vital Dynamics: The natural human natality or recruitment rate denoted by Λ and mortality (death) rate denoted by μ. The schematic diagram is shown in Figure 1.

$$\frac{dS}{dt} = \Lambda - \beta S(I + A_A) - \mu S,$$

$$\frac{dE}{dt} = \beta S(I + I_A) - \gamma E - \mu E,$$

$$\frac{dI}{dt} = p_1\gamma E - (\mu + \theta)I - \alpha I,$$

$$\frac{dI_A}{dt} = p_2\gamma E - \epsilon I_A - \mu I_A,$$

$$\frac{dQ}{dt} = p_3\gamma E - \sigma Q - \mu Q$$

$$\frac{dR}{dt} = \alpha I + \epsilon I_A + \sigma Q - \mu R, \tag{1}$$

with nonnegative initial conditions given by

$$S(0) > 0,\ E(0) > 0,\ I(0) > 0,\ I_A > 0,\ Q(0) > 0,\ R(0) > 0.$$

All the parameters of the system 1 are assumed to be positive for all time $t > 0$.

Due to the implementation of various precautionary measures, like social distancing, self-isolation, personal hygiene, wearing masks, media awareness, etc. We assume that there will be some delay in the time taken by each susceptible person to be exposed and likely infected. We introduce the concept of time delay and use parameter τ which takes into account this delay in the delaying of susceptibility to be exposed and likely infected due to measure; τ is the time delay due to delaying in the transmission of the disease from infected to susceptible due to precautionary measures.

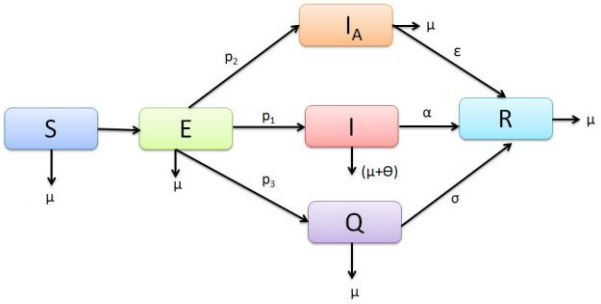

Figure 1. Schematic Diagram of $SELL_AQR$.

$$\frac{dS}{dt} = \Lambda - \beta SI(t-\tau) - \beta SI_A - \mu S,$$

$$\frac{dE}{dt} = \beta SI(t-\tau) - \beta SI_A - \gamma E - \mu E,$$

$$\frac{dI}{dt} = p_1\gamma E - (\mu + \theta)I - \alpha I,$$

$$\frac{dI_A}{dt} = p_2\gamma E - \epsilon I_A - \mu I_A,$$

$$\frac{dQ}{dt} = p_3\gamma E - \sigma Q - \mu Q$$

$$\frac{dR}{dt} = \alpha I + \epsilon I_A + \sigma Q - \mu R, \tag{2}$$

Consider the initial conditions for system 2 are of the form:

$$S(\eta) = f_1(\eta),\ E(\eta) = f_2(\eta),\ I(\eta) = f_3(\eta),\ I_A(\eta) = f_4(\eta),\ Q(\eta) = f_5(\eta),\ R(\eta) = f_6(\eta).$$

where $f_i(\eta) \in C([-\tau, 0], R_+^6)$, which is a Banach space of continuous functions from $[-\tau, 0]$ into $\mathbb{R}_+^6$, such that, $f_\eta \geq 0$ for $\eta \in [-\tau, 0)$, and $f_i(0) > 0$, $\forall\, i = 1, 2, 3, 4, 5, 6, 7$. Table 1 briefly describes all the parameters of the model.

3. Dynamics of Non-Delayed System 1

3.1 Positivity of Solutions

For the COVID-19 model system 1 to be epidemiologically realistic, it is necessary to prove that all the state variables remain positive for all time.

Table 1. Parameter Description.

Parameter	Description	Values
Λ	Birth rate	52000
μ	Death rate	0.0245
β	Rate of transmission	1.7399
γ	Rate of transition from Exposed to I,IA and Q	0.1923
p_1	Fraction of population moves from Exposed to symptomatic class	0.3362
p_2	Fraction of population moves from Exposed to asymptomatic class	0.4204
p_3	Fraction of population moves from Exposed to quarantine class	0.2434
α	Recovery rate of symptomatic infected class	0.07 day^{-1}
ϵ	Recovery rate of asymptomatic infection	0.9 day^{-1}
σ	Recovery rate of quarantine class	0.9 day^{-1}
θ	Rate of disease-induced death	0.0001 day^{-1}

Theorem 1. *Let the initial data be* $\{(S, E, I, I_A, Q, R) \leq 0\} \in \phi$. *Then the solution set* $\{S(t), E(t), I(t), I_A(t), Q(t), R(t)\}$ *of the model system is non negative for all time t.*

Proof. Considering the non-linear system of the model 1, we take the first equation

$$\frac{dS}{dt} = \Lambda - \beta S(I + I_A) - \mu S,$$

$$\frac{dS}{dt} \geq -[\beta S(I + I_A) + \mu]S,$$

$$\int \frac{dS}{S} \geq -\int [\beta S(I + I_A) + \mu]dt,$$

$$\ln S \geq -[\beta S(I + I_A) + \mu]t + c,$$

$$S \geq e^{-[\beta S(I+I_A)+\mu]t} + e^c,$$

$$S(t) \geq S(0)e^{-[\beta S(I+I_A)+\mu]t}.$$

Similarly, it can also been shown that $E(t) > 0$, $I(t), > 0$, I_A $Q(t) > 0$, $R(t) > 0$ for all $t > 0$. Therefore, the disease is uniformly persistent for every positive solution. □

3.2 Invariant Region

Theorem 2. *For the initial conditions* 2, *the solutions of system* 1 *are contained in the region* $\phi \subset \mathbb{R}_+^6$ *defined by*

$$\phi = \left[\{S(t), E(t), I(t), I_A(t), Q(t), R(t)\} \in R_+^6 : N(t) \leq \frac{\Lambda}{\mu}\right].$$

Proof. Let $N = S + E + I + I_A + Q + R$

$$\frac{dN}{dt} = \Lambda - (S + E + I + I_A + Q + R)\mu - \theta I$$

$$\frac{dN}{dt} = \Lambda - \mu N - \theta I$$

$$\frac{dN}{dt} \leq \Lambda - \mu N$$

$$\frac{dN}{dt} + \mu N \leq \Lambda$$

$$Ne^{\mu t} \leq \int e^{\mu t}\Lambda + C$$

$$Ne^{\mu t} \leq \frac{\Lambda e^{\mu t}}{\mu} + C$$

$$N \leq \frac{\Lambda}{\mu} + Ce^{-\mu t}.$$

At $t \to \infty$, $N \to \frac{\Lambda}{\mu}$. Clearly

$$\phi = \left[\{S(t), E(t), I(t), I_A(t), Q(t), R(t)\} \in R_+^6 : N(t) \leq \frac{\Lambda}{\mu}\right].$$

3.3 Analysis of Disease-Free Equilibrium (DFE) E_0

The model gets DFE when the disease has zero induction

Taking the first equation of system (1) with $E = I = I_A = Q = R = 0$ into consideration, we arrive at

$$S_0 = \frac{\Lambda}{\mu}.$$

Then, the disease-free equilibrium (DFE) state E_0 is given by

$$E_0 = \left[\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\right].$$

4. Basic Reproductive Number R_0

R_0 refers to the average number of secondarily infected persons infected by one primary infected patient during the infectious period. To obtain the basic reproduction number, we used the next generation matrix method by Diekmann et al. and Dietz, where $\mathcal{F}$ is the matrix of the new infection terms and $\mathcal{V}$ is the matrix of the transition terms.

At disease-free equilibrium

$$E_0 = \left[\frac{\Lambda}{(\delta + \mu)}, \frac{\delta\Lambda}{\mu(\delta + \mu)}, 0, 0, 0, 0\right]$$

$$\mathcal{F} = \begin{bmatrix} 0 & \frac{\beta\Lambda}{\mu} & \frac{\beta\Lambda}{\mu} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$\mathcal{V} = \begin{bmatrix} \gamma + \mu & 0 & 0 & 0 & 0 \\ -p_1\gamma & (\mu + \theta + \alpha) & 0 & 0 & 0 \\ -p_2\gamma & 0 & \epsilon + \mu & 0 & 0 \\ -p_3\gamma & 0 & 0 & \sigma + \mu & 0 \\ 0 & -\alpha & -\epsilon & -\sigma & \mu \end{bmatrix}.$$

Now, $\mathcal{FV}^{-1}$

$$= \begin{bmatrix} \frac{\beta\gamma\Lambda P_1}{\mu(\mu+\gamma)(\alpha+\theta+\mu)} + \frac{\beta\gamma\Lambda P_2}{\mu(\mu+\epsilon)(\mu+\gamma)} & \frac{\beta\Lambda}{\mu(\alpha+\theta+\mu)} & \frac{\beta\Lambda}{\mu(\mu+\epsilon)} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

The basic Reproduction number is given by

$$R_0 = \frac{\beta\gamma\Lambda P_1}{\mu(\mu+\gamma)(\alpha+\theta+\mu)} + \frac{\beta\gamma\Lambda P_2}{\mu(\mu+\epsilon)(\mu+\gamma)}.$$

5. Stability Analysis of DFE

5.1 Local Stability of Disease-free Equilibrium

Theorem 3. *The Disease Free Equilibrium DEF is locally asymptotically stable if* $R_0 < 1$.

Proof. The Jacobian matrix w.r.t. system 1 is given by

$$J = \begin{bmatrix} -\mu & 0 & -\beta S_0 & -\beta S_0 & 0 & 0 \\ 0 & -(\gamma+\mu) & \beta S_0 & \beta S_0 & 0 & 0 \\ 0 & p_1\gamma & -(\mu+\theta+\alpha) & 0 & 0 & 0 \\ 0 & p_2\gamma & 0 & -(\epsilon+\mu) & 0 & 0 \\ 0 & p_3\gamma & 0 & 0 & -(\sigma+\mu) & 0 \\ 0 & 0 & \alpha & \epsilon & \sigma & -\mu \end{bmatrix}$$

which implies

$$J_{DFE} = \begin{bmatrix} -l_1 & \beta S_0 & \beta S_0 & 0 \\ p_1\gamma & -l_2 & 0 & 0 \\ p_2\gamma & 0 & -l_3 & 0 \\ p_3\gamma & 0 & 0 & -l_4 \end{bmatrix}.$$

Where,

$$l_1 = (\gamma+\mu),\ l_2 = (\mu+\theta+\alpha),\ l_3 = (\epsilon+\mu),\ l_4 = \sigma+\mu.$$

Clearly, two eigenvalues of the matrix JDFE are negative such as -μ and -

μ. The remaining eigenvalues are the roots of the following Polynomial equation

$$\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0$$

where,

$$a_3 = -(l_1 - l_2 - l_3 - l_4) = (l_1 + l_2 + l_3 + l_4)$$

$$a_2 = (l_1 + l_2 + l_3 + l_4)^2/2 - (l_1^2 + l_2^2 + l_3^2 + l_4^2)/2 - \beta\gamma S_0(p_1 + p_2)$$

$$a_1 = l_1l_2l_3 + l_1l_2l_4 + l_1l_3l_4 + l_2l_3l_4 - \beta\gamma l_2p_2S_0 - \beta\gamma l_3p_1S_0 - \beta\gamma l_4p_1S_0 - \beta\gamma l_4p_2S_0$$

$$a_0 = l_1l_2l_3l_4 - \beta\gamma l_2l_4p_2S_0 - \beta\gamma l_3l_4p_1.$$

According to the Routh-Hurwitz criterion, the above equation will give negative roots or roots with negative real parts if the following condition is satisfied:

$$a_3 > 0, \begin{vmatrix} a_3 & a_1 \\ 1 & a_2 \end{vmatrix} > 0, \begin{vmatrix} a_3 & a_1 & 0 \\ 1 & a_2 & a_0 \\ 0 & a_3 & a_1 \end{vmatrix} > 0.$$

Hence, the disease-free equilibrium point E_0 of the system is locally asymptotically stable, when $R_0 < 1$. □

5.2 Global Stability of Disease-free Equilibrium

We now study the global stability of disease-free equilibrium, using the theorem by Castillo-Chavez et al. [23]

Theorem 4. *If the given mathematical model can be written in the form*:

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

$$\frac{dY}{dt} = G(X, Y), G(X, Y) = 0 \tag{3}$$

where $X = S^T$, $Y = (E, I, I_A, Q, R)^T$, *denoting the number of uninfected individuals and denoting the number of COVID-19-infected people respectively. Let the disease free equilibrium of this system be*

$$U_0 = (X^*, 0) = \left(\frac{\Lambda}{\mu}, 0\right)$$

where 0 *is a zero vector*.

For the global asymptotically stable, the following condition ($H1$) *and* ($H2$) *must be satisfied*.

($H1$): *For* $\frac{dX}{dt} = F(X, 0)$, 0 *is global asymptotically stable*.

($H2$): $G(X, Y) = AY - \hat{G}(X, Y)$, $\hat{G}(X, Y) \geq 0$ *for* $(X, Y) \in \Omega$

where $A = D_Y G(X^*, 0)$ *is an M-matrix* (*the off-diagonal elements of A are nonnegative*) *and* Ω *is the region where the model makes biological sense. If the given system of differential equations of our model satisfies the given condition in* (2) *then the fixed point* $U_0 = (X^*, 0)$ *is a global asymptotically stable* (*g.a.s*) *equilibrium of* (2) *provided* $R_0 < 1$, *and the assumption* ($H1$) *and* ($H2$) *are satisfied*.

Theorem 5. (*global asymptotic stability of DFE*) *The DFE* E_0 *of model* 1 *is global asymptotically stable if* $R_0 < 1$.

Proof. First, we rewrite the system of differential equation of our model 1 as $X = ST$ and $Y = (E, I, I_A, Q, R)^T$.

Then, the DFE is given by

$$U_0 = (X^*, 0) = \left(\frac{\Lambda}{\mu}, 0\right)$$

and the system $\frac{dX}{dt} = F(X, 0)$ becomes

$$\dot{S} = \Lambda - \mu S.$$

This equation has a unique equilibrium point

$$X^* = \left(\frac{\Lambda}{\mu}, 0\right) \tag{4}$$

which is globally asymptotically stable. Therefore, condition (H1) is satisfied.

We now verify the second condition (H2). For model 1, we have

$$G(X, Y) = \begin{bmatrix} \beta S(I + I_A) - \gamma E - \mu E p_1 \gamma E - (\mu + \theta + \alpha) I \\ p_2 \gamma E - (\epsilon + \mu) I_A \\ p_3 \gamma E - (\sigma + \mu) Q \\ \alpha I + \epsilon I_A + \sigma Q - \mu R \end{bmatrix}$$

$$D_Y G(X^*, 0) = A = F - V$$

$$= \begin{bmatrix} -(\gamma + \mu) & \beta S_0 & \beta S_0 & 0 & 0 \\ p_1 \gamma & -(\mu + \theta + \alpha) & 0 & 0 & 0 \\ p_2 \gamma & 0 & -(\epsilon + \mu) & 0 & 0 \\ p_3 \gamma & 0 & 0 & -(\sigma + \mu) & 0 \\ 0 & 0 & \epsilon & \sigma & -\mu \end{bmatrix}$$

Clearly, we see that A is an M-matrix, i.e. all the off-diagonal elements of A are non-negative.

$$\hat{G}(X, Y) = AY - G(X, Y) = \begin{bmatrix} [\beta(I + I_A)](S - S_0) \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

for all $\hat{G}(X, Y) \geq 0$ for all $(X, Y) \in \Omega$. Therefore, conditions (H1) and (H2) are satisfied. Hence, disease-free equilibrium is globally asymptotically stable.

6. Stability Analysis of EE

6.1 Existence of Endemic Equilibrium point

Let us denote the Endemic Equilibrium by $E_1 = (S^*, V^*, E^*, I^*, I_A^*, R^*)$

$$S^* = \frac{\Lambda}{\mu + \beta\left(\frac{\gamma p_2}{\mu + \epsilon} + \frac{\gamma p_1}{\alpha + \theta + \mu}\right) E^*}$$

$$I^* = \frac{p_1 \gamma E^*}{\alpha + \theta + \mu}$$

$$I_A^* = \frac{p_2\gamma E^*}{\epsilon + \mu}$$

$$Q^* = \frac{p_3\gamma E^*}{\sigma + \mu}$$

$$R^* = \frac{\left[\frac{p_1\gamma\alpha}{\alpha + \gamma + \mu} + frac\epsilon\gamma p_2\mu + \epsilon + \frac{p_3\gamma\sigma}{\sigma + \mu}\right]E^*}{\mu}$$

where

$$A + \left[\frac{\gamma p_2}{\mu + \epsilon} + \frac{\gamma p_1}{\alpha + \theta + \mu}\right]. \qquad \square$$

6.2 Local stability of endemic equilibrium

Theorem 6. *The endemic equilibrium* E_1 *is locally asymptotically stable if* $R_0 > 1$, *otherwise it is unstable.*

Proof. The Jacobian matrix of the system 1 at endemic equilibrium point E_1 is obtained as follows:

$$J_{E1} = \begin{bmatrix} -q & 0 & -\beta S^* & -\beta S^* & 0 & 0 \\ a_{21} & -r & \beta S^* & \beta S^* & 0 & 0 \\ 0 & p_1\gamma & -s & 0 & 0 & 0 \\ 0 & p_2\gamma & 0 & -t & 0 & 0 \\ 0 & p_3\gamma & 0 & 0 & -u & 0 \\ 0 & 0 & \alpha & \epsilon & \sigma & -u \end{bmatrix}$$

where $q = [\beta(I^* + I_A^*) + \mu]$, $r = (\gamma + \mu)$, $s = (\mu + \theta + \alpha)$, $t = (\epsilon + \mu)$

$u = (\sigma + \mu)$, $a_{21} = \beta(I^* + I_A^*)$. Clearly, one eigenvalue of the matrix J_{E1} is negative -μ and the remaining eigenvalues are the roots of the following Polynomial equation:

$$\lambda^5 + c_4\lambda^4 + c_3\lambda^3 + c_2\lambda^2 + c_1\lambda^2 + c_1\lambda + c_0 = 0$$

where

$$c_4 = (q + r + s + t + u)$$

$$c_3 = (q + r + s + t + u)^2/2 - (q^2 + r^2 + s^2 + t^2 + u^2)/2 - \beta\gamma S^*(p_1 + p_2)$$

$$c_2 = qr(s + t + u) + (qs + rs)(t + u) + tu(q + r) + \beta\gamma S^*[(a_{21} - q - u)$$

$$(p_1 + p_2) - (sp_1 + tp_2)]$$

$$c_1 = qrs(t + u) + qtu(r + s) + rstu + \beta\gamma S^*(a_{21}s + a_{21}u - qu)(p_1 + p_2)$$

$$- \beta\gamma S^*(sp_1 + tp_2) - \beta\gamma S^*(sp_1 + up_2)$$

$$c_0 = qr(s + t + u) + (qs + rs)(t + u) + tu(q + r) + \beta\gamma S^*[(a_{21} - q - u)$$

$$(p_1 + p_2) - (sp_1 + tp_2)]$$

According to the Routh-Hurwitz criterion, the above equation will give negative roots or negative real parts if the following condition is satisfied:

$$c_4 > 0, \begin{vmatrix} c_4 & c_2 \\ 1 & c_3 \end{vmatrix} > 0, \begin{vmatrix} c_4 & c_2 & c_0 \\ 1 & c_3 & c_1 \\ 0 & a_4 & a_2 \end{vmatrix} > 0, \begin{vmatrix} c_4 & c_2 & c_0 & 0 \\ 1 & c_3 & c_1 & 0 \\ 0 & c_4 & c_2 & c_0 \\ 0 & 1 & c_3 & c_1 \end{vmatrix} > 0$$

Hence, the endemic equilibrium point E_1 of the system is locally asymptotically stable when $R_0 > 1$.

7. Dynamic with Delay 2

The Positivity of the system 2 can be proved in a similar way as

[24] and the boundedness of system 2 can be proved in a similar way as in Section 3.2.

7.1 Equilibrium points and their stability

As mentioned by Tipsri and Chinviriyasit [17], the equilibrium solutions are the same for the system with and without time delay. Therefore, to obtain the equilibrium points, we use $\tau = 0$. Hence, the Disease-Free and Endemic Equilibrium points of the system 2 are the same as obtained in section 3.3 and 6.1 respectively.

7.2 Local stability of disease-free equilibrium point

In this subsection, we will discuss the stability of the system 2 around a disease-free equilibrium point. For $\tau > 0$

The Jacobian matrix w.r.t. system 2 is given by

$$J = \begin{bmatrix} -\mu & 0 & -\beta S_0 e^{-\lambda\tau} & -\beta S_0 & 0 & 0 \\ 0 & -(\gamma+\mu) & \beta S_0 e^{-\lambda\tau} & \beta S_0 & 0 & 0 \\ 0 & p_1\gamma & -(\mu+\theta+\alpha) & 0 & 0 & 0 \\ 0 & p_2\gamma & 0 & -(\epsilon+\mu) & 0 & 0 \\ 0 & p_3\gamma & 0 & 0 & -(\sigma+\mu) & 0 \\ 0 & 0 & \alpha & \epsilon & \sigma & -\mu \end{bmatrix}$$

which implies

$$J_{DFE} = \begin{bmatrix} -l_1 & \beta S_0 e^{-\lambda\tau} & \beta S_0 & 0 \\ p_1\gamma & -l_2 & 0 & 0 \\ p_2\gamma & 0 & -l_3 & 0 \\ p_3\gamma & 0 & 0 & -l_4 \end{bmatrix}.$$

Where,

$$l_1 = (\gamma+\mu),\ l_2 = (\mu+\theta+\alpha),\ l_3 = (\epsilon+\mu),\ l_4 = \sigma+\mu.$$

Clearly, two eigenvalues of the matrix JDFE are negative such as -μ and -μ. The remaining eigenvalues are the roots of the following Polynomial equation.

$$\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0 \tag{5}$$

where,

$$a_3 = -(l_1 - l_2 - l_3 - l_4) = (l_1 + l_2 + l_3 + l_4)$$

$$a_2 = (l_1 + l_2 + l_3 + l_4)^2/2 - (l_1^2 + l_2^2 + l_3^2 + l_4^2)/2 - \beta\gamma S_0 e^{-\lambda\tau}(p_1 + p_2)$$

$$a_1 = l_1l_2l_3 + l_1l_2l_4 + l_1l_3l_4 + l_2l_3l_4 - \beta\gamma e^{-\lambda\tau}l_2p_2S_0 - \beta\gamma e^{-\lambda\tau}l_3p_1S_0$$

$$-\beta\gamma l_4p_1S_0 - \beta\gamma l_4p_2S_0$$

$$a_0 = l_1l_2l_3l_4 - \beta\gamma e^{-\lambda\tau}l_2l_4p_2S_0 - \beta\gamma l_3l_4p_1.$$

Ruan and Wei's corollary 2.4 [25] states that for $t > 0$ if instability arises for a specific value of the delay, a characteristic root of 5 must intersect the imaginary axis.

The characteristic equation can also be written as

$$f(\lambda) = (\lambda^4 + b_3\lambda^3 + b_2\lambda^2 + b_1\lambda + b_0) + e^{-\lambda\tau}(c_3\lambda^3 + c_2\lambda^2 + c_1\lambda + c_0) = 0 \quad (6)$$

For $\tau > 0$, (6) is a transcendental characteristic equation and the roots will be of the form, $\lambda = \eta(\tau) + i\omega(tau)$, where $\omega > 0$. As explained by Mukandavire [26], the roots of a transcendental equation will have positive real parts if and only if it has purely imaginary roots. We will aim to obtain the conditions for which no such purely imaginary root exists for 6. These conditions will be then sufficient to conclude that all the roots of 6 have negative real parts. Consider, $\lambda = i\omega(\omega > 0)$ is a purely imaginary root of 6. Then, 6 becomes

$$\omega^4 - ib_3\omega^3 + b_2\omega^2 + ib_1\omega + b_0 + [\cos(\lambda\tau) - i\sin(\lambda\tau)]$$

$$(-ic_3\omega^3 + c_2\omega^2 + ic_1\omega + c_0) = 0.$$

Separating real-imaginary parts,

Real:

$$\omega^4 - b_2\omega^2 + b_0 - \cos(\lambda\tau)[c_2\omega^2 - c_0] - \sin(\lambda\tau)[c_3\omega^3 - c_1\omega] = 0$$

Complex:

$$-b_3\omega^3 + b_1\omega - \cos(\omega\tau)[c_2\omega^2 - c_0] + \sin(\omega\tau)[c_2\omega^2 - c_0] = 0$$

Squaring both sides of the above two equations and adding we get,

$$\omega^8 + \omega^6(-2b_2 + b_3^2 + c_3^2) + \omega^4(b_2^2 + 2b_0 - 2b_1b_3 + 2c_1c_3 - c_2)$$

$$+ \omega^2(-2b_0b_2 + b_1^2 - c_1^2 + 2c_0c_2) + b_0^2 - c_0^2 = 0.$$

Taking $s = \omega^2$. We have

$$s^4 + s^3(-2b_2 + b_3^2 + c_3^2) + s^2(b_2^2 + 2b_0 - 2b_1b_3 + 2c_1c_3 - c_2)$$

$$+ s(-2b_0b_2 + b_1^2 - c_1^2 + 2c_0c_2) + (b_0^2 - c_0^2) = 0 \quad (7)$$

if we assume that

$C2$:

$$(-2b_2 + b_3^2 + c_3^2) = g_3 > 0$$

$$(b_2^2 + 2b_0 - 2b_1b_3 + 2c_1c_3 - c_2) = g_2 > 0$$

$$(-2b_0b_2 + b_1^2 - c_1^2 + 2c_0c_2) = g_1 > 0$$

$$(b_0^2 - c_0^2) = g_0 > 0$$

and

$$\begin{vmatrix} g_3 & g_1 \\ 1 & g_2 \end{vmatrix} > 0, \begin{vmatrix} g_3 & g_1 & 0 \\ 1 & g_2 & g_0 \\ 0 & g_3 & g_1 \end{vmatrix} > 0$$

then by Routh-Hurwitz criterion the roots for (7) will have negative real parts. However, there does not exist ω such that $s = \omega^2$ is negative. This poses a contradiction.

Hence, whenever the conditions in ($C2$) are true, there does not exist a purely imaginary root of the transcendental equation (6). Hence, we have the following theorem.

Theorem 7. *Let,* $R_0 < 1$ *then for* $\tau > 0$, *the disease-free equilibrium point of system* (2) *is locally asymptotically stable if condition in* ($C2$) *is satisfied. Also, the disease-free equilibrium point of the system* (2) *is unstable for* $R_0 > 0$.

7.3 Local stability of endemic equilibrium point

The Jacobian matrix of the system 1 at endemic equilibrium point E_1 is obtained as follows:

$$J_{E1} = \begin{bmatrix} -q & 0 & -\beta S^* e^{-\lambda\tau} & -\beta S^* & 0 & 0 \\ a_{21} & -r & \beta S^* e^{-\lambda\tau} & \beta S^* & 0 & 0 \\ 0 & p_1\gamma & -s & 0 & 0 & 0 \\ 0 & p_2\gamma & 0 & -t & 0 & 0 \\ 0 & p_3\gamma & 0 & 0 & -u & 0 \\ 0 & 0 & \alpha & \epsilon & \sigma & -\mu \end{bmatrix}$$

where $q = [\beta(I^* e^{-\lambda\tau} + I_A^*) + \mu]$, $r = (\gamma + \mu)$, $s = (\mu + \theta + \alpha)$, $t = (\epsilon + \mu)$, $u = (\sigma + \mu)$, $a_{21} = \beta(I^* e^{-\lambda\tau} + I_A^*)$. Clearly, one eigenvalue of the matrix J_{E1} is negative -μ and the remaining eigenvalues are the roots of the following Polynomial equation:

$$\lambda^5 + c_4\lambda^4 + c_3\lambda^3 + c_2\lambda^2 + c_1\lambda + c_0 = 0$$

where

$$c_4 = (q + r + s + t + u)$$

$$c_3 = (q + r + s + t + u)^2/2 - (q^2 + r^2 + s^2 + t^2 + u^2)/2 - \beta\gamma S^* e^{-\lambda\tau}(p_1 + p_2)$$

$$c_2 = qr(s + t + u) + (qs + rs)(t + u) + tu(q + r) + \beta\gamma S^*[(a_{21} - q - u)$$

$$(p_1 + p_2) - (sp_1 + tp_2)]$$

$$c_1 = qrs(t + u) + qtu(r + s) + rstu + \beta\gamma S^*(a_{21}s + a_{21}u - qu)(p_1 + p_2)$$

$$- \beta\gamma S^*(sp_1 + tp_2) - \beta\gamma S^* e^{-\lambda\tau}(sp_1 + up_2)$$

$$c_0 = qr(s + t + u) + (qs + rs)(t + u) + tu(q + r) + \beta\gamma S^*[(a_{21} - q - u)$$

$$(p_1 + p_2) - (sp_1 + tp_2)]$$

For $\tau > 0$. The transcendental characteristic equation is given by

$$\lambda^5 + f_4\lambda^4 + f_3\lambda^3 + f_2\lambda^2 + f_1\lambda + f_0 + e^{-\lambda\tau}(j_4\lambda^4 + j_3\lambda^3 + j_2\lambda^2 + j_1\lambda + j_0) = 0 \quad (8)$$

the roots will be of the form, $\lambda = \eta(\tau) + i\omega(\tau)$, where $\omega > 0$. As done previously for the local stability of E_0, we will aim to obtain the conditions for which no purely imaginary root exists for equation (8). Let if possible, Equation (8) have a purely complex root of the form: $\lambda = i\omega$ Then, (8) becomes:

$$i\omega^5 + f_4\omega^4 + if_3\omega^3 + f_2\omega^2 + if_1\omega + f_0[\cos(\lambda\tau) - i\sin(\lambda\tau)]$$

$$(j_4\omega^4 + j_3\omega^3 + j_2\omega^2 + j_1\omega + j_0) = 0$$

Separating real-imaginary parts:

Real:

$$F_4\omega^4 - f_2\omega^2 + f_0 + \cos(\lambda\tau)(j_4\omega^4 - j_2\omega^2 j_0) - \sin(\lambda\tau)(j_3\omega^3 - j\omega)$$

Complex:

$$\omega^5 - f_3\omega^3 + f_2\omega + \cos(\lambda\tau)(j_3\omega^3 - j\omega) - \sin(\lambda\tau)(j_4\omega^4 - j_2\omega^3 j_0).$$

Squaring both sides of the above equations and adding we get

$$\omega^1 0 + v_1\omega^8 + v_2\omega^6 + v_3\omega^4 + v_4\omega^2 + v_5 = 0 \tag{9}$$

where,

$$v_1 = f_4^2 - 2f_1 - j_4^2$$

$$v_2 = -2f_4f_2 + 2f_1 + 2j_4j$$

$$v_3 = f_2^2 + 2f_4f_0 - 2f_3f_1 + j_2^2\omega^4 - 2j_4j_0$$

$$v_4 = -2f_2f_0 + f_1^2 - j_2^2 + 2j_3j + 2j_2j_0$$

$$v_5 = f_0^2 - j_0^2.$$

Put $s = \omega^2$ in (9) we get

$$s^5 + v_1s^4 + v_2s^3 + v_3s^2 + v_4s + v_5 = 0$$

If we assume $|H_n|$ for $n = 1, 2, 3, 4, 5,$ where for each n, H_n is a Hurwitz matrix of order $n * n,$ with general form:

$$H_n = \begin{bmatrix} v_1 & 1 & 0 & \ldots & 0 \\ v_3 & v_2 & v_1 & \ldots & 0 \\ v_5 & v_4 & v_3 & \ldots & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \tag{10}$$

with $v_j = 0$ if $j > 5$ or $j < 0$. According to the Routh-Hurwitz criterion, the roots of Equation (9) will exhibit negative real parts. Nevertheless, there is no value of ω that can make the expression $s = \omega^2$ negative. This leads to a contradiction. Consequently, the conditions stated in (10) are enough to establish that all roots of Equation (8) hold negative real parts for $\tau > 0$. As a result, we can state the following theorem.

Theorem 8. *Suppose* E_1 *is an endemic equilibrium point of the system* (2), *it will be locally asymptotically stable for* $\tau > 0$ *under the condition that each of the seven Hurwitz matrices defined as in* (10) *satisfies two criteria*:

1. *The determinant of the matrix, denoted as* $| H_n |$, *must be greater than zero* (*i.e.*, $| H_n | > 0$).

2. *The basic reproduction number* R_0 *must be greater than* 1 (*i.e.*, $R_0 > 1$). *Otherwise unstable.*

8. Sensitivity Analysis

In this section, we examine the impact of the parameters used to express the basic reproduction number, R_0, through sensitivity analysis.

This demonstrates that an alteration in these parameters results in an alteration in R_0. It is used to identify the variables with a significant impact on R_0 and determine which ones should be the focus of intervention measures. Sensitivity indices make it possible to quantify the proportional change in a variable when a parameter is altered.

The forward sensitivity index of a variable, with regard to a specific parameter, is used for that

$$\alpha_{\phi}^{R_0} = \frac{\partial R_0}{\partial \phi} \frac{\phi}{R_0}$$

where $\phi = [\Lambda, \beta, \mu, \gamma, \theta, \alpha, \epsilon, P_1, P_2]$. The analytical equation for the sensitivity of R_0 to each parameter it comprises can be calculated using the formula mentioned above.

As a result, Figure 2 shows the sensitivity index of parameters i.e. $\Lambda, \beta, \mu, \gamma, \theta, \alpha, \epsilon, P_1, P_2$ respectively on R_0.

The positive indices indicate a direct relationship between the parameters and R_0, that is if the parameter increases/decrease then the value of R_0 will increase/decrease.

Therefore in order to control COVID-19 from the population, we need to

reduce the Basic Reproduction number, we can achieve this by reducing the parameters which give positive indices i.e. γ, r, β, P_1, P_2, Λ, here birth rate Λ and rate of transmission β are the most sensitive parameters of R_0 In light of the uncontrollable nature of the birth rate, our focus shifts to managing the rate of disease transmission. To achieve this, we must limit our contact rate through measures like quarantine and social distancing. By taking responsible actions and collectively embracing these precautions, we can build a shield of protection against infectious diseases, fostering a healthier and safer society. Also $\alpha_{\beta}^{R_0} = +1$ means that if β increase by 1% than R_0 will also increase by 1%.

The negative indices indicate that there is an inverse relationship between the parameters and R_0, that is if the parameter decrease/increases then the value of R_0 will increase/decrease. μ, θ, α, ϵ have negative indices, among the μ is the highest sensitive if the death rate increase than R_0 decrease. The strength of intervention h has negative indices which imply that if we implement strict intervention measures then R_0 will decrease which will lead to decreasing the Infected population.

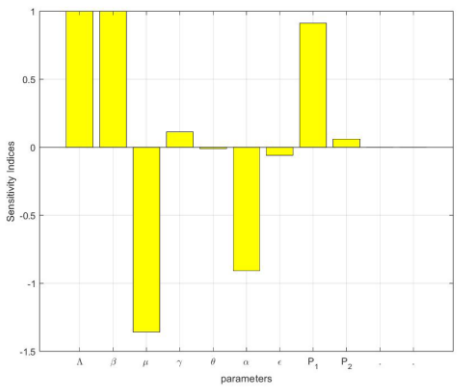

Figure 2. Forward sensitivity of R_0

In Figure 3, we observe contour plots representing the relationship between the two parameters β and μ, and their corresponding values of R_0. The contour plots reveal that R_0 exhibits an upward trend as β increases and

μ decreases. As R_0 significantly influences the spread of the disease, it becomes crucial to understand how it depends on these distinct parameters. This knowledge empowers us to implement appropriate measures aimed at reducing R_0, thereby effectively controlling the disease's transmission.

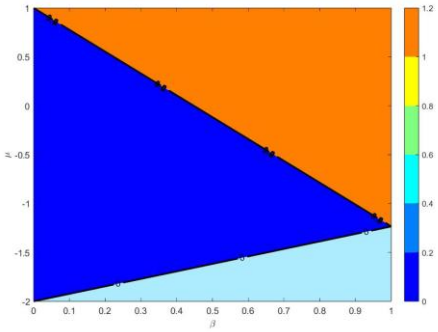

Figure 3. Contour plots of the basic reproduction number R_0 with respect to β and μ.

9. Numerical Simulation

For the Numerical Simulation of the proposed model, we illustrate the mathematical findings using the MATLAB program, the value of parameters are listed in the table. Figure 4 shows the Variation of $SEII_AQR$ without time delay corresponding to the values of $R_0 > 1$ for different values of initial numbers of each compartment with time $t = 0$ to 100. Figure 5 shows the Variation of $SEII_AQR$ without time delay corresponding to the values of $R_0 > 1$ for different values of initial numbers of each compartment with time $t = 0$ to 100.

By altering the values of tau τ, the impact of the time delay is observed. In figures we see a simulation of a system showing a variation in population with the effect of time delay on $SEII_AQR$ when $\tau = 0.5, 3, 5, 7$. From this, we found out that if the time delay τ increases, it means there is a longer delay between the time when an individual becomes exposed and the time they start infecting others. Various precautionary measures will delay the Susceptible from being Exposed and becoming infected. As τ increases, the

number of susceptible individuals will decrease initially. This is because the infection will spread at a slower rate, and more people will move from the susceptible compartment to the exposed compartment due to the delay in transmission.

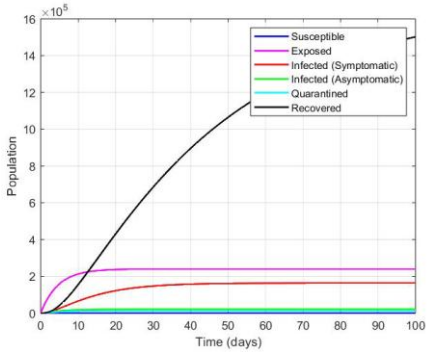

Figure 4. Variation of $SEII_AQR$ without time delay corresponding to the values of $R_0 > 1$ for different values of initial numbers of each compartment with time $t = 0$ to 100.

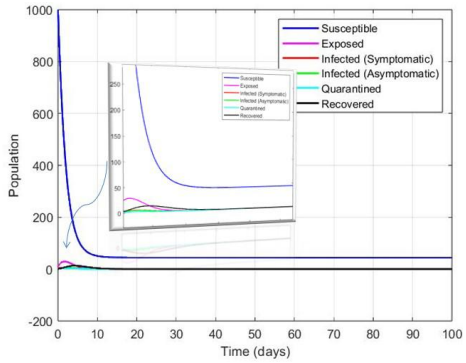

Figure 5. Variation of $SEII_AQR$ without time delay corresponding to the values of $R_0 > 1$ for different values of initial numbers of each compartment with time $t = 0$ to 100.

The number of exposed individuals will increase with an increase in τ. This is because the longer time delay allows more individuals to stay in the exposed compartment before becoming infectious. As a result, the compartment of exposed individuals will grow larger.

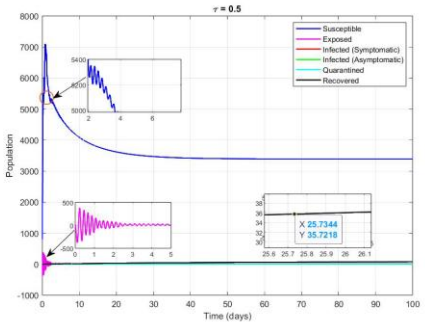

Figure 6. Simulation of a system showing the variation of the population with the effect of time delay on $SEII_AQR$ when $\tau = 0.5$.

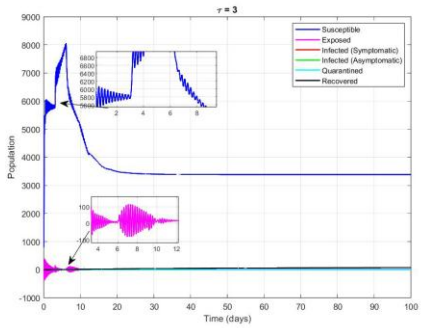

Figure 7. Simulation of a system showing the variation of the population with the effect of time delay on $SEII_AQR$ when $\tau = 3$.

The number of symptomatic infected individuals will decrease with an increase in τ. This is because there is a longer period for the infection to spread before individuals become symptomatic and move from the exposed compartment to the infected compartment. With a longer time delay in transmission, more individuals will move from the Exposed compartment to the Asymptomatic Infection compartment instead of becoming symptomatic and moving to the Symptomatic Infected compartment immediately.

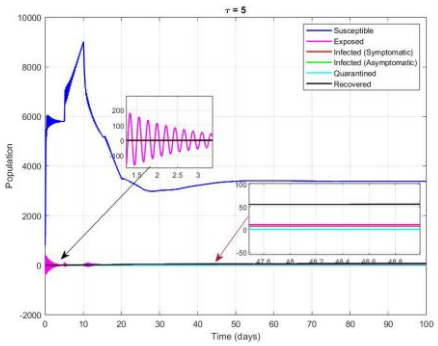

Figure 8. Simulation of a system showing the variation of the population with the effect of time delay on $SEII_AQR$ when $\tau = 5$.

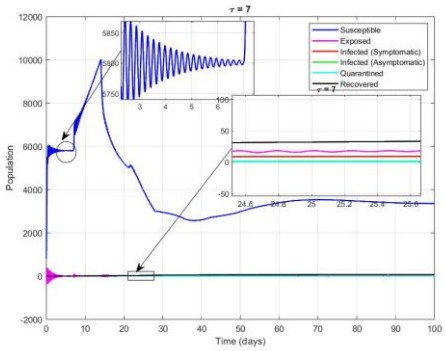

Figure 9. Simulation of a system showing the variation of the population with the effect of time delay on $SEII_AQR$ when $\tau = 7$.

The number of people in the Quarantined compartment is likely to initially rise as the time delay tau increases. This is because those in the quarantined compartment are those who have been exposed to the virus but are not yet infectious because of the quarantine and the prolonged transmission delay. Instead, they shift from the exposed compartment to the quarantined compartment.

The number of recovered individuals will increase with an increase in τ. This is because the longer time delay allows more individuals to recover before becoming symptomatic and subsequently being quarantined or isolated.

In the initial phase, we see oscillatory behaviour, which arises due to the impact of precautionary measures like social distancing, self-isolation, and personal hygiene. These measures slow down the transmission of the disease, resulting in a longer time before an exposed individual becomes infected. As a result, the dynamics of the disease undergo oscillations, with periods of increasing and decreasing exposed individuals. Due to its influence on the effectiveness of disease control strategies. Our finding can help in setting the timing of control measures, as implementing interventions during periods of high transmission may have a more significant impact on disease control. It is possible to better prepare for and respond to outbreaks by anticipating their time and intensity.

From Figure 10 we also see large oscillatory behaviour this is due to the high intensity of transmission as we increase β. Oscillations can lead to fluctuations in the number of infectious individuals over time. Figure 11 shows the effect of variations in Transmission Rate β on $SEII_AR$ Model Dynamic.

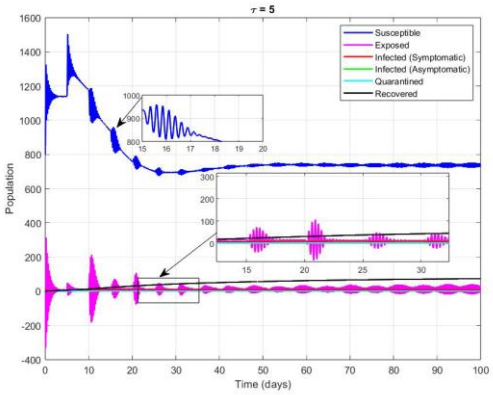

Figure 10. Variation of SEIIAQR with effect of time delay when $\tau = 5\tau = 5$ and higher values of β.

10. Conclusion

In order to curb the COVID-19 diseases' rapid spread, a number of preventative measures have been developed because the virus has irreparably affected society as a whole. Lockdowns, social exclusion, and

other therapies have all proved effective, but they also hinder the corona viruses capacity to propagate. It is crucial to appreciate the implications of these delays in order to create efficient pandemic response tactics.

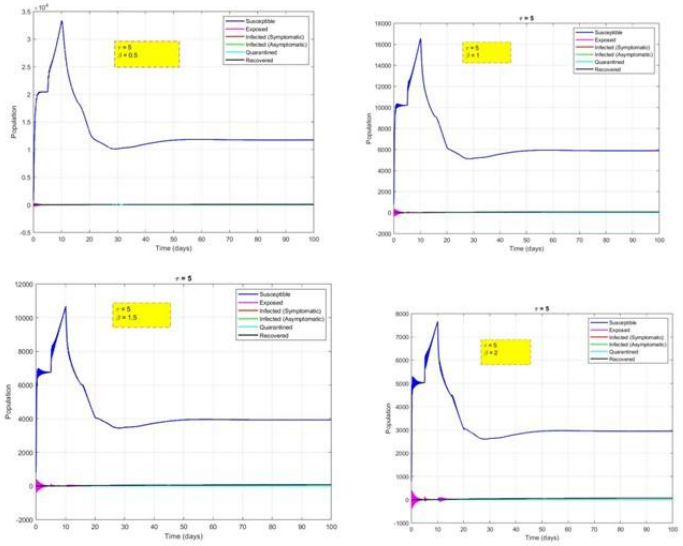

Figure 11. Effect of Variations in Transmission Rate β on $SEII_AQR$ Model Dynamics.

In this study, mathematical modelling is investigated, and delay differential equations (DDEs) are used to look into how delays caused by preventative measures affect the dynamics of COVID-19.

We have developed a deterministic compartmental model, denoted as SEIIAQR, to characterize the mechanism of disease transmission. The total human population, denoted as N, is divided into six compartments: Susceptible (S), Exposed (E), Symptomatic Infection (I), Asymptomatic Infection (IA), Quarantine (Q), and individuals who have either recovered from or succumbed to COVID-19 (R). In light of various precautionary measures, such as social distancing, self-isolation, personal hygiene, mask-wearing, and media awareness, we consider the presence of a delay in the time taken for susceptible individuals to be exposed and potentially infected. To account for this delay caused by precautionary measures, we introduce the parameter τ, representing the extent of the delay in susceptibility to exposure

and infection. In other words, τ captures the time lag in disease transmission from infected to susceptible individuals due to the implementation of preventive interventions.

First, we analyze the Dynamics of a non-delayed system 1, we explored the presence and stability of two critical points in the model: the Disease-Free Equilibrium (DFE) and the Endemic Equilibrium (EE). Through stability analysis, we determined that the DFE is locally asymptotically stable when the basic reproduction number, denoted as R_0, is less than 1, and it becomes globally asymptotically stable under the same condition that is if $R_0 < 1$. On the other hand, the EE is locally asymptotically stable when R_0 is greater than 1.

Next we Dynamic with delay 2, for $\tau > 0$, we derive the expression for Disease-Free Equilibrium (DFE) and the Endemic Equilibrium (EE). We provide stability criteria for both the Disease-Free Equilibrium (DFE) and the Endemic Equilibrium (EE). For $R_0 < 1$, the DFE of the system (2) is locally asymptotically stable when the condition in (C2) is satisfied and $\tau > 0$. Conversely, the DFE is unstable for $R_0 > 1$.

Moreover, for E_1, an endemic equilibrium point of the system (2), it will be locally asymptotically stable with $\tau > 0$, subject to two conditions being met for each of the seven Hurwitz matrices (H_n) as defined in (10). The first condition requires the determinant of the matrix, represented as $|H_n|$, to be greater than zero ($|H_n| > 0$). The second condition demands that the basic reproduction number, R_0, must be greater than 1 ($R_0 > 1$). Failure to satisfy these criteria renders the EE unstable. These findings offer crucial insights into the stability properties of both equilibrium points, contributing to a deeper understanding of disease dynamics, especially considering time delays.

We also investigate the impact of various parameters on the basic reproduction number, denoted as R_0, through sensitivity analysis. By examining how changes in these parameters affect R_0, we aim to identify key variables that significantly influence the transmission of COVID-19 and

should be targeted for intervention measures. The forward sensitivity index, denoted as $\alpha_{\phi}^{R_0}$, is utilized to quantify the proportional change in R0 with respect to specific parameters, including Λ, β, μ, γ, θ, α, ϵ, P_1, and P_2. The sensitivity indices reveal whether a parameter has a positive or negative relationship with R_0, indicating whether an increase or decrease in the parameter will lead to a corresponding increase or decrease in R_0. Among the parameters with positive indices, β, the rate of disease transmission, and Λ, the birth rate, are the most sensitive to R_0, suggesting that managing disease transmission and limiting contact rates through measures like quarantine and social distancing are crucial in controlling COVID-19. Conversely, parameters with negative indices, such as μ, the death rate, indicate an inverse relationship with R_0, suggesting that increasing intervention measures can lead to a decrease in R_0 and the infected population. Contour plots illustrate the relationship between parameters β and μ and their corresponding values of R_0, highlighting how R_0 varies with changes in these specific parameters and the potential impact on disease transmission. Understanding these dependencies empowers the implementation of targeted interventions aimed at reducing R_0, thus effectively controlling the spread of COVID-19.

In our numerical simulation of the proposed SEIIAQR model using MATLAB, we analyzed the impact of time delay τ and illustrated the findings with Figures 4, 5, 6, 7, 8, and 9. As τ increases, there is a longer delay between exposure and infection, caused by precautionary measures. The number of susceptible individuals initially decreases due to slower transmission, while the number of exposed individuals increases with more time for incubation. Symptomatic infected individuals decrease with increased τ, as the infection takes longer to develop symptoms.

Moreover, the number of asymptomatic infected individuals rises due to the longer time delay in transmission, and more exposed individuals move to the asymptomatic compartment. The number of people in quarantine initially increases due to prolonged transmission delay, shifting from the exposed compartment. Additionally, the number of recovered individuals increases, as they have more time to recover before becoming symptomatic.

The model exhibits oscillatory behaviour during the initial phase, influenced by precautionary measures, leading to varying numbers of exposed individuals. Understanding the effects of τ helps in setting optimal timing for control measures to combat disease transmission effectively. Anticipating outbreak timing and intensity aids in better preparation and response.

Declarations

Conflict of interest/Competing interests: On behalf of all authors, the corresponding author states that there is no conflict of interest.

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