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Effectiveness of Non-Pharmaceutical Interventions on COVID-19 Dynamics

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ABSTRACT: Since the outbreak of the Coronavirus disease (COVID-19) in 2020, various modalities have been implemented to help curb the disease as it is still endemic in different countries. Some of such modalities included non-pharmaceutical interventions such as social distancing, hand hygiene, mask wearing amongst others. In this work, we examined the effectiveness of those non-pharmaceutical interventions on the coronavirus disease using a deterministic SEIR model. This consists of investigating the disease-free and endemic equilibria, basic reproduction number and stability. The local stability of the disease-free equilibrium was determined by solving the Jacobian matrix of the system of the system of differential equations while the basic reproduction number was calculated using the next generation matrix method. Numerical simulations to determine the active factor(s) in the transmission, preventive and possible elimination of the disease were carried out using a computational software called Maple. It was revealed that when people comply with these non-pharmaceutical interventions the rate of recovery increases and the spread of the disease is reduced greatly.

KEYWORDS: Coronavirus, Non-Pharmaceutical, Interventions, basic reproduction number, social distancing, Jacobian matrix, pandemic

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I. INTRODUCTION

The Coronavirus, also known as COVID-19, which is caused by a novel virus called severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). It originated in Wuhan, China, with the first cases reported in December 2019 and later spreading to other countries. The World Health Organization (WHO, 2021) declared it a global health emergency in January 2020 and a pandemic in march 2020 (Cennimo, 2023). The virus is believed to have originated from a seafood and wet animal market where the first victims contracted the disease.

COVID-19 primarily spreads between people in close contact, such as conversational distance, through small liquid particles released from an infected person's mouth or nose when they cough, sneeze, speak, sing or breathe. It can also spread through long-range airborne transmission in poorly ventilated or crowded indoor settings (WHO, 2021). Additionally, people can become infected by touching surfaces or objects contaminated by the virus and then touching their eyes, nose, or mouth. The severity of COVID-19 symptoms varies, ranging from very mild to severe. Some individuals may experience no symptoms at all but can still spread the virus, known as asymptomatic transmission. Others may develop worsened symptoms such as pneumonia or respiratory failure.

As a result of the highly contagious nature of the virus and the rapid global spread some nonpharmaceutical interventions (NPIs) were put in place for Covid-19 to help mitigate the spread of the virus, reduce the burden on healthcare systems and protect public health. Some of such NPIs included social distancing, mask-wearing, hand hygiene and restrictions on large gatherings were implemented to reduce person-to-person transmission on the virus. Several researchers have also worked on the impact of some of these non-pharmaceutical interventions for Covid-19 using various numerical techniques. Okuonghae and Omame (2020) worked on the impact of various non-pharmaceutical control measures (government and personal) on the

2024

population dynamics of the novel coronavirus disease 2019 (COVID-19) in Lagos, Nigeria, using an appropriately formulated mathematical model. They used numerical stimulations to show the effect of control measure specifically the common social distancing, use of face mask and case detection (via contact tracing and subsequent testing) on the dynamics of Covid. The numerical simulations of the model showed that if at least 55% of the population comply with the social distancing regulations with about 55% of the population effectively using their face masks while in public, the disease will eventually die out in the population and that if the case detection rate for symptomatic individuals is raised to about 0.8 per day with about 55% of the population complying with the social distancing regulations, it will lead to a great decrease in the incidence and prevalence of Covid-19. Aslan et al., 2020 analyzed the dynamics of local outbreaks of COVID-19 by developing a SEIQR type deterministic model which uses a system of ordinary differential equations. From the data gotten from the outbreak in Hubei they were able to predict the trajectory of daily cases, daily deaths, and other features of the Hubei outbreak. Through numerical experiments they observed the effects of quarantine, social distancing, and COVID-19 testing on the dynamics of the outbreak. Meanwhile, Enahoro et al., (2020) developed a mathematical model to understand the transmission dynamics and control of Covid-19 in Nigeria, one of the epicenters of Covid-19 in Africa. The epidemiological implication of the result showed that the pandemic can be effectively controlled or even eliminated in Nigeria if the control strategies implemented can bring and maintain the epidemiological threshold (R_o) to a value less than unity. It was however shown that Covid-19 can be effectively controlled using social distancing measures provided its effectiveness level is at least moderate. Also, in considering the grave implications of the continuous spread of coronavirus disease, Idisi et al., (2021) formulated a SEIHRD epidemic model which consisted of the Susceptible, Exposed, Infected, Hospitalized, Recovered and Deceased individuals to gain insight into the disease transmission dynamics with impacts of proposing control measures. The model captured the impact of undetected infectious individuals and detected hospitalized individuals with saturated treatment on the spread, death and recovery of Covid-19 patients in Nigeria. Results obtained suggested that decreasing the transmission rate for infective alone is not sufficient to eradicate the disease because of the presence of backward bifurcation, and recommended that Nigerians must also adhere strictly to COVID-19 protocols in mitigating the spread and demise of the coronavirus disease. Onitilo and Daniel (2022) developed a SEAIQR model to examine the transmission mechanism of COVID-19 among humans. The population was distributed into Suceptible, Exposed, Asymptomatic infected, symptomatic Infected, Quarantined and Recovered humans respectively. The existence and stability of disease free equilibrium were established. Results showed that the effectiveness of control measures (reducing contact rate and usage of face mask) when being applied. It is noticed that the best option is to observe social distance against the use of a mask. The effective approach is the compliance with both control measure which are social distancing and usage of mask. It was recommended that there should be educational campaigns on the impact of embracing social distancing, wearing a mask, need to be vaccinated as well as the enforcement and sanctions for non-compliance with the control measures.

In this study, SEIR model that incorporated the Effectiveness of Non-Pharmaceutical Interventions (NPIs) on COVID-19 Dynamics is considered. Despite the effort of previous researchers, studying the effectiveness of NPIs on COVID-19 dynamics is essential for guiding public health responses, optimizing resource allocation, informing policy decisions, managing risks, and complementing vaccination efforts in controlling the spread of the virus.

II. MATHEMATICAL/PROBLEM FORMULATION

In this study, the model was based on the SEIR framework which divides the population into compartments: Susceptible (*S*), Exposed (*E*), Infectious (*I*) and Recovered (*R*). This model explores the impact of various NPIs interventions such as mask wearing, social distancing, hand hygiene and quarantine on the spread of COVID-19. The human population is born/recruited into susceptible population at a rate *b*. The term $\beta SI/N$ describes the rate at which susceptible individuals gets exposed to the disease and consequently infected as a result of not employing any of the interventions that has been put into place.

Assumptions: Taking all the subclasses enumerated above into consideration, we assume the following:

- 1. Recovered population could still become susceptible.
- 2. Exposed persons could become infected after exposure.

3. Infected humans could recover or die due to the disease, every other person in the system could die a natural death.

Mathematically these interactions are described by a system of ordinary differential equations as shown below:

$$\frac{dS}{dt} = b - \frac{\beta SI}{N} - \delta S - \mu S + \tau R \tag{1}$$

$$\frac{dE}{dt} = \frac{\beta SI}{N} - \varphi E - \mu E \tag{2}$$

$$\frac{dI}{dt} = \sigma E - \gamma I - \lambda I - \mu I \tag{3}$$

$$\frac{dR}{dt} = \gamma I + \delta S - \mu R - \tau R \tag{4}$$

Subject to the initial conditions

$$S(0) = 9000, E(0) = 400, J(0) = 50, R(0) = 550$$
(5)

with N(t) = S(t) + E(t) + I(t) + R(t).

Parameters	Biological significance	Values
b	Recruitment rate	0.00018/day
μ	natural death rate	0.0002/day
β	transmission rate without interventions	0.00414
δ	reduction in transmission due to NPIs	0.0115
σ	rate of transition from exposed to infectious class	0.09
γ	recovery rate	0.15
λ	disease-induced death rate	0.0018
τ	rate of transition from recovered to susceptible class	0.075

III. BASIC ANALYSES OF THE MODEL

3.1. Positivity or non-negativity of Solutions

For the model (1) to be epidemiologically meaningful and mathematically well posed, it is necessary to establish that all solutions of system with positive initial data will remain positive for all times t > 0. This will be established in the following theorem.

Theorem 3.1. Positivity of Solution:

Suppose $\Gamma = \{(S, E, I, R) \in R^4 : S(0) > 0, E(0) > 0, I(0) > 0, R(0) > 0\}$, then the solution set $\{S, E, I, R\}$ is positive for all $t \ge 0$.

Proof. Observe that from the (3) equation,

$$\frac{dI(t)}{dt} = \sigma E - \gamma I - \lambda I - \mu I \le -(\gamma + \lambda + \mu)I$$

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That is

$$\frac{dI(t)}{dt} \ge -(\gamma + \lambda + \mu)I$$

Integrating the above,

$$\ln I(t) \ge -(\gamma + \lambda + \mu)t + k$$

Imposing the initial condition gives

$$I(t) \ge I(0)e^{-(\gamma+\lambda+\mu)t} \tag{6}$$

Now from the first equation

$$\frac{dS}{dt} = b - \frac{\beta SI}{N} - \delta S - \mu S + \tau R \ge -\frac{\beta SI}{N} - (\delta + \mu)S$$

That is

$$\frac{dS(t)}{S(t)} \ge -\left(\frac{\beta I(t)}{N} - (\delta + \mu)\right) dt$$

Substituting for I(t) and integrate gives

$$S(t) \ge S(0)e^{\left(\beta J(0)\frac{e^{\left(-(\gamma+\lambda+\mu)t\right)}-1}{N(\gamma+\lambda+\mu)}\right)}e^{-(\mu+\delta)t}$$
(7)

Executing similar procedures for the second and fourth equations, gives

$$E(t) \ge E(0)e^{-(\varphi+\mu)t} \tag{8}$$

$$R(t) \ge R(0)e^{-(\mu+\tau)t} \tag{9}$$

It could be observed from equations (6)-(9) that,

(1)
$$S(t) \ge S(0), E(t) \ge E(0), I(t) \ge I(0), R(t) \ge R(0)$$
 when $t = 0$

(2)
$$\max_{i} \phi(t)_{i} = \phi(0)_{i} \forall i \text{ at } t \ge 0, where i = 1 \cdots 4, \phi = (S, E, I, R)$$

It follows that all solutions of the model are non-negative. This completes the proof.

3.2. Feasible Region for System Solutions

Let us discuss the region in which the total population size exists. It is important to show the region where every solution of the model exists, and all such solutions must be bounded. We shall obtain such bound for the total population size. This is shown in the proof of the theorem below.

Theorem 3.2. *Feasible Region:*

The sets

$$\Gamma 1 = \left\{ (S, E, I, R) \in R_{+}^{4} : 0 \le S + E + I + R = N \le \frac{\alpha}{\delta} \right\}$$
(10)

are feasible solution sets for the model (1)-(4) subject to (5).

Proof. We recall that the total human population size at time *t* is given by

N(t) = S(t) + E(t) + I(t) + R(t).

Differentiating this with respect to time, we obtain

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$$\frac{dN(t)}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = b - \mu S - \mu E - \mu I - \lambda I - \mu R$$
$$= -\mu(S + E + I + R) + b - \lambda I \ge b - \mu N$$
$$\frac{dN(t)}{dt} \ge b - \mu N$$

and solving for N(t) gives

$$N(t) \ge \frac{b}{\mu} + N(0)e^{-\mu t}.$$

$$N(t) \ge \frac{b}{\mu}$$
(11)

As $t \to \infty$, we obtain

Therefore, the threshold human population level is b/μ . It follows that the feasible solution sets of the model remain in the regions: $\Gamma_1 = \{(S, E, I, R) \in R^4_+ : 0 \le S + E + I + R = N \le b/\mu\}$. Observe that if the population is higher than the threshold level, the population reduces to the carrying capacity. If the population is less than the threshold level, then the solutions of the model remain in the invariant region for all t > 0. Therefore, the regions Γ_1 are positively invariant. This completes the proof.

3.3. Equilibrium Point

It is important to obtain the equilibrium points of disease mathematical models. The equilibrium points are very important in decision making pertaining the disease-control and possible elimination. There are two types of equilibrium points that are considered in the mathematical modeling of infectious diseases: the Disease-Free Equilibrium (DFE) point and the Disease-Endemic Equilibrium (DEE) point.

3.3.1. The Disease-Free Equilibrium Point (DFEP)

This represents the average size of each of the compartments when the entire population is free from the infection. It is denoted by \mathbb{E}_0 . We obtain \mathbb{E}_0 by equating the right-hand side of the model (1) to zero and solving the resulting algebraic system of equations. Since we are considering the disease-free equilibrium point, we put I = 0, and which when substituted into the equations gives E = 0. We then have:

$$S = \frac{b(\mu + \tau)}{\mu(\delta + \mu + \tau)}$$
 and $R = \frac{b\delta}{\mu(\delta + \mu + \tau)}$

Therefore,

$$\mathbb{E}_{0} = \left(\frac{b(\mu + \tau)}{\mu(\delta + \mu + \tau)}, 0, 0, \frac{b\delta}{\mu(\delta + \mu + \tau)}\right)$$

3.3.1. The Endemic Equilibrium Point (EEP)

The endemic equilibrium point is the average size of each of the model compartments, when the disease has become part of the human population. The model admits an endemic equilibrium $\mathbb{E}_{e} = (S, E, I, R)_{e}$ when I > 0. \mathbb{E}_{e} is obtained by equating the right-hand side of the model (1) to zero and solving the corresponding system. Thus, we obtain the following result:

$$\mathbb{E}_{e} = \left(\frac{Nc_{1}b_{1}}{\beta\sigma}, -\frac{(N\delta\tau b_{1}c_{1} - Na_{1}b_{1}c_{1}d_{1} + b\beta\sigma d_{1})c_{1}}{\beta(\gamma\sigma\tau - b_{1}c_{1}d_{1})\sigma}, -\frac{N\delta\tau b_{1}c_{1} - Na_{1}b_{1}c_{1}d_{1} + b\beta\sigma d_{1}}{\beta(\gamma\sigma\tau - b_{1}c_{1}d_{1})\sigma}, -\frac{N\delta\tau b_{1}^{2}c_{1}^{2} + N\gamma\sigma a_{1}b_{1}c_{1} - b\beta\gamma\sigma^{2}}{\beta(\gamma\sigma\tau - b_{1}c_{1}d_{1})\sigma}\right)$$

Where: $a_1 = \delta + \mu$, $b_1 = \sigma + \mu$, $c_1 = \gamma + \lambda + \mu$, $d_1 = \mu + \tau$

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Local Stability of the Disease-free Equilibrium

We shall use the Jacobian matrix $J(\mathbb{E}_0)$ in establishing the local stability of the disease-free equilibrium. The Jacobian matrix which is evaluated at the disease-free equilibrium, is given by

Theorem

The disease-free equilibrium (DFE) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof

For local stability, the Jacobian matrix with respect to the system (1) at the disease-free equilibrium is given by:

$$\begin{bmatrix} -\delta - \mu - \Lambda & 0 & -\frac{\beta S}{N} & \tau \\ 0 & -\sigma - \mu - \Lambda & \frac{\beta S}{N} & 0 \\ 0 & \sigma & -\gamma - \lambda - \mu - \Lambda & 0 \\ \delta & 0 & \gamma & -\tau - \mu - \Lambda \end{bmatrix}$$

The characteristics equation is given by

$$(-\Lambda - \mu)(-\Lambda - \delta - \mu - \tau)$$

$$\begin{pmatrix} \frac{1}{2} \frac{\sqrt{N(N(\gamma - \sigma + \lambda)^2 e_1 + 4\sigma b\beta d_1)e_1} - (2\mu + \gamma + \sigma + 2\Lambda + \lambda)Ne_1}{Ne_1} \\ \\ \frac{1}{2} \frac{-\sqrt{N(N(\gamma - \sigma + \lambda)^2 e_1 + 4\sigma b\beta d_1)e_1} - (2\mu + \gamma + \sigma + 2\Lambda + \lambda)Ne_1}{Ne_1} \end{pmatrix} = 0$$

Where: $d_1 = \mu + \tau$, $e_1 = \mu(\delta + \mu + \tau)$

$$\begin{split} \Lambda_1 &= -\mu \\ \Lambda_2 &= -\delta - \mu - \tau \\ \Lambda_3 &= -\frac{1}{2} \frac{(2\mu + \gamma + \sigma + \lambda)Ne_1 - \sqrt{N(N(\gamma - \sigma + \lambda)^2 e_1 + 4\sigma b\beta d_1)e_1}}{Ne_1} \\ \Lambda_4 &= -\frac{1}{2} \frac{(2\mu + \gamma + \sigma + \lambda)Ne_1 + \sqrt{N(N(\gamma - \sigma + \lambda)^2 e_1 + 4\sigma b\beta d_1)e_1}}{Ne_1} \end{split}$$

 Λ_3 and Λ_4 holds provided $\sqrt{N(N(\gamma - \sigma + \lambda)^2 e_1 + 4\sigma b\beta d_1)e_1} < (2\mu + \gamma + \sigma + \lambda)Ne_1$

Hence from the above the disease-free equilibrium is locally asymptotically stable. This completes the proof.

3.4. The Basic Reproduction Number

The basic reproduction number is the average number of secondary infections caused by a single infectious individual in an entirely susceptible population during his/her infective period. The next generation matrix approach is used to obtain R_0 . Let X(t) = (E, I) and obtain that

$$X'(t) = \mathcal{F}(t) - \mathcal{V}(t)$$

where:

$$\mathcal{F}(t) = \begin{pmatrix} \frac{\beta SI}{N} \\ 0 \end{pmatrix}$$
 and $\mathcal{V}(t) = \begin{pmatrix} -(\sigma + \mu)E \\ \sigma E - (\gamma + \lambda + \mu)I \end{pmatrix}$

Evaluating the derivatives of F and V at the disease-free equilibrium point obtained above, yields FV^{-1} as seen below:

$$F\mathcal{V}^{-1} = \begin{pmatrix} -\frac{\beta S\sigma}{N(\sigma+\mu)(\gamma+\lambda+\mu)} & -\frac{\beta S}{N(\gamma+\lambda+\mu)} \\ 0 & 0 \end{pmatrix}$$

By solving the dominant eigenvalue of the next generation matrix FV^{-1} , we get the basic reproduction number to be

$$R_0 = -\frac{\beta S\sigma}{N(\sigma+\mu)(\gamma+\lambda+\mu)}$$

Therefore, the basic reproduction number of the given system of equations denoted by R_0 is:

$$R_0 = -\frac{\beta b(\mu + \tau)\sigma}{N\mu(\delta + \mu + \tau)(\sigma + \mu)(\gamma + \lambda + \mu)}$$

Effective Reproduction Number: The effective reproduction number (R_{eff}) is a critical epidemiological measure that offers insights into the transmission dynamics of infectious diseases, guiding public health responses during epidemics and pandemics. It signifies the average number of new infections generated by each infectious individual at a particular time during such outbreaks.

In the context of susceptible populations, denoted as *S* and *S*^{*} before and after factoring in immunity acquired through natural infection or vaccination, respectively, the basic reproduction number (R_0) is typically expressed in terms of epidemiological parameters like the transmission rate (β) and the mean infectious period (*D*). However, we can reframe R_0 by relating it to the effective susceptible population (S^*) and the standard susceptible population (*S*) through the following relationship:

$$0 \le S^* < S$$

Thus

$$0 \le \frac{S^*}{S} < 1 \tag{12}$$

Multiply (12) by R_0 implies

$$0 \le R_0 \frac{S^*}{S} < R_0 \tag{13}$$

From equation (13), the relationship between R_{eff} , R_0 S, and S^{*} can be described using the following equation

$$R_{eff} = R_0 \frac{S^*}{S}$$

Sensitivity Analysis

Intervention strategies to reduce the mortality and morbidity due to covid 19 perhaps any other epidemiology treatment and control should target the parameters that have a high impact on the effective reproduction number, R_0 . Sensitivity analysis is used to obtain the sensitivity index that is a measure of the relative change in a state variable when a parameter changes. We compute the sensitivity indices of R_0 to the model parameters with the

approach used by Chitnis et al. (2008). These indices show the importance of each individual parameter in the disease transmission dynamics and prevalence. The sensitivity of a parameter, say β , of R_0 is defined as

$$\xi_{\beta}^{R_0} = \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0}$$
(14)

The sensitivity indices of the parameters are thus presented as follows:

$$\begin{split} \xi_{\beta}^{R_{0}} &= 1 > 0 \qquad \qquad \xi_{\tau}^{R_{0}} = \frac{\tau\delta}{(\mu + \tau)(\delta + \mu + \tau)} > 0 \qquad \qquad \xi_{\sigma}^{R_{0}} = \frac{\mu}{\sigma + \mu} > 0 \\ \xi_{\mu}^{R_{0}} &= -\left(\frac{\tau}{(\mu + \tau)} + \frac{\mu}{\delta + \mu + \tau} + \frac{\mu}{(\sigma + \mu)} + \frac{\mu}{\gamma + \lambda + \mu}\right) < 0 \qquad \qquad \xi_{b}^{R_{0}} = 1 > 0 \\ \xi_{\delta}^{R_{0}} &= -\frac{\delta}{\delta + \mu + \tau} < 0 \qquad \qquad \xi_{\gamma}^{R_{0}} = -\frac{\gamma}{\gamma + \lambda + \mu} < 0 \qquad \qquad \qquad \xi_{\lambda}^{R_{0}} = -\frac{\lambda}{\gamma + \lambda + \mu} < 0 \end{split}$$

The analysis revealed that the positively sensitive parameters of the basic reproduction number, R0, are the recruitment rate (b) into the susceptible class, the probability (β) that each contact is effective enough to cause infection, the rate of transition from exposed to infectious class (σ), rate of transition from recovered to susceptible class (τ). Thus, reducing the number of susceptible individuals, reducing or eliminating contact with infected persons, effectively restricting infected humans from adding to the infected population, and ensuring that protected individuals remain protected can greatly lower the value of the basic reproduction number (R_0) and thereby increasing the stability of the disease-free equilibrium. Increasing the values of the positively sensitive parameters has the effect of increasing the value of the basic reproduction number (R_0), which implies an increase in the endemicity of the disease since the indices have positive signs. On the other hand, when the parameter values λ , δ , γ and μ are decreased while the rest of the parameter values are kept fixed, the value of R_0 decreases. This shows a decrease in the disease endemicity because the indices have negative signs.

Table 2: Numerical values of sensitivity indices of R_0

Parameter	Sensitivity Index	
β	1.0000	
b	1.0000	
σ	0.6570	
τ	0.0135	
λ	-0.0056	
δ	-0.0444	
γ	-0.4627	
μ	-2.1578	

IV. NUMERICAL SIMULATIONS

We illustrate the analytical results of the model by carrying out numerical simulation of the models using a set of estimated parameter values obtained from literature. The system is simulated using ODE solvers coded in MAPLE programming language (MAPLE 2022). The numerical simulation of the model under NPIs intervention is carried out to investigate the impact of the key parameters on the spread of Covid 19 and how the disease can be controlled. The parameter values are presented in Table 1.

From Fig. 10 through to 13, we see the effect that the natural death rate has on each of the classes. A higher natural death rate can reduce the size of the susceptible population over time as individuals leave the population due to natural causes which could slow down the spread of the disease. On the exposed class a higher natural death rate reduces the number of individuals in this class before they become infectious which leads to a lower number of individuals transitioning to the infectious class. On the infectious class the impact of the natural death rate depends on the disease induced death rate as shown in Fig. 7. If the death rate from the disease is higher than the natural death rate, the infectious class would decline due to deaths but if the natural death rate is higher, it may counteract the effect of the disease on the infectious class. The natural death rate affects the recovered class by contributing to the number of individuals who leave the infectious class due to death. Fig. 8 and 9 shows the effect of rate of transition from exposed to infectious class means individuals spend less

time in the exposed state before becoming infectious. This can lead to a shorter incubation period and a smaller exposed class size at any given time as individuals move more quickly into the infectious state. A higher transition from exposed to infectious class results in a faster increase in the infectious population as more individuals become infectious sooner. This can lead to a more rapid spread of the disease and potentially larger peak infectious population sizes.

The effect of recovery rate on the infectious and recovered classes can be seen in Fig. 6. A higher recovery rate means individuals spend less time in the infectious state before moving to the recovered state. This leads to a faster decline in the infectious population over time as more individuals recover from the disease and move out of the infectious class. Fig. 5 shows the effect of the transition rate from recovered to susceptible class. A higher transition rate from recovered to susceptible implies a faster rate at which individuals lose immunity and become susceptible again which leads to a decrease in the size of the population. Fig. 3 and 4 shows the effect of reduction in transmission due to NPIs on the susceptible and recovered class respectively. NPIs can reduce the transmission of the disease leading to fewer individuals becoming infected and transitioning to the recovered class. This can slow down the rate at which individuals are removed from the population due to recovery or death. As the transmission of the disease decreases due to NPIs, fewer individuals become infected and the susceptible population may decline at a slower rate or even stabilize. Fig. 2 on the other hand shows the effect of transmission without interventions on the exposed class. The absence of NPIs would result in a higher transmission rate leading to more individuals becoming exposed to the disease. The exposed class would grow larger more quickly reflecting the increased rate of new infections. Overall, the absence of NPIs would accelerate disease transmission resulting in more individuals transitioning from the susceptible to the exposed class. This would intensify the spread of the disease and potentially lead to larger outbreaks.



Fig. 2. Effect of transmission without interventions on the exposed class



Fig. 4. Effect of reduction in transmission due to



Fig. 3. Effect of reduction in transmission due to NPIs on the susceptible class



Fig. 5. Effect of transition rate from recovered to susceptible class on the recovered class





Fig. 6. Effect of recovery rate on the infected class



Fig. 8. Effect of rate of transition from exposed to infectious class on exposed class



Fig. 10. Effect of the natural death rate on infected humans



Fig. 7. Effect of disease induced death on infected class



Fig. 9. Effect of rate of transition from exposed to infectious class on infected class



Fig. 11. Effect of the natural death rate on exposed humans



Fig. 12. Effect of the natural death rate on recovered humans



Fig. 13. Effect of the natural death rate on susceptible humans

V. SUMMARY, CONCLUSION AND RECOMMENDATION

In this paper, we examined a SEIR model to explore the effectiveness of non-pharmaceutical interventions that have been put into place to help curb the spread of Covid-19. We determined the existence and local stability of the disease-free equilibrium along with the existence of the endemic equilibrium. Then, the basic reproduction number was computed using the next generation matrix. From the numerical simulations of the model, it was shown that when more people comply with the various interventions, the spread of the disease is reduced greatly but when the rate of those who don't comply with such interventions is n the increase it leads to an increase in the spread of the disease.

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