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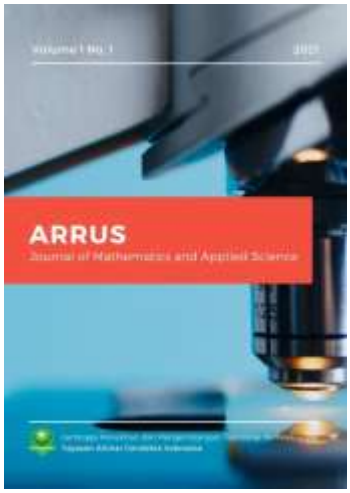

RESEARCH ARTICLE

Bifurcation Analysis Of An Infectious Respiratory Disease With Lockdown And Social Distancing

Henry Milimo Wanjala*, Mark Onyango& Jimrise Ochwach

Department of Physical Sciences, Faculty of Science, Chuka University, Chuka, Kenya.

*Corresponding author: Department of Physical Sciences, Faculty of Science, Chuka University, Chuka, Kenya

E-mail: teomat240@gmail.com

Abstract: This paper analyzes the impact of lockdown, social distancing, and isolation of symptomatic patients on the transmission of infectious respiratory diseases. The study uses predictive mathematical models to explore disease progression and evaluate control strategies. The results show that the disease-free equilibrium is locally stable but globally unstable, indicating that while these measures can slow the infection, they alone cannot eradicate the disease. Local stability was assessed using the determinant-trace matrix method, while global stability was analyzed through the Lyapunov-Krasovskii method, revealing instability around the global endemic equilibrium. Bifurcation analysis was conducted to identify critical points where small parameter changes could cause significant shifts in system behavior. Numerical simulations were performed using Python's NumPy and PyPlot libraries to understand the dynamics of disease spread and evaluate various intervention strategies. The simulations demonstrated how changes in control measures affect the disease's trajectory. In the absence of effective treatments or vaccines, the findings suggest that social distancing, lockdowns, and isolation are vital for controlling the spread of the pandemic and reducing mortality. These strategies will be essential for mitigating the impact of the disease in the short term.

Keywords: Lockdown, Social Distancing, Stability Analysis, Bifurcation Analysis, Numerical Simulation

1. Introduction

Mathematical modelling has been extensively used to describe a varied field of phenomenon. Epidemiological models have been applied in describing the course of diseases. Governments have devised several strategies and imposed regulations and restrictions to decelerate the spread, control the cost of human lives and reduce the load on the health care industry (Prabakaran et al., 2021). Several models have been developed in the study of respiratory infectious diseases, however less emphasis has been directed on the impact of lockdown policies and how to optimally apply it. Lockdowns have been effective in not only enforcing social distancing but also in providing a window for other public health measures to be formulated (Pasayat, Pati & Maharana, 2020). In the study by Ferguson et al., (2020), it concluded that though vaccines could not be developed at the onset of an epidemic, the shelter-in place strategy would still be worthy to be applied. The 21 – day lockdown was found to be effective in significantly reducing the number of deaths where the symptomatic

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population in India was high (Singley & Callender, 2020). Ahmed et al, (2020) developed a model on the analysis of the impact of quarantine and other social restriction measures and concluded that the scheme would be effective in minimizing transmission in asymptomatic cases. It was found that the later Wuhan takes the lockdown measures, the lower the peak value of new infections and the smaller the final scale. Moreover, although the late implementation of lockdown measures will reduce the scale of the epidemic in this city, due to the fact that Wuhan's export disease will increase with the delay of time, it will have an uncontrollable impact on other provinces in China and even the world (Tang et al., 2020). Tian et al, (2020) also concluded the effects of control strategies on COVID-19 transmission in Wuhan during the first 50 days from December 31, 2019, to February 19, 2020 that lockdown measure makes the people outside Wuhan to cope with the COVID-19 ahead of time (Tang et al., 2020, Tyson et al., 2020).

In controlling an epidemic, behavioral modifications such as social distancing, home confinements, and consistent use of personal protective measures have proven effective in curbing the spread of infections (Wangari et al., 2021). Social distancing involves altering individual behaviors to reduce contact rates between infected and uninfected persons, thereby preventing disease transmission (Sardar et al., 2020). The study by Bouchnita, Chekroun & Jebrane (2021) investigated the effect of social distancing combined with contact tracing and concluded that these strategies slow down infections and ultimately contain the spread of coronavirus disease 2019 (COVID-19). Additionally, findings by Muto et al., (2020) recommended behavioral changes among the Japanese population to control the disease spread. Mathematical models incorporating social control measures have been crucial in managing the transmission of infectious diseases (Rachah 2022). This analysis demonstrates that population-level behavioral actions are effective non-medical interventions for altering the course of viral-driven catastrophes (Sun et al., 2020). Reluga (2020) developed a mathematical model to assess the impact of vaccination alongside social distancing and concluded that while vaccination is effective, it should be combined with social distancing. Sardar et al., (2020) examined the importance of social distancing, concluding it is most beneficial when the reproduction number is below 2. Bouchnita, Chekroun & Jebrane (2021) showed that adopting relaxed social distancing measures reduces the number of infected cases but does not shorten the duration of epidemic waves.

During an epidemic, the availability of vaccines and acquired immunity among recovered individuals often leads to a relaxation of lockdown and social distancing measures. However, due to challenges such as vaccination rates and vaccine distribution, discarding these methods entirely is not feasible. Accurate predictions of various factors, such as the pandemic's end date, duration of lockdowns, and trends in transmission, are crucial for guiding pandemic response and precautionary measures. This study aims to analyze the effectiveness of social distancing and lockdown, alongside isolation, as the primary strategies employed to mitigate an epidemic.

2. Model Formulation And Development

The total population, N, is divided into six distinct groups: susceptible (S), exposed (E), asymptomatic (A), symptomatic (I), isolated (J), and recovered (R). The susceptible population increases through births at a rate Λ. The rate of transition from susceptible to exposed due to infection is λ. To mitigate the infection rate, a lockdown measure is imposed at a rate p, where $p \in (0,1)$. If lockdown is fully implemented, $p \approx 0$, and if there is no lockdown, $p \approx 1$. In this context, lockdown refers to the restriction of movement across administrative boundaries. Additionally, social distancing is practiced at a rate s, with effective social distancing corresponding to $s \approx 1$, and no social distancing corresponding to $s \approx 0$. Social distancing here means maintaining a safe distance during interactions.

After a latency period ω, a fraction η of the exposed individuals become asymptomatic, while the rest become symptomatic. Asymptomatic individuals recover at a rate ρA, while

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symptomatic individuals are isolated through hospitalization or home-based care at a rate γ and recover at a rate ρJ. Asymptomatic individuals are considered highly infectious since they do not take any precautions. The symptomatic, exposed and isolated individuals have lower infectivity rates denoted by ϵ1, ϵ2 and ϵ3 respectively. The effective contact rate is β and a constant death rate μ is assumed across all compartments.

The model flow chart is represented in figure 1

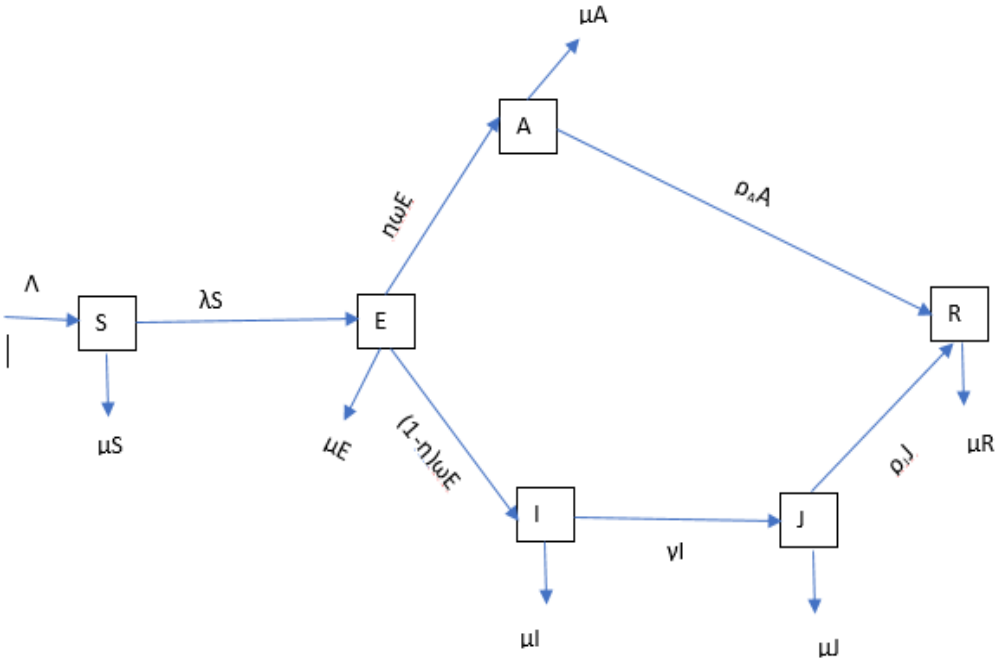

Figure 1; Model Flow Chart

The model equations are represented in equation :

$$\frac{dS}{dt} = \Lambda - (\lambda + \mu)S$$

$$\frac{dE}{dt} = \lambda S - (\mu + \omega)E$$

$$\frac{dA}{dt} = \eta\omega E - (\mu + \rho_A)A$$

$$\frac{dI}{dt} = (1-\eta)\omega E - (\mu + \gamma)I$$

$$\frac{dJ}{dt} = \gamma I - (\mu + \rho_J)J$$

$$\frac{dR}{dt} = \rho_J J + \rho_A A - \mu R$$

The force of infection, λ is given by equation ::

$$\lambda = \beta p(1-s)(A + \epsilon_1 I + \epsilon_2 E + \epsilon_3 J)$$

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The model parameters and the parameter values are shown in the table 1 below:

Table 1: Parameter Values

Symbol	Parameter	Value	Source
Λ	Recruitment rate by birth	0.00018 days^{-1}	Mwalili et al.,(2020)
μ	Natural death rate	4.563 $\times 10^{-5}$ days^{-1}	Mwalili et al.,(2020)
ρ_A	Rate of recovery of asymptomatic patients	3.277 $\times 10^{-1}$ days^{-1}	Al-Harbi & Al-Tuwairqi (2022)
ρ_J	Rate of recovery of isolated patients	1/14 days^{-1}	Tyson et al., (2020)
ω	Latency period	1/6 days^{-1}	Alsofayan et al., (2020)
γ	rate of transfer of I to J	0.025 days^{-1}	Ferguson et al., (2020)
β	Effective contact rate	0.5 days^{-1}	Li et al., (2020)
$\epsilon_{1,(2),(3)}$	Infections by the Symptomatics,(Exposed),(Isolated)	0.48	Wangari et al., (2021)
η	Fraction of those Asymptomatic	0.7	Mwalili et al., (2021)
p	Lockdown effectiveness	0.5	Estimated
s	Social Distancing Effectiveness	0.5	Estimated

3. Model Analysis

3.1. *Positivity of the Solution*

Since the model system (1) pertains to living organisms, the associated state variables are non-negative for all time t > 0. Therefore, the solutions to model (1) with initial data remain positive for all time t > 0.

Theorem 3.1 *The region* $\mathcal{D} = \{(S(t), E(t), A(t), I(t), J(t), R(t)) \in \mathfrak{R}_+^6 : N(t) \leq \frac{\Lambda}{\mu}\}$

is positively invariant and attracting with respect to model 1.

Proof;

Solving the first equation of 1 for S(t) at time, t > 0, it is obtained that;

$$\frac{dS}{dt} = \Lambda - (\lambda + \mu)S$$

$$\frac{dS}{dt} \geq -(\lambda + \mu)S$$

$$\int \frac{dS}{S} \geq -\int (\lambda + \mu)dt$$

$$\int_{S(0)}^{S} \frac{dS}{S} \geq -\int (\lambda + \mu)dt$$

$$InS - InS(0) \geq -\int (\lambda + \mu)dt$$

$$In\frac{S}{S(0)} \geq -\int (\lambda + \mu)dt$$

$$\frac{S}{S(0)} \geq e^{-\int(\lambda+\mu)dt}$$

$$S \geq S(0)e^{-\int(\lambda+\mu)dt}$$

Clearly, $S(0)e^{-\int(\lambda+\mu)dt}$ is a function that remains non-negative with respect to time, ensuring that S(t) remains positive. This process can be applied to discuss the positivity of other state variables using the corresponding system equations. Thus, the solutions of the system (1) with non-negative initial conditions such that E(t) > 0, E(t) > 0, A(t) > 0, I(t) > 0, J(t) > 0, R(t) > 0 will remain for all time $t \geq 0$.

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3.2. *Invariant Region*

Theorem 3.2; *There is a domain $\mathcal{H}$ in which the solution set {S(t), E(t), A(t), I(t), J(t), R(t)} of model equation (1) remains positively invariant.*

Proof;

The total human population can be determined by, N(t) = S(t) + E(t) + A(t) + I(t) + J(t) + R(t).

Then the time derivatives of N(t) along the solutions of model system (1) gives the following:

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

In the absence of the disease, in the population,

$$\frac{dN}{dt} \leq \Lambda - \mu N \Longrightarrow N(t) = \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right) e^{-\mu t}$$

N (0) = S (0) + E (0) + A (0) + S (0) + J (0) + R (0)

Thus if, $\mathrm{N}(0) \leq \frac{\Lambda}{\mu}$, then, $\mathrm{N}(t) \leq \frac{\Lambda}{\mu}$ as $t \to \infty$.

Therefore, $\mathcal{H} = \{(\mathrm{S}(t), \mathrm{E}(t), \mathrm{A}(t), \mathrm{I}(t), \mathrm{J}(t), \mathrm{R}(t)) \in \Re_{+}^{6} : \mathrm{N}(t) \leq \frac{\Lambda}{\mu}\}$ is the feasible solution of model equation (1) which implies the total number of human populations is positively invariant. Therefore, the model is biologically meaningful and mathematically well posed in the region $\mathcal{H}$.

3.3. *Basic Reproduction Number*

The basic reproduction number $\mathcal{R}_0$ measures the number of new infections caused by an initial patient in a fully susceptible population.

To determine $\mathcal{R}_0$, the Next Generation Matrix (NGM) is used. This involves the Jacobian matrix, which is obtained from the model's equations and is crucial for calculating the reproduction number.

Theorem 3.3 *The basic reproduction number $\mathcal{R}_0$ for the epidemiological model 1 is given by equation 2:*

$$\mathcal{R}_0 = \frac{m\beta_0(1-\eta)\omega\epsilon_1}{k_1 k_3} + \frac{m\beta_0\omega\eta}{k_1 k_2} + \frac{m\beta_0\gamma\epsilon_3}{k_1 k_3 k_4} + \frac{m\beta_0\epsilon_2}{k_1}$$

where: $m = \frac{\Lambda}{\mu}$, *and* $k_1 = (\mu + \omega)$, $k_2 = (\mu + \rho_A)$, $k_3 = (\mu + \gamma)$, $k_4 = (\mu + \rho_J)$, $\beta_0 = \beta\, p\, s$

Proof;

The basic reproduction number can be defined as the spectral radius of the matrix product FV^{-1}. To calculate this, we extract the infectious subsystem from the model system (1) and derive the transmission matrix (F) and the transition matrix (V), as detailed in matrices 1 and 2 .

$$F = \begin{pmatrix} \beta_0\epsilon_2 & \beta_0 & \beta_0\epsilon_1 & \beta_0\epsilon_3 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

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And

$$V = \begin{pmatrix} -k_1 & 0 & 0 & 0 \\ \eta\omega & -k_2 & 0 & 0 \\ (1-\eta)\omega & 0 & -k_3 & 0 \\ 0 & 0 & \gamma & -k_4 \end{pmatrix}$$

And

$$FV^{-1} = \begin{pmatrix} a & -\frac{\beta_0}{k_2} & -\frac{\beta_0\epsilon_1}{k_3} - \frac{\gamma\beta_0\epsilon_3}{k_3k_4} & -\frac{\beta_0\epsilon_3}{k_4} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

Where, $a = -\frac{\eta\omega\beta_0}{k_1k_2} + \frac{(-\omega k_2k_4+\eta\omega k_2k_4)\beta_0\epsilon_1}{k_1k_2k_3k_4} + \frac{\beta_0\epsilon_2}{k_1} + \frac{(-\gamma\omega k_2+\gamma\eta\omega k_2)\beta_0\epsilon_3}{k_1k_2k_3k_4}$

Thus, the basic reproduction equation is given in equation 4;

$$\mathcal{R}_0 = \frac{m\beta_0(1-\eta)\omega\epsilon_1}{k_1k_3} + \frac{m\beta_0\omega\eta}{k_1k_2} + \frac{m\beta_0\gamma\epsilon_3}{k_1k_3k_4} + \frac{m\beta_0\epsilon_2}{k_1} \qquad 4$$

3.4. Equilibrium Analysis

3.4.1. Disease Free Equilibrium Point;

The Disease-Free Equilibrium (DFE) for system 1 is achieved when all infection-related classes are set to zero, resulting in the formulation of equation 5.

$$\mathcal{E}_0 = (S^0, E^0, A^0, I^0, J^0, R^0)$$

$$\mathcal{E}_0 = \left(\frac{\Lambda}{\mu}, 0,0,0,0,0\right) \qquad 5$$

3.4.2. Endemic Equilibrium Point;

The endemic equilibrium is the stable condition of the system 1 when the infection is present, meaning that$\lambda \neq 0$. Let's use M^{**} to represent any arbitrary endemic equilibrium within the model system 1.

$$M^{**} = (S^{**}, E^{**}, A^{**}, I^{**}, J^{**}, R^{**})$$

Furthermore,

$$\lambda^{**} = \beta p(1-s)(\epsilon_2E^{**} + A^{**} + \epsilon_1I^{**} + \epsilon_3J^{**})$$

By setting the left-hand side of equation 1 to 0 we get equation :

$$\left(\frac{\Lambda}{\lambda^{**}+\mu}, \frac{\lambda^{**}S^{**}}{\omega+\mu}, \frac{\eta\omega E^{**}}{\mu+\rho_A}, \frac{(1-\eta)\omega E^{**}}{\mu+\gamma}, \frac{\gamma I^{**}}{\mu+\rho_J}, \frac{\rho_A A^{**}+\rho_J J^{**}}{\mu}\right)$$

3.5. Local Stability Analysis

Local stability analysis would be conducted using the determinant-trace approach as outlined by (Connelly, 2023).

Theorem 3.5 *Assume the first order partial derivatives of f and g are continuous in some open set containing the equilibrium point* $(\hat{x}, \hat{y})$. *Then, the equilibrium is locally asymptotically stable if:*

(a). Tr (J) > 0, and
(b). det (J) > 0

where, J is the Jacobian matrix evaluated at the equilibrium. In addition, the equilibrium is unstable if either Tr(J) > 0 or Det(J) < 0.

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3.5.1. Local Stability Analysis of the Disease-Free Equilibrium Point

Computing the Jacobian matrix of equation 1 at DFE, it yields the matrix 1 below:

$$\mathcal{J}_f = \begin{pmatrix} -k_1 & -\beta_0\epsilon_2 & -\beta_0 & -\beta_0\epsilon_1 & -\beta_0\epsilon_3 & 0 \\ 0 & \beta_0\epsilon_2 - (\omega+\mu) & \beta_0 & \beta_0\epsilon_1 & \beta_0\epsilon_3 & 0 \\ 0 & \eta\omega & -(\mu+\rho_A) & 0 & 0 & 0 \\ 0 & (1-\eta)\omega & 0 & -(\mu+\gamma) & 0 & 0 \\ 0 & 0 & 0 & -(\gamma) & -(\mu+\rho_2) & 0 \\ 0 & 0 & \rho_A & 0 & \rho_2 & -\mu \end{pmatrix}$$

Where: $k_1 = -(\mu), k_2 = -(\mu+\omega), k_3 = -(\mu + rho_A), k_4 = -(\mu+\gamma), k_5 = -(\mu + \rho_J), k_6 = -(\mu), m = \frac{\Lambda}{\mu},$

Based on matrix 12 and numerical parameter substitution, it is concluded that:

(a). Tr $(\mathcal{J}_{\not f})$ < 0, and
(b). Det $(\mathcal{J}_{\not f}) < 0$

Therefore, the Disease-Free Equilibrium (DFE) is locally asymptotically unstable. This suggests that under the current model conditions, the disease will continue to persist in the community.

3.5.2. Local Stability Analysis of the Endemic Equilibrium Point

Computing the Jacobian matrix of model equation 1 at the endemic equilibrium point yields.

$$\mathcal{J}_f = \begin{pmatrix} -\mu-\lambda & -m_{\beta,0}\epsilon_2 & -m_{\beta,0} & -m_{\beta,0}\epsilon_1 & -m_{\beta,0}\epsilon_3 & 0 \\ \lambda & m_{\beta,0}\epsilon_2 - (\omega+\mu) & m_{\beta,0} & m_{\beta,0}\epsilon_1 & m_{\beta,0}\epsilon_3 & 0 \\ 0 & \eta\omega & -(\mu+\rho_A) & 0 & 0 & 0 \\ 0 & (1-\eta)\omega & 0 & -(\mu+\gamma) & 0 & 0 \\ 0 & 0 & 0 & -\gamma & -(\mu+\rho_2) & 0 \\ 0 & 0 & \rho_A & 0 & \rho_2 & -\mu \end{pmatrix}$$

Based on matrix 5 and numerical parameter substitution, it is concluded that:

(a). Tr $(\mathcal{J}_{\not f})$ < 0, and
(b). Det $(\mathcal{J}_{\not f}) > 0$

Therefore, the Endemic Equilibrium (EE) is locally asymptotically stable. This suggests that under the current model conditions, the disease will continue to persist in the community.

3.6. Global Stability Analysis

We employ the Lyapunov-Krasovskii method for analyzing the global asymptotic stability.

Theorem 3.6: *Consider the autonomous system defined by* $\hat{x} = f(x)$*, with the equilibrium point of interest being the origin. Let A(x) denote the Jacobian matrix of the system,* $A(x) = \frac{\partial f}{\partial x}$*. If the matrix* $F = A + A^T$ *is negative neighborhood* Ω*, then, the equilibrium point at the origin is asymptotically stable. A Lyapunov function for this system is*

$$V(x) = f^T(x)f(x)$$

If Ω *is the entire state space and, in addition,* $V(x) \to \infty$*,* $[||x||] \to \infty$*, then, the equilibrium point is said to be globally asymptotically stable.*

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3.6.1. Global Stability Analysis of the Disease-Free Equilibrium Point

The global stability analysis is performed by constructing the Jacobian matrix of the model system (1) and solving it at the DFE as shown in the matrix 6:

$$\mathcal{J}_f = \begin{pmatrix} -k_1 & -\beta_0\epsilon_2 & -\beta_0 & -\beta_0\epsilon_1 & -\beta_0\epsilon_3 & 0 \\ 0 & \beta_0\epsilon_2 - (\omega+\mu) & \beta_0 & \beta_0\epsilon_1 & \beta_0\epsilon_3 & 0 \\ 0 & \eta\omega & -(\mu+\rho_A) & 0 & 0 & 0 \\ 0 & (1-\eta)\omega & 0 & -(\mu+\gamma) & 0 & 0 \\ 0 & 0 & 0 & -(\gamma) & -(\mu+\rho_2) & 0 \\ 0 & 0 & \rho_A & 0 & \rho_2 & -\mu \end{pmatrix}$$

Where: $k_1 = -(\mu), k_2 = -(\mu+\omega), k_3 = -(\mu + rho_A), k_4 = -(\mu+\gamma), k_5 = -(\mu+\rho_J), k_6 = -(\mu), m = \frac{\Lambda}{\mu}$

From the matrix 6 above, the transpose $\mathrm{F^T(x)}$ of F(x) is as shown in matrix 7 below:

$$F^T(x) = \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & 0 \\ -\beta_0\epsilon_2 & -\mu-\omega & \eta\omega(1-\eta)\omega & 0 & 0 & \\ -\beta_0 & \beta_0\epsilon_2 & -\mu-\rho_A & 0 & \rho_A & \\ -\beta_0\epsilon_1 & \beta_0\epsilon_1 & 0 & -\mu-\gamma & -\gamma & \\ -\beta_0\epsilon_3 & \beta_0\epsilon_3 & 0 & 0 & -\mu-\rho_2 & \rho_2 \\ 0 & 0 & 0 & 0 & 0 & -\mu \end{pmatrix}$$

From the matrix 7 above, $\mathrm{\hat{F}(x)}$ is as shown in matrix 8 below:

$$\hat{F}(x) = F^T(x) + F(x)$$

This implies that $\mathrm{\hat{F}(x)}$ is as in matrix 8 below:

$$\hat{F}(x) = \begin{pmatrix} -2\mu & 0 & 0 & 0 & 0 & 0 \\ -\beta_0\epsilon_2 & -2\mu-2\omega+2\beta_0\epsilon_2 & \eta\omega+\beta_0 & (1-\eta)\omega(1-\eta)\omega+\beta_0\epsilon_1 & \beta_0\epsilon_3 & 0 \\ -\beta_0 & \beta_0\epsilon_2 & -2\mu-2\rho_A & 0 & 0 & \rho_A \\ -\beta_0\epsilon_1 & \beta_0\epsilon_1 & 0 & -2\gamma-2\mu & -\gamma & 0 \\ -\beta_0\epsilon_3 & \beta_0\epsilon_3 & 0 & 0 & -2\mu-2\rho_2 & \rho_2 \\ 0 & 0 & \rho_A & 0 & \rho_2 & -2\mu \end{pmatrix}$$

The matrix $\mathrm{\hat{F}(x)}$ is to be checked if it is negative definite. This is by getting the principal determinants of the matrix $\hat{F}(x)$.

(a). $\mathrm{2k_1}$ is negative definite, and
(b). Det ($\hat{F}(x)$) is not negative definite

So, the matrix $\hat{F}(x)$ is not negative definite. This, is not a Lyapunov function for the system F(x), and, thus,

$$V(x) \neq f^T(x)f(x)$$

If Ω is the entire state space and, in addition, $V(x) \to \infty$, $[||x||] \to \infty$, then, the equilibrium point is said to be globally asymptotically unstable.

3.6.2. Global Stability Analysis of the Endemic Equilibrium Point

The global stability analysis is performed by constructing the Jacobian matrix of the model system (1) and solving it at the EE as shown in matrix 9

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$$\mathcal{J}_f = \begin{pmatrix} -\mu-\lambda & -m_{\beta,0}\epsilon_2 & -m_{\beta,0} & -m_{\beta,0}\epsilon_1 & -m_{\beta,0}\epsilon_3 & 0 \\ \lambda & m_{\beta,0}\epsilon_2-(\omega+\mu) & m_{\beta,0} & m_{\beta,0}\epsilon_1 & m_{\beta,0}\epsilon_3 & 0 \\ 0 & \eta\omega & -(\mu+\rho_A) & 0 & 0 & 0 \\ 0 & (1-\eta)\omega & 0 & -(\mu+\gamma) & 0 & 0 \\ 0 & 0 & 0 & -\gamma & -(\mu+\rho_2) & 0 \\ 0 & 0 & \rho_A & 0 & \rho_2 & -\mu \end{pmatrix}$$

From the matrix 9 above, the transpose $\mathrm{F^T(x)}$ of F(x) is as shown in matrix 10 below:

$$F^T(x) = \begin{pmatrix} -\lambda-\mu & \lambda & 0 & 0 & 0 & 0 \\ -m_{\beta,0}\epsilon_2 & -\mu-\omega+m_{\beta,0}\epsilon_2 & \eta\omega & (1-\eta)\omega & 0 & 0 \\ -m_{\beta,0} & m_{\beta,0} & -\mu-\rho_A & 0 & 0 & \rho_A \\ -m_{\beta,0}\epsilon_1 & m_{\beta,0}\epsilon_1 & 0 & -\gamma-\mu & -\gamma & 0 \\ -m_{\beta,0}\epsilon_3 & m_{\beta,0}\epsilon_3 & 0 & 0 & -\mu-\rho_2 & \rho_2 \\ 0 & 0 & 0 & 0 & 0 & -\mu \end{pmatrix}$$

From the matrix 10 above, $\hat{F}(x)$ is as shown in matrix 11 below:

$$\hat{F}(x) = F^T(x) + F(x)$$

This implies that $\hat{F}(x)$ is as in matrix 11 below:

$$\hat{F}(x) =$$

$$\begin{pmatrix} -2\lambda-2\mu & \lambda-m_{\beta,0}\epsilon_2 & -m_{\beta,0} & -m_{\beta,0}\epsilon_1 & -m_{\beta,0}\epsilon_3 & 0 \\ \lambda-m_{\beta,0}\epsilon_2 & -2\mu-2\omega+2m_{\beta,0}\epsilon_2 & \eta\omega+m_{\beta,0} & (1-\eta)\omega+m_{\beta,0}\epsilon_1 & m_{\beta,0}\epsilon_3 & 0 \\ -m_{\beta,0} & \eta\omega+m_{\beta,0} & -2\mu-2\rho_A & 0 & 0 & \rho_A \\ -m_{\beta,0}\epsilon_1 & (1-\eta)\omega+m_{\beta,0}\epsilon_1 & 0 & -2\gamma-2\mu & -\gamma & 0 \\ -m_{\beta,0}\epsilon_3 & m_{\beta,0}\epsilon_3 & 0 & -\gamma & -2\mu-2\rho_2 & \rho_2 \\ 0 & 0 & \rho_A & 0 & \rho_2 & -2\mu \end{pmatrix}$$

The matrix $\hat{F}(x)$ is to be checked if it is negative definite. This is by getting the principal determinants of the matrix $\hat{F}(x)$.

(a). $2k_1$ is negative definite, and
(b). Det ($\hat{F}(x)$) is negative definite

So, the matrix $\hat{F}(x)$ is negative definite. This, is a Lyapunov function for the system F(x), and, thus,

$$V(x) = f^T(x)f(x)$$

If Ω is the entire state space and, in addition, $V(x) \to \infty$, $\left[||x||\right] \to \infty$, then, the equilibrium point is said to be globally asymptotically stable.

3.7. Bifurcation Analysis

Bifurcation analysis is critical in identifying parameter values or thresholds where qualitative changes in the system's behavior occur. The changes can signify transitions between disease free states, endemic states and even periodic outbreaks. These thresholds are crucial for predicting and controlling disease dynamics.

Theorem 3.7 of (Castillo-Chavez & Song, 2004); Consider the following general system of ordinary differential equations with a parameter ϕ.

$$\frac{dx}{dt} = f(x,0), f: R^n \times R \to R \; and \; f \in \backslash mathdsC^2(R^n \times R)$$

Where 0 is an equilibrium point of the system (that is, $f(0,\phi) = 0, \forall$) and assume

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(1). $A = D_x f(0,0) = \left[\frac{\partial f_i}{\partial x_j}(0,0)\right]$ is the linearization matrix of 6 around the equilibrium point 0 with ϕ evaluated at 0, zero is a simple eigenvalue of A and other eigenvalues of A have negative real parts:

(2). Matrix A has a right eigenvector w and left eigenvector v (each corresponding to zero eigenvalues)

Let *fk* be the k-th component of f and:

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \theta}(0,0)$$

The local dynamics of the model system 1 around 0 is totally determined by the signs of a and b.

(a). a>0, b>0, when $\phi < 0$ with $|\phi| \ll 1$, 0 is locally asymptotically stable and there exists a positive unstable equilibrium; when $0 < \phi \ll 1$, 0 is unstable and there exist a negative and locally asymptotically stable equilibrium.

(b). a<0, b<0. When $\phi < 0$ with $|\phi| \ll 1$, 0 is locally asymptotically stable and there exists a positive unstable equilibrium; when $0 < \phi \ll 1$, 0 is unstable and there exist a positive unstable equilibrium.

(c). a>0, b<0, when $\phi < 0$ with $|\phi| \ll 1$, 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi \ll 1$, 0 is stable, and a positive unstable equilibrium appears.

(d). a<0, b>0. When ϕ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.

Let the model system be written in the vector form:

$$\frac{dX}{dt} = G(X)$$

Where;

$$X = (x_1, x_2, x_3, x_4, x_5, x_6)^T \quad \text{and} \quad G = (g_1, g_2, g_3, g_4, g_5, g_6)$$

so that;

$$S = x_1, E = x_2, A = x_3, I = x_4, J = x_5, R = x_6$$

Then the model system becomes:

$$\frac{dx_1}{dt} = \Lambda - (\lambda + \mu)x_1$$

$$\frac{dx_2}{dt} = \lambda x_1 - (\mu + \omega)x_2$$

$$\frac{dx_3}{dt} = \eta\omega x_2 - (\mu + \rho_A)x_3$$

$$\frac{dx_4}{dt} = (1 - \eta)\omega x_2 - (\mu + \gamma)x_4$$

$$\frac{dx_5}{dt} = \gamma x_4 - (\mu + \rho_J)x_5$$

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$$\frac{dx_6}{dt} = \rho_J x_5 + \rho_A x_3 - \mu x_6$$

Let β be the bifurcation parameter, then $R_0 = 1$ in equation (4) and solving for β yields:

$$\beta = \hat{\beta} = \frac{k_1 k_2 k_3 k_4}{m\beta_0 k_3 k_4(\eta\omega + k_2\epsilon_2) - (1-\eta)\omega k_2(k_4\epsilon_1 + \gamma\epsilon_3)}$$

So that the disease-free equilibrium, E_0, is locally stable when $\beta < \hat{\beta}$, and is unstable when $\beta > \hat{\beta}$. Therefore, $\hat{\beta}$ is the bifurcation value.

The linearized matrix of system (1) around the disease-free equilibrium, E_0, and evaluated at $\hat{\beta}$ is given by:

$$\mathcal{J}(E_0|\hat{\beta}) = \begin{pmatrix} k_1 & a_1 & a_2 & a_3 & a_4 & 0 \\ 0 & k_2 + a_5 & 0 & a_6 & a_7 & a_8 \\ 0 & a_9 & k_3 & 0 & 0 & 0 \\ 0 & a_{10} & 0 & k_4 & 0 & 0 \\ 0 & 0 & 0 & \gamma & k_5 & 0 \\ 0 & 0 & \rho_A & 0 & \rho_J & k_6 \end{pmatrix}$$

Where; $k_1 = -\mu, k_2 = -(\omega + \mu), k_3 = -(\mu + \rho_A), K_4 = -(\mu + \gamma), k_5 = -(\mu + \rho_J), k_6 = -\mu, a_1 = -m\beta_0\epsilon_2, a_2 = -m\beta_0, a_3 = -m\beta_0\epsilon_1, a_4 = -m\beta_0\epsilon_3, a_5 = -m\beta_0\epsilon_2 + k_2, a_6 = -m\beta_0, a_7 = -m\beta_0\epsilon_1, a_8 = -m\beta_0\epsilon_3, a_9 = \eta\omega, a_{10} = (1-\eta)$

The eigenvalues are six which are real and negative except zero eigenvalues, which can be obtained by Wolfram Mathematica software as $k_1, k_2, k_3, k_4, k_5, k_6$. The zero is a simple eigen value of the Jacobian matrix $\mathcal{J}(E_0|\hat{\beta})$ and the eigen values are equal and negative. Therefore, the DFE, E_0 is a non-hyperbolic equilibrium, which is in line with the assumption in theorem 3.7 of Castillo-Chavez & Song, (2004).

As a result, the center manifold theory can be applied to determine the local stability of DFE point, E_0. The right eigen vector, $m = (m_1, m_2, m_3, m_4, m_5, m_6)^T$ and left eigenvector, $v = (v_1, v_2, v_3, v_4, v_5, v_6)$ associated with this simple zero eigen eigenvalues of the matrix $\mathcal{J}(E_0|\hat{\beta})$ such that v.m = 1, can be obtained by multiplying vJ and mJ and setting each of them equal to zero.

The resulting system of the right eigenvalues become;

$$m_1 k_1 + m_2 a_1 + m_3 a_3 + m_4 a_4 = 0$$
$$m_2 a_5 + m_3 a_6 + m_4 a_7 + m_5 a_8 = 0$$
$$m_2 a_9 + m_3 k_3 = 0$$
$$m_2 a_{10} + m_4 k_4 = 0$$
$$m_4 \gamma + m_5 k_5 = 0$$
$$m_3 \rho_A + m_5 \rho_J + m_6 k_6 = 0$$

From the equation 14 above,

$$let, m_2 = m_2 > 0, then;$$
$$m_3 = \frac{-m_2 a_9}{k_3}$$
$$m_4 = \frac{-k_2 m_2}{a_{10}}$$

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$$m_5 = \frac{-\gamma k_2 m_2}{a_{10} k_5}$$

$$m_6 = \frac{-1}{k_6}\left(m_3 \rho_A + m_5 \rho_J\right)$$

$$m_1 = \frac{-1}{k_1}(m_2 a_1 + m_3 a_3 + m_4 a_4)$$

Also, the left eigenvector $\mathrm{v} = (\mathrm{v}_1, \mathrm{v}_2, \mathrm{v}_3, \mathrm{v}_4, \mathrm{v}_5, \mathrm{v}_6)$ corresponding to the zero eigenvalue is obtained from $\mathcal{J}\left(\mathrm{E}_0 \middle| \hat{\beta}\right) = 0$ which yields;

$$v_1 k_1 = o$$

$$v_1 a_1 + v_2 a_5 + v_3 a_9 + v_4 a_{10} = 0$$

$$v_1 a_2 + v_2 a_6 + v_3 k_3 + v_6 \rho_A = 0$$

$$v_1 a_3 + v_2 a_7 + v_4 k_4 + v_5 \gamma = 0$$

$$v_1 a_4 + v_2 a_8 + v_5 k_5 + v_6 \rho_J = 0$$

$$v_6 k_6 = 0$$

From equation 15 above, $\mathrm{v}_1 = \mathrm{v}_6 = 0$; then;

$$v_3 = \frac{-a_6 v_2}{k_3}$$

$$v_5 = \frac{-a_8 v_2}{k_5}$$

$$v_4 = \frac{v_2 a_7 v_5 \gamma}{k_3}$$

To satisfy the condition $(\mathrm{v}.\mathrm{m} = 1)$, we determine the value of (v_2). To compute the bifurcation coefficients a and b as defined in Theorem 3.7, we consider system model 1 in the following form:

$$\frac{dX}{dt} = f = (f_1, f_2, f_3, f_4, f_5, f_6)^T$$

Where,

$$X = (x_1, x_2, x_3, x_4, x_5 x_6)^T$$

The coefficients a and b are derived from the partial derivatives in equations 33 and 35, respectively:

$$a = \sum_{k,i,j=1}^{6} v_k m_i m_j \frac{\partial^2 f_k}{\partial x_i \, \partial x_j}(0,0)$$

And,

$$b = \sum_{k,i=1}^{6} v_k m_i \frac{\partial^2 f_k}{\partial x_i \, \partial \hat{\xi}}(0,0)$$

Since the components of $\mathrm{v}_1, \mathrm{v}_6$ are zero we do not need to find the derivatives off$_1, \mathrm{f}_6$. From the derivative of the remaining $\mathrm{f}_2, \mathrm{f}_3, \mathrm{f}_4, \mathrm{f}_5$, the only ones that have non-zero partial derivatives are considered such that:

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$$\frac{\partial^2 f_2}{\partial x_1 \partial x_2} = \frac{\partial^2 f_2}{\partial x_2 \partial x_1} = \epsilon_2,$$
$$\frac{\partial^2 f_3}{\partial x_1 \partial x_3} = \frac{\partial^2 f_3}{\partial x_3 \partial x_1} = 1$$
$$\frac{\partial^2 f_4}{\partial x_1 \partial x_4} = \frac{\partial^2 f_4}{\partial x_4 \partial x_1} = \epsilon_1,$$
$$\frac{\partial^2 f_5}{\partial x_1 \partial x_5} = \frac{\partial^2 f_5}{\partial x_5 \partial x_1} = \epsilon_3$$

Considering, the study of \cite{27}, the indication of either having a forward or backward bifurcation is determined by a. Consequently, it yields equation 16:

$$a = 2v_1 m_1 m_4 \epsilon_2 + 2v_3 m_1 m_3 + 2v_4 m_1 m_4 + 2v_5 m_1 m_5 > 0 \quad (16)$$

Therefore, a mathematical system consisting of equations as presented in equation 1 exhibits a retrograde bifurcation at $\mathcal{R}_0 = 1$, given that $\beta > \hat{\beta}$ (representing the effective contact rate). This suggests that the complete elimination of the COVID-19 virus cannot be definitively achieved solely by ensuring that $\mathcal{R}_0 < 0$. There exists a potential scenario where a resurgence of the epidemic might occur at a later time. The occurrence of this retrograde bifurcation in the context of COVID-19 has been investigated in studies conducted by (Oluyori et al., (2020); Kassa et al., (2020)).

4. Numerical Analysis

A numerical simulation was conducted in model 1. The model was calibrated with parameter values derived from published research, with some values estimated to provide a comprehensive analysis for this investigation. The parameter values specified in Table 1 were used to conduct numerical simulations in this investigation. The numerical values were simulated over a time period of $0 \leq t \leq 100$ days, covering the entire disease progression. A baseline population of 1000 individuals was utilized to represent the total population. The simulations were executed using PYTHON software, JUPYTER serving as an integrated development environment (IDE), and the results were displayed in graphical format. The graphs clearly show that without interventions the disease would rise the health burden to the community as shown in figure 2 and 3.

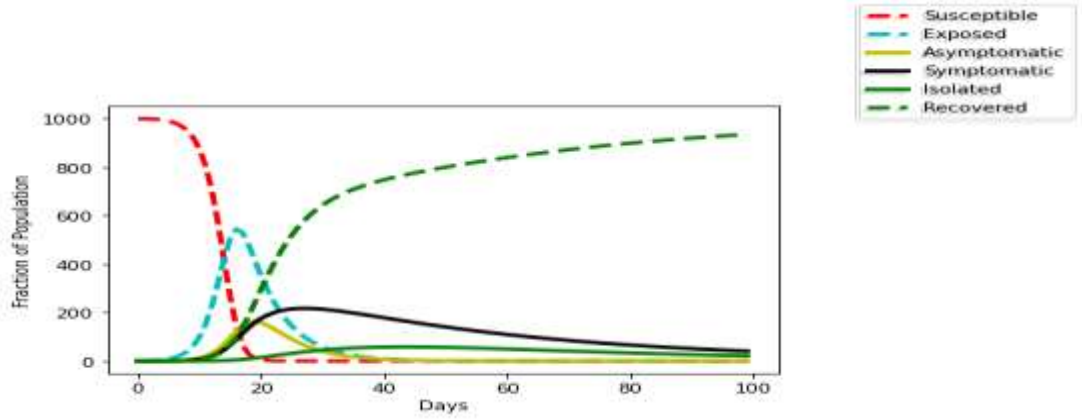

Figure 2: Presence of Interventions

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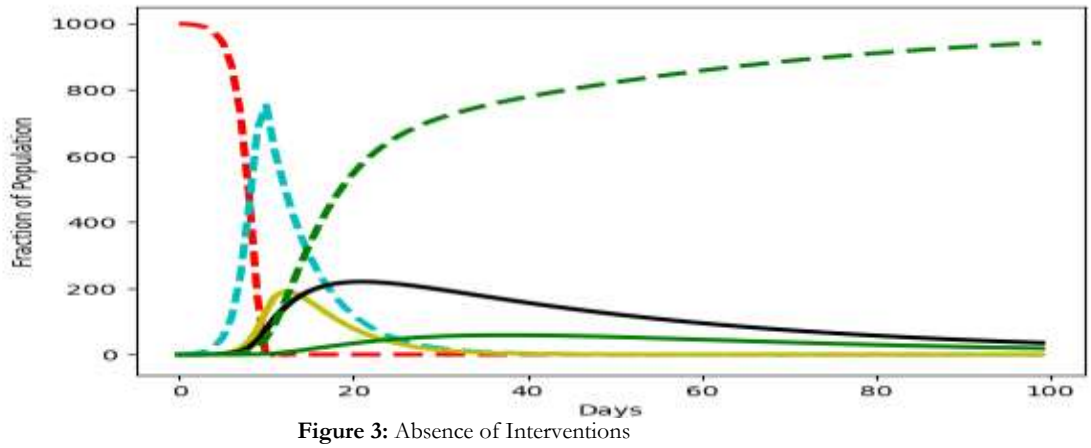

Figure 3: Absence of Interventions

4.1. *Impact of Lockdown*

The examination of the effect of lockdown measures on the prevalence of asymptomatic individuals is conducted. Figure 4 illustrates that strict compliance with the lockdown measures leads to a modification in behavior that reduces the number of infections. Additionally, it prolongs the projected date of highest infection rates, allowing for adequate readiness of medical resources and staff. The lockdown measure p greatly influences disease transmission. Increasing p reduces the transmission rate, resulting in a lower infection peak and a delayed spread. This underscores the critical role of implementing and adhering to lockdown measures to effectively control the spread of infectious diseases.

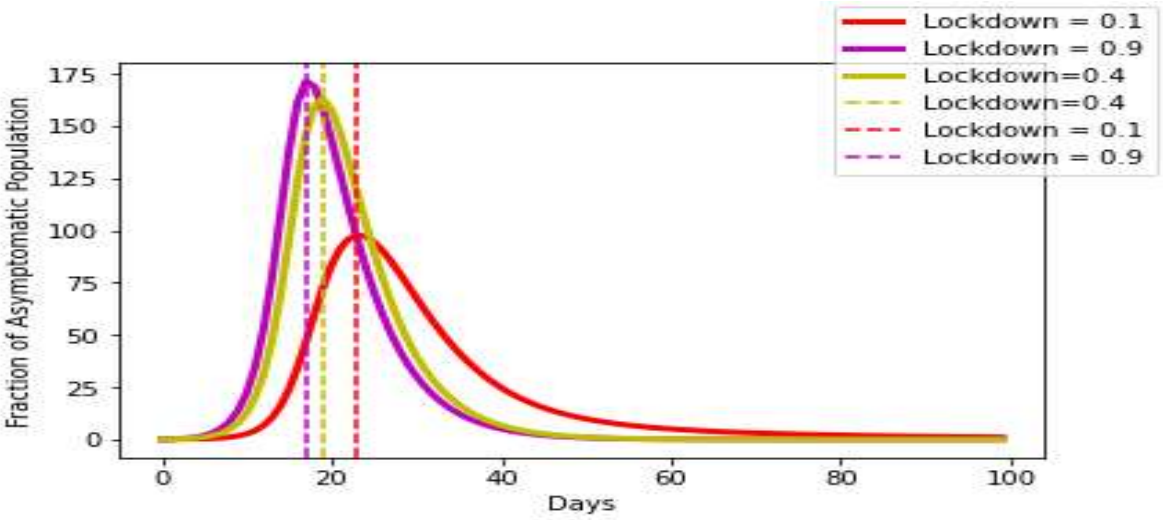

Figure 4: Impact of Lockdown on Asymptomatic

4.2. *Impact of Social Distancing*

The simulation models disease spread using an SEAIR (Susceptible-Exposed-Asymptomatic-Infectious-Recovered) framework, highlighting the significant impact of social distancing, s, on disease transmission. Increasing s, reduces the transmission rate, resulting in a lower peak and delayed spread of infection. This underscores the critical importance of implementing

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and adhering to social distancing measures to effectively control the spread of infectious diseases, as represented in figure 5.

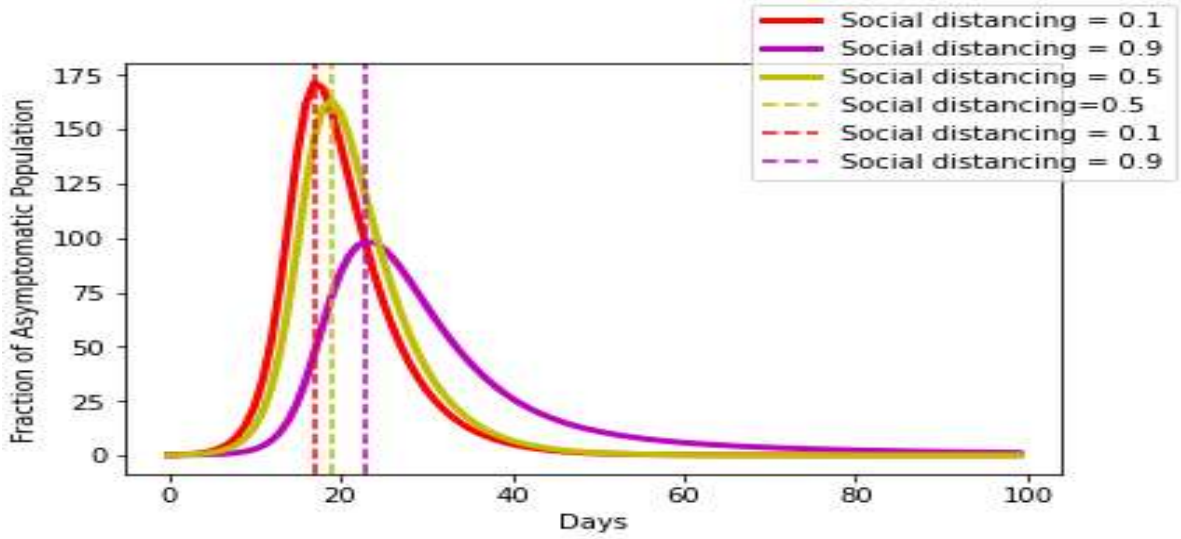

Figure 5: Impact of Social Distancing on Asymptomatic

4.3. Impact of Social Distancing and Lock-down

The graph and the calculated peaks demonstrate the critical role of social distancing in controlling the spread of a disease. Higher social distancing measures (indicated by higher values of s) lead to a slower and more controlled spread, reducing the peak value of the asymptomatic population and delaying its occurrence. This highlights the importance of implementing effective social distancing measures to mitigate the impact of infectious diseases as represented in figure 6.

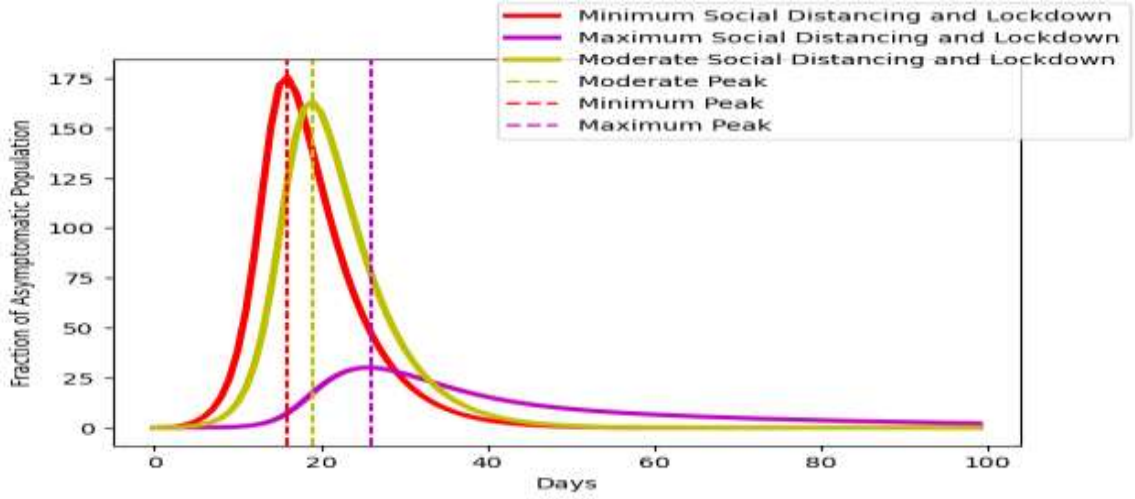

Figure 6: Impact of social distancing and Lockdown effectiveness

5. Conclusion

.Pandemics have emerged throughout history, making it essential to continually find the best strategies to mitigate their effects and minimize transmission. This paper has developed a mathematical model to explore the infection dynamics and control measures of an infectious respiratory disease among humans. A system of ordinary differential equations was formulated, incorporating lockdown, social distancing, and isolation as strategies to be analyzed. By determining the basic reproduction number (R0), the impact of these strategies was assessed. Through numerical substitution of parameters, the progression of infections

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was analyzed under varying rates of strategy implementation. The model equations are dynamic and can be applied to most infectious diseases, adaptable to different circumstances by modifying disease and behavior patterns. While this model does not account for vaccination or other pharmaceutical interventions, it acknowledges their significant roles in reducing healthcare burden and outbreak peak time and duration. Social distancing practices can reduce the severity of an epidemic, but the benefits of social distancing depend on the extent to which it is used by individuals. The benefits of social distancing and lockdowns are that it gives a room of opportunity for the development of vaccines and treatment regimens where not possible or to give time for production and distribution of the necessary doses. Although lockdown and social distancing are effective, they should not be prolonged to avoid population fatigue.

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