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A mathematical model for COVID-19 transmission : The effects of the presymptomatic infectious individuals and the infectivity factor on the dynamics of the infection

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Abstract

Recent studies affirmatively revealed that infectious cases of the virus are not restricted to asymptomatic and symptomatic. There is an infectious class – the presymptomatic infectious individuals, a non-trivial infectious case which has posed more threat to humanity. These individuals' symptoms are delayed due to their intermediate immune system and then become a threat to the population after 21 days of the incubation period. In this study, we proposed 7-dimensional nonlinear ordinary differential equations of the form $SEI_sI_aI_pTR$ which incorporate the presymptomatic infectious individuals and then considered the effect

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of infectivity factors on all forms of infectious status to emphasize the presymptomatic individuals cannot be ignored in the treatment of infected individuals. The non-negativity and boundedness of the dynamical system were at all-time proved to be true. Also, the endemic and disease-free points of equilibrium were detremined to be locally and globally asymptotically stabled. The dynamical behaviors are succinctly conducted with the use of MATLAB.

Subject Classification: *92B05, 92D30, 93D05.*

Keywords: *COVID-19, Presymptomatic, Symptomatic, Asymptomatic, Infectivity factors.*

1. Introduction

A mathematical model is a representation of real-world problems incorporating mathematical parameters, constraints, and equations coupling with a possible way out or solutions. It is a powerful tool for present and future predictions. This branch of mathematics called epidemiology is commonly implemented in various fields, which are social sciences, medicines, management, agriculture, and so on. Mathematical models can be segmented into linear, non-linear, deterministic, and or stochastic. Amongst others, mathematical models have been used severally in health sectors to contrive the upsurge of disease, and shun or heal these sicknesses [2, 11].

Coronavirus (COVID-19) previously known as a new coronavirus (2019-nCoV) resulted from Severe Acute Respiratory Syndrome CoronaVirus 2 (SARS-CoV-2) is highly infectious [20] and has constituted an enormous threat to the global public health. It was reported by the (WHO) to have emerged in Wuhan City, in the Hubei regiion of China in the concluding part of the year 2019. They are a relatively great family size of zoonotic viruses. They are said to be transmissible from animals to humans but not vice versa. Strains of the virus abound [22] and these viruses' birth symptoms are as naive as the typical cold to much more acute sicknesses such as Middle East Respiratory Syndrome (MERS) that spread form Camelus dromedaries (a single humped camel) to humans and Severe Acute Respiratory Syndrome that is spread from *Civettictis civettta* to humans.

The recently discovered coronavirus Severe Acute Respiratory Syndrome-Coronavirus-2 (SAR-CoV-2) is said to be a new kind of coronaviruses that is yet to be visualized in Humans, where symptoms connected to respiration, cough, fever, seizure of breath, are possible signs of the infection with the recent discovery of coronaviruses. Sequel to acute

cases, this disease may lead to kidney failure, pneumonia, and also death [22].

From various researches, the WHO then stated the following standard recommendations to reduce the rate of exposure as well as transmission. This can be encapsulated as respiratory hygiene, hand hygiene, and so on. The following were the recommendations [17]:

1) More regular hand washing with soap and water or a hydro-alcoholic solution.
2) Covering of mouth and nose with a handkerchief or with the zone of your elbow if you sneeze or cough: the handkerchief should be disposed immediately after such is done. Refrain from touching those who are coughing and/or have a fever.
3) When experiencing symptoms such as cough, dyspnea, or fever, promptly seek medical attention from a physician and disclose all travel-related injuries.
4) Steer clear of unprotected direct contact with live animals and surfaces that have come into contact with animals in markets where new coronavirus cases have been reported.
5) In the end, it is best to avoid consuming animal products that are raw or undercooked. Raw meat, milk, and organ meats should be handled with extreme caution before following good food safety procedures to prevent cross-contamination with raw food.

Presently, vaccines for the current coronavirus SARS-CoV-2 are yet to be discovered, however, surfacing symptoms can be treated depending on the clinical conditions of the affected individuals and the quality of supportive care available to infected people [21].

Numerous modeling approaches, from stochastic [8, 15] to deterministic [3, 5, 7, 10, 13, 14, 15, 18, 19, 23], have been undertaken to analyze the COVID-19 pandemic's propagation. In [8], a mathematical model for SARS-CoV-2 transmission dynamics using four (4) datasets coupled with an additional two (2) datasets was proposed and studied to access the propagation of covid-19 in Wuhan from December 2019 to February 2020 [12, 24]. By incorporating compartments to represent changes between reporting and disease states, as well as Poisson and Binomial Observation processes for infection, the model SEIR was able to account for delays in symptom onset and reporting. A compartmentalized

age-structured model of the dynamics of COVID-19 transmission in the estimated population of Ontario, Canada was implemented in [16]. The study examined three scenarios: enhanced case finding; a mix of enhanced case finding and less stringent social distancing measures; and a base case with inadequate testing, isolation, and quarantine. Prior to the anticipated bed occupancy in intensive care units, interventions were either dynamically cycled on and off or applied for predetermined durations. Using a two-year time horizon, they gave the median and credible intervals (CrI) from 100 replicates of each scenario.

An extension to the age-structured compartmental model of COVID-19 was done by [18]. The authors emphatically analyzed the age-structured constraints and all other demographic factors. They concluded that age is not a significant factor that hastens the level of infectiousness but gender and transmission type are sacrosanct factors to be put into consideration during the upsurge of COVID-19. And in [19], an unprecedented model was proposed that analyzed the dynamics of COVID-19 transmission specifically in Bangkok, Thailand. The authors clinically perused the implications of control strategies employed including how effective they were in preventing COVID-19 outbreaks in Thailand. Some models have been proposed in the past to address the epidemics of infectious diseases [1, 6]. A Bats-Hosts-Reservoir-People transmission network model was examined in [5] to simulate the possibility of transmission from the infection source, which is likely a bat, to human. This model was latter simplify to Reservoir-People (RP) model. R_0 was calculated to be 2.30 between reservoir and person and 3.58 between individuals in persons. In [10], they formulated a compartmental SEIRNDC model where they take into account: the effect of governmental, individual behavioral response, zoonotic transmission dynamics, and emigration of a huge population. The estimated value of the reproduction number was 2.8. Considering [13], the study aimed to create and analyze the new deterministic endemic SEIRUS compartmental model of dynamics of coronavirus transmission by incorporating quarantine protocols and behavioral modifications as well as social distancing in the management and eradication of the disease in the most exposed sub-population. The evaluated R_0 is -0.009505 which justified that the secondary infection will not last. The study in [23] presented a novel mathematical model of the form SEIRV for the virus which incorporated diverse transmission dynamics paths, such as the environment to human and human-to-human routes. Particularly, an environmental compartmentalized variable that represented the pathogen concentration in the environmental reservoir

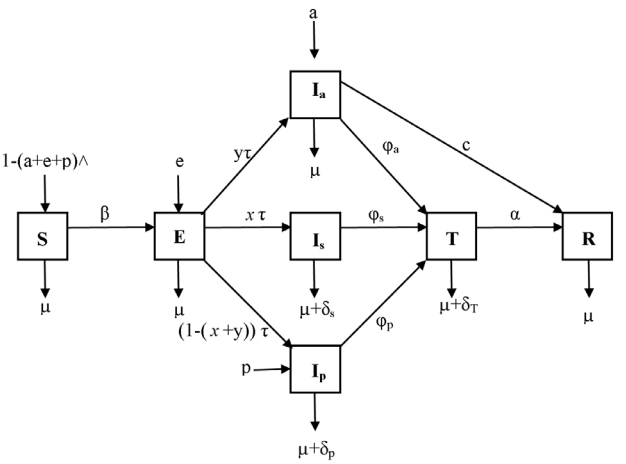
was introduced and estimated variables of R_0 were obtained for all succinctly stated conditions.

Studies have shown that the non-isolation of infectious humans can cause bring risk to the presiding population thereby inflating the secondary infection parameter R_0, an isolation class was added in [3] to the preexisting SEIQR model since it was evident that the only potential route of transmission of COVID-19 is human to human contact. The study implemented the nonstandard finite difference (NSFD) scheme and Runge-Kutta fourth-order method to confirm R_0 is less than one. And in [13], a non-existing nonlinear deterministic epidemic model for Coronavirus (Covid-19) coupling transmission paths from three known infectious classes-symptomatic, asymptomatic, and hospitalized individuals was developed based on the collective number of noted active cases of the disease. Qualitative and quantitative behavioral analysis of the model was investigated over an extended period. Moreover, optimal control and economically feasible control strategy with the ability to flatten the virus infection curve over an appointed period of time were proposed. Furthermore, [14] carried out a sensitivity analysis in their model, stating possible conditions for the endemic state of the virus. They formulated a SEQAIR model with more emphasis on the pattern of transmission amongst humans, therefore, disregarding the reservoir variable, and then added new death cases parameter to the model. In this study, the estimated value of reproduction number was approximately 3.097 and the sensitivity analysis index was -0.911. In [7], an SEIHR(S) model that assumed a homogeneous-mixing approximation and frequency-dependent disease transmission was considered putting into consideration: the percentage of arrivals for both asymptomatic, latently infected individuals and infectivity factors. The R_0 is of the kind $R_1+R_2+R_3$ which represented the reproduction number for symptomatic, asymptomatic, and hospitalized respectively. Their study captured Uganda and its environs and their total estimated value of reproduction number is 0.20617 where all individualized R_n, n=1, 2, 3 are also less than one.

With the existing and emerging vaccines to globally tame the killer virus coupled with numerous studies of the mathematical model to give insight into transmission routes and possible taming strategies, this study aim at putting a spotlight on the effect the presymptomatic infectious individuals have on the transmission dynamics of the virus The exposed class are not restricted to asymptomatic (sub clinic) and symptomatic alone. Further studies made us understand that there are presymptomatic

cases of covid-19. These are individuals whose immune systems are intermediate. They tend to behave like asymptomatic immediately after the incubation period by not showing signs but eventually showing signs. This set of infectious individuals will silently spread the virus in the population and should not be overlooked as they can be said to be extremely dangerous to the affected population. So, we, therefore, added presymptomatic infectious individuals to our model which greatly affected the per-capital recruitment rate, presymptomatic and treatment infectivity factors, treatment compartment, and also death cases of Covid-19 in the NCDC observed compartments without forgetting that some of the asymptomatic infectious individuals get recovered even without treatment.

2. 2.0 Conceptual Model Formulation and equations



S - Susceptible individuals; E - Exposed individuals; I_s - Symptomatic infectious individuals; I_a - Sub-clinic infectious individuals; I_p - Pre-symptomatic infectious individuals; T - Treatment class for infectious individuals; R - Recovered individuals

$a(t)$ - represents the percentage of arrivals that are asymptomatic

$e(t)$ - represents the percentage of arrivals who are exposed

$p(t)$ - represents the percentage of arrivals that are pre-asymptomatic

$\wedge$ - represents per capita recruitment rate; β - Contact rate

m - Infectivity factor for asymptomatic individuals

n - Infectivity factor for pre-asymptomatic individuals

b - Infectivity factor for the individual in treatment class

$\mu_S, \mu_E, \mu_a, \mu_a, \mu_p, \mu_T, \mu_R$ - respective natural mortality rates for susceptible, exposed, and symptomatic individuals

τ - Progression rate from the exposed class to the three existing classes: symptomatic, sub-clinic and pre-asymptomatic

x - Percentage of exposed individuals that becomes symptomatic

y - Percentage of exposed individuals that becomes sub-clinic

ϕ_s - Treatment rate of symptomatic infectious individuals

ϕ_a - Treatment rate of sub-clinic infectious individuals

ϕ_p - Treatment rate of pre-asymptomatic infectious individuals

c - Recovery rate of sub-clinic infectious individuals

$\delta_s, \delta_p, \delta_T$ - Respective disease-induced mortality rate of symptomatic, pre-symptomatic, and treatment infectious class;

α - Recovery rate of treatment individuals

2.1 *Model assumptions*

1) In lieu of the description of the parameter stated above, the following assumptions are included:
2) Prior to infection of an individual, an exposed individual can be identified through, for example, contact tracing and then consciously compartmentalized into symptomatic, sub-clinic, and pre-symptomatic because of the difference in the immune system leading to an absence of symptoms.
3) When it comes to keeping the integrity of disease transmission, hospitalized or quarantined individuals who are exposed after being contact-traced are assumed not to have contributed to the spread of the illness.
4) The sub-clinic individuals which are the 'silent spreader' are assumed to be less likely to succumb and this leads to recovery without treatment depending on the body system, though the absence of symptoms does not imply the absence of harm to both the infectious individuals and the public. An initial study [11] indicated that sub-clinic individuals may eventually suffer lung damage.

5) Individuals in treatment class have the intensity of spreading the disease although at a much slower rate than the free-leaving infectious people.
6) Albeit viral volumes have been found to be the same between sub-clinic, symptomatic, and pre-symptomatic individuals, the sub-clinic infectious persons may have considerably low infectivity since they may not sneeze or cough or have a high/fluctuating temperature as much as symptomatic. This however needs to be further succinctly explained.
7) Some asymptomatic infectious individuals have been popularly known to recover without treatment. They only need to be in self-isolation.

2.2 *The model*

Sequel to the above description, a compartment-based model to describe the dynamics of the disease transmission in Nigeria is presented below.

$$\left.\begin{aligned}
\frac{dS}{dt} &= [1-(a+\rho+e)] \wedge -\beta S(mI_a + I_s + nI_p + bT) - \mu_s S --- 2.1 \\
\frac{dE}{dt} &= e \wedge +\beta S(mI_a + I_s + nI_p + bT) - (\tau + \mu_e)E ------- 2.2 \\
\frac{dI_s}{dt} &= x\tau E - (\phi_s + \mu_{IS} + \delta_{IS})I_s ---------- 2.3 \\
\frac{dI_a}{dt} &= y\tau E + a \wedge -(\phi_a + \mu_a + c)I_a ---------- 2.4 \\
\frac{dI_p}{dt} &= [1-(x+y)]\tau E + \rho \wedge -(\phi_p + \mu_p + \delta_p)I_p ------- 2.5 \\
\frac{dT}{dt} &= \phi_s I_s + \phi_a I_a + \phi_p I_p - (\alpha + \mu_T + \delta_T)T ---------- 2.6 \\
\frac{dR}{dt} &= \alpha T - \mu_R R + cI_a ---------- 2.7
\end{aligned}\right\} \quad (2)$$

Where

$S(t=0) \geq 0$, $E(t=0) \geq 0$, $I_s(t=0) \geq 0$, $I_a(t=0) \geq 0$, $I_p(t=0) \geq 0$, $T(t=0) \geq 0$ and $R(t=0) \geq 0$; $N(t) = S(t) + E(t) + I_s(t) + I_a(t) + I_p(t) + T(t) + R(t)$

3. Positivity and boundedness of the solution

We now study the non – negativity of equation (2) with the initial condition (3).

Theorem 1: *Solutions of the system of equations (2) with starting condition (3) are positive for all* $t \geq 0$

Proof: Assume $[S(t), E(t), I_s(t), I_a(t), I_p(t), T(t), R(t)]$ is a solution of equation (2) with the initial condition (3). Let us consider $E(t)$ for $t \geq 0$. It succeeds from (2.2). That is;

$$\frac{dE}{dt} = e \wedge + \beta S(mI_a + I_s + nI_p + bT) - (\tau + \mu_e)E$$

Removing the positive terms gives;

$$\frac{dE}{dt} \leq -(\tau + \mu_e)E \Rightarrow \frac{dE}{dt} + (\tau + \mu_e)E \leq 0.$$

$$\Rightarrow \qquad E(t) = E_0 e^{-(\tau+\mu_e)t} \succ 0;\ \forall t \geq 0\ ; E_0 = E(0) \succ 0,$$

Then, the sum of the positive terms of $E(t)$ is positive at all time.

Recall from (2.3) that

$$\frac{dI_s}{dt} \leq -(\phi_s + \mu_{IS} + \delta_{IS})I_s;\ I_s(t) = I_{s_0} e^{-(\varphi_s+\mu_{IS}+\delta_{IS})t} \succ 0\ \forall t \geq 0$$

Extending to equations 2.4, 2.5, 2.6 and 2.7 respectively;

$$I_a(t) = I_{a_0} e^{-(\phi_a+\mu_a+c)t} \succ 0\ \ ;\ \ \forall\ t \geq 0; \qquad I_p(t) = I_{p_0} e^{-(\phi_p+\mu_p+\delta_p)t} \succ 0\ \ ;\ \forall\ t \geq 0,$$

$$T(t) = T_0 e^{-(\alpha+\mu_T+\delta_T)t} \succ 0\ \ ;\ \forall \geq 0; \qquad R(t) = R_0 e^{-\mu_R t} \succ 0\ \ ;\ \forall\ t \geq 0.$$

Next, we show that $S(t)$ is positive. From;

$$\begin{aligned} \frac{dS}{dt} &= [1-(a+p+e)] \wedge -\beta S(mI_a + I_s + nI_p + bT) - \mu_s S \\ &= [1-(a+p+e)] \wedge -[\beta(mI_a + I_s + nI_p + bT + \mu_s)]S \end{aligned} \tag{4}$$

Where $0 \leq a(t) \prec 1$, $0 \leq p(t) \prec 1$, $0 \leq e(t) \prec 1$ and $a(t) + p(t) + e(t) \prec 1$ which implies $1-(a+p+e) \succ 1$.

All these show that $I_a(t) \succ 0$, $I_s(t) \succ 0$, $I_p(t) \succ 0$ and $T(t) \succ 0$.

Assume the inconsistency; then, let t_1 be the first time such that $S(t_1) = 0$. By (2.1) through (2.4), we have;

$\left.\frac{dS(t)}{dt}\right|_{t=t_1} = [1-(a+p+e)] \wedge \succ 0$ implying $S(t) \prec 0$ for $t \in (t_1 - \xi, t_1)$, where ξ is an arbitrary small non-negative constant. This is a contradiction.

It follows that $S(t)$ is always positive for all $t \geq 0$. This completes the proof.

Theorem 2: *Solutions of the equation (2) are ultimately bounded.*

Proof: Considering theorem 1, solutions of (2) with starting condition (3) are non-negative for all $t \geq 0$.

Let $N(t) = S(t) + E(t) + I_s(t) + I_a(t) + I_p(t) + T(t) + R(t)$. Then from equations (2), we have;

$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI_s(t)}{dt} + \frac{dI_a(t)}{dt} + \frac{dI_p(t)}{dt} + \frac{dT(t)}{dt} + \frac{dR(t)}{dt}$. That is;

$$\frac{dN(t)}{dt} = \wedge - \mu_s S - \mu_e E - \mu_{IS} I_s - \mu_a I_a - \mu_p I_p - \mu_T T - \mu_R R - \delta_{IS} I_s - \delta_p I_p - \delta_T$$

Since μ is natural mortality rate, then let $\mu_s = \mu_R = \mu_{IS} = \mu_a = \mu_p = \mu_r = \mu$ and $\delta_{IS} = \delta_p = \delta_T = \delta$. Assume the disease is absent, that is, $\delta = 0$, then we have that;

$\frac{dN}{dt} \leq \wedge - \mu(S + E + I_s + I_a + I_p + T + R)$. This implies;

$$\frac{dN}{dt} \leq \wedge - \mu N \tag{5}$$

Using idea of linear differential equation coupled with Grownwall's inequality in [4], this gives;

$N(t) \leq \frac{\wedge}{\mu} + \left(N_0 - \frac{\wedge}{\mu}\right) e^{-\mu t}$ which means $N(t) \leq \frac{\wedge}{\mu}$ for all $t \geq 0$ whenever $N_0 \leq \frac{\wedge}{\mu}$ (6)

This shows clearly that $\lim_{t \to \infty} \sup remum N \leq \frac{\wedge}{\mu}$

Hence, $N(t) \prec \frac{\wedge}{\mu} + \xi$ for all big t, with the value ξ as an arbitrary small non-negative constant. Thus, $S(t)$, $E(t)$, $I_s(t)$, $I_a(t)$, $I_p(t)$, $T(t)$, $R(t)$ are at the end bounded.

3.2 Basic reproductive number

The importance of R_0 among the parameters of epidemiological models cannot be over-emphasized. It indicates the reproductive system of models and invasion of infection is can simply be predicted by simulating R_0. One of the objectives of this work is to analyze all parameters that will ensure the value of R_0 is less than unity. We would engage the next-generation matrix method and also implement the method used by [7].

From equation (2), let;

$$X_1 = [E, I_s]^T, \overset{*}{X}_1 = [F_1 + V_1]X \tag{7}$$

$$X_2 = [E, I_a]^T, \overset{*}{X}_2 = [F_2 + V_2]X \tag{8}$$

$$X_3 = [E, I_p]^T, \overset{*}{X}_3 = [F_3 + V_3]X \tag{9}$$

$$X_4 = [E, I_s, I_p, T]^T, \overset{*}{X}_4 = [F_4 + V_4]X \tag{10}$$

Here the reproductive number R_0 is given by $R_0 = R_1 + R_2 + R_3 + R_4$.

Where R_1 is reproductive rate of the symptomatic individuals, R_2 the reproductive rate of sub-clinic individuals, R_3 the reproductive rate of pre-symptomatic individuals, and R_4 the reproductive rate of individuals in treatment class where we combine the symptomatic and pre-symptomatic individuals. Also, the time rate a person spends in the various compartment I_s, I_a, I_p and T are obtained.

From equation (7);

$$X_1 = [E, I_s]^T, \overset{*}{X}_1 = [F_1 + V_1]X$$

Considering (2), Let $E = A_1$ and $I_s = A_2$ for F_1, $E = B_1$ and $I_s = B_2$ for V_1.

Rewriting F_1 and V_1 in Jacobian matrix form;

$$F_1 = \begin{pmatrix} \frac{\partial A_1}{\partial E} & \frac{\partial A_1}{\partial I_s} \\ \frac{\partial A_2}{\partial E} & \frac{\partial A_2}{\partial I_s} \end{pmatrix}, \quad V_1 = \begin{pmatrix} \frac{\partial B_1}{\partial E} & \frac{\partial B_1}{\partial I_s} \\ \frac{\partial B_2}{\partial E} & \frac{\partial B_2}{\partial I_s} \end{pmatrix} \Rightarrow \quad F_1 = \begin{pmatrix} 0 & \beta S \\ 0 & 0 \end{pmatrix}, \quad V_1 = \begin{pmatrix} \tau + \mu_e & 0 \\ -x\tau & \phi_s + \mu_{IS} + \delta_{IS} \end{pmatrix}$$

Since $R_0 = \rho(FV^{-1})$, $R_1 = \rho(F_1 V_1^{-1})$ and $S \cong N = \frac{\wedge}{\mu}$, $|V_1| = (\tau + \mu_e)(\phi_s + \mu_{IS} + \delta_{IS})$

$$V_1^{-1} = \frac{1}{(\tau+\mu_e)(\phi_s+\mu_{IS}+\delta_{IS})} \begin{pmatrix} (\phi_s+\mu_{IS}+\delta_{IS}) & 0 \\ x\tau & (\tau+\mu_e) \end{pmatrix}$$

$$= \begin{pmatrix} \frac{1}{(\tau+\mu_e)} & 0 \\ \frac{x\tau}{(\tau+\mu_e)(\phi_s+\mu_{IS}+\delta_{IS})} & \frac{1}{(\phi_s+\mu_{IS}+\delta_{IS})} \end{pmatrix}$$

$$R_1 = \begin{pmatrix} \frac{\beta \wedge x\tau}{\mu(\tau+\mu_e)(\phi_s+\mu_{IS}+\delta_{IS})} & \frac{\beta \wedge}{\mu(\phi_S+\mu_{IS}+\delta_{IS})} \\ 0 & 0 \end{pmatrix}$$

This implies

$$R_1 = \frac{\beta \wedge x\tau}{\mu(\tau+\mu_e)(\phi_s+\mu_{IS}+\delta_{IS})} \tag{11}$$

From equation (8); $X_2 = [E, I_a]^T$, $\overset{*}{X}_2 = [F_2 + V_2]X$.

$$F_2 = \begin{pmatrix} 0 & \frac{\beta \wedge m}{\mu} \\ 0 & 0 \end{pmatrix}, \; V_2 = \begin{pmatrix} (\tau+\mu_e) & 0 \\ -y\tau & (\phi_a+\mu_a+c) \end{pmatrix}$$

$$|V_2| = (\tau+\mu_e)(\phi_a+\mu_a+c)$$

$$V_2^{-1} = \begin{pmatrix} \frac{1}{(\tau+\mu_e)} & 0 \\ \frac{y\tau}{(\tau+\mu_e)(\phi_a+\mu_a+c)} & \frac{1}{(\phi_a+\mu_a+c)} \end{pmatrix}$$

$$R_2 = \begin{pmatrix} \frac{\beta \wedge my\tau}{\mu(\tau+\mu_e)(\phi_a+\mu_a+c)} & \frac{\beta \wedge m}{\mu(\phi_a+\mu_a+c)} \\ 0 & 0 \end{pmatrix}$$

This implies

$$R_2 = \frac{\beta \wedge my\tau}{\mu(\tau+\mu_e)(\phi_a+\mu_a+c)} \tag{12}$$

From equation (9), $X_3 = [E, I_p]^T$, $\overset{*}{X}_3 = [F_3 + V_3]X$

$$F_3 = \begin{pmatrix} 0 & \frac{\beta \wedge n}{\mu} \\ 0 & 0 \end{pmatrix}, \; V_3 = \begin{pmatrix} (\tau+\mu_e) & 0 \\ -(1-(x+y))\tau & (\phi_p+\phi\mu_p+\delta_p) \end{pmatrix}$$

$$|V_3| = (\tau+\mu_e)(\phi_p+\mu_p+\delta_p)$$

$$V_3^{-1} = \begin{pmatrix} \frac{1}{\tau+\mu_e} & 0 \\ \frac{(1-(x+y))\tau}{(\tau+\mu_e)(\phi_p+\mu_p+\delta_p)} & \frac{1}{\phi_p+\mu_p+\delta_p} \end{pmatrix}$$

That is, $$R_3 = \begin{pmatrix} \frac{\beta \wedge n\tau(1-(x+y))}{\mu(\tau+\mu_e)(\phi_p+\mu_p+\delta_p)} & \frac{\beta \wedge n}{\mu(\phi_p+\mu_p+\delta_p)} \\ 0 & 0 \end{pmatrix}$$

Therefore,

$$R_3 = \frac{\beta \wedge n\tau(1-(x+y))}{\mu(\tau+\mu_e)(\phi_p+\mu_p+\delta_p)} \tag{13}$$

From equation (10), $X_4 = [E, I_s, I_p, T]^T$, $\overset{*}{X}_4 = [F_4 + V_4]X$

$$F_4 = \begin{pmatrix} 0 & \frac{\beta\wedge}{\mu} & \frac{\beta\wedge n}{\mu} & \frac{\beta\wedge b}{\mu} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V_4 = \begin{pmatrix} (\tau+\mu_e) & 0 & 0 & 0 \\ -x\tau & (\phi_s+\mu_{IS}+\delta_{IS}) & 0 & 0 \\ -(1-(x+y))\tau & 0 & (\phi_p+\mu_p+\delta_p) & 0 \\ 0 & -\phi_s & -\phi_p & (\alpha+\mu_p+\delta_p) \end{pmatrix},$$

Obtaining the minor (M) of V_4;

$$M = \begin{pmatrix} ABD & -BDx\tau & AD(1-(x+y))\tau & -x\tau\phi_s B - A\phi_p(1-(x+y))\tau \\ 0 & BD(\tau+\mu_e) & 0 & B(\tau+\mu_e) \\ 0 & 0 & AD(\tau+\mu_e) & -A\phi_p(\tau+\mu_e) \\ 0 & 0 & 0 & AB(\tau+\mu_e) \end{pmatrix}$$

And the cofactor C;

$$C = \begin{pmatrix} ABD & x\tau BD & AD(1-(x+t))\tau & Bx\tau + A\phi_p(1-(x+y))\tau \\ 0 & BD(\tau+\mu_e) & 0 & B\phi_s(\tau+\mu_e) \\ 0 & 0 & AD(\tau+\mu_e) & A\phi_p(\tau+\mu_e) \\ 0 & 0 & 0 & AB(\tau+\mu_e) \end{pmatrix}$$

$$|V_4| = ABD(\tau+\mu_e)$$

Where $A = \phi_s + \mu_{IS} + \delta_{IS}$, $B = \phi_p + \mu_p + \delta_p$ and $D = \alpha + \mu_p + \delta_p$.

Using the adjoint matrix of the cofactors, we have;

And this implies $R_4 = \frac{\beta\wedge b\tau[Bx\phi_s + A\phi_p(1-x-y)]}{ABD\mu(\tau+\mu_e)}$

$$R_4 = \frac{\beta\wedge b\tau[x\phi_s(\phi_p+\mu_p+\delta_p)+\phi_p(\phi_s+\mu_{IS}+\delta_{IS})(1-x-y)]}{\mu(\tau+\mu_e)(\phi_s+\mu_{IS}+\delta_{IS})(\phi_p+\mu_p+\delta_p)(\alpha+\mu_T+\delta_T)} \tag{14}$$

3.3 *Analysis of the Stability of the model*

3.3.1 The Local stability analysis

Theorem: *The infection-free equilibrium of the system (2.1) –(2.7) is local asymptotically stable if all eigenvalues of the system Jacobian are non-positive real values.*

Proof: At disease-free equilibrium, $\mathrm{E}^0 = \left(\frac{\wedge}{\mu}, 0, 0, 0, 0, 0, 0\right)$, the Jacobian matrix, $J(S, E, I_s, I_a, I_p, T, R)$ is;

$$J = \begin{bmatrix} -\beta(mI_a + I_s + nI_p + bT) - \mu_s & 0 & -\beta S & -\beta Sm & -\beta Sn & -\beta Sb & 0 \\ \beta(mI_a + I_s + nI_p + bT) & -(\tau + \mu_e) & \beta S & \beta Sm & \beta Sn & \beta Sb & 0 \\ 0 & x\tau & -(\phi_s + \mu_{IS} + \delta_{IS}) & 0 & 0 & 0 & 0 \\ 0 & y\tau & 0 & -(\phi_a + \mu_a + c) & 0 & 0 & 0 \\ 0 & (1-(x+y))\tau & 0 & 0 & -(\phi_p + \mu_p + \delta p) & 0 & 0 \\ 0 & 0 & \phi_s & \phi_a & \phi_p & -(\alpha + \mu_T + \delta_T) & 0 \\ 0 & 0 & 0 & c & 0 & \alpha & -\mu_R \end{bmatrix}$$

Let $m = n = b = 0$, then we have;

$$J^* = \begin{bmatrix} -\mu_S & 0 & -\frac{\beta\wedge}{\mu} & 0 & 0 & 0 & 0 \\ 0 & -(\tau + \mu_e) & \frac{\beta\wedge}{\mu} & 0 & 0 & 0 & 0 \\ 0 & x\tau & -(\phi_s + \mu_{IS} + \delta_{IS}) & 0 & 0 & 0 & 0 \\ 0 & y\tau & 0 & -(\phi_a + \mu_a + c) & 0 & 0 & 0 \\ 0 & (1-(x+y))\tau & 0 & 0 & -(\phi_p + \mu_p + \delta_p) & 0 & 0 \\ 0 & 0 & \phi_S & \phi_a & \phi_p & -(\alpha + \mu_T + \delta_T) & 0 \\ 0 & 0 & 0 & c & 0 & \alpha & -\mu_R \end{bmatrix}$$

$$|J^* - \lambda I| = \begin{bmatrix} -\mu_s - \lambda & 0 & -\frac{\beta\wedge}{\mu} & 0 & 0 & 0 & 0 \\ 0 & -(\tau + \mu_e) - \lambda & \frac{\beta\wedge}{\mu} & 0 & 0 & 0 & 0 \\ 0 & x\tau & -(\phi_s + \mu_{IS} + \delta_{IS}) - \lambda & 0 & 0 & 0 & 0 \\ 0 & y\tau & 0 & -(\phi_a + \mu_a + c) - \lambda & 0 & 0 & 0 \\ 0 & (1-(x+y))\tau & 0 & 0 & -(\phi_p + \mu_p + \delta_p) - \lambda & 0 & 0 \\ 0 & 0 & \phi_s & \phi_a & \phi_p & -(\alpha + \mu_T + \delta_T) - \lambda & 0 \\ 0 & 0 & 0 & c & 0 & \alpha & -\mu_R - \lambda \end{bmatrix}$$

In the first column, the only entry that is not zero is $-\mu_s - \lambda$, therefore; $\lambda_1 = -\mu_s$

Deleting the first row and column gives;

$$J_0 = \begin{bmatrix} -(\tau + \mu_e) - \lambda & \frac{\beta\wedge}{\mu} & 0 & 0 & 0 & 0 \\ x\tau & -(\phi_S + \mu_{IS} + \delta_{IS}) - \lambda & 0 & 0 & 0 & 0 \\ y\tau & 0 & -(\phi_a + \mu_a + c) - \lambda & 0 & 0 & 0 \\ (1-(x+y))\tau & 0 & 0 & -(\phi_p + \mu_p + \delta_p) - \lambda & 0 & 0 \\ 0 & \phi_s & \phi_a & \phi_p & -(\alpha + \mu_T + \delta_T) - \lambda & 0 \\ 0 & 0 & c & 0 & \alpha & -\mu_R - \lambda \end{bmatrix}$$

Similarly, the only entry in the sixth column that is not zero is $-\mu_R - \lambda$ and this implies $\lambda_2 = -\mu_R$.

Deleting the first row and column from the matrix J_0 and subsequent matrices gives, $\lambda_3 = -(\alpha + \mu_T + \delta_T)$, $\lambda_4 = -(\phi_p + \mu_p + \delta_p)$, $\lambda_5 = -(\phi_a + \mu_a + c)$, $\lambda_6 = -(\tau + \mu_e)$ and $\lambda_7 = -(\phi_s + \mu_{IS} + \delta_{IS})\ (\tau - \mu_e) + \frac{\beta \wedge}{\mu} x\tau$. That is;

$$\lambda_7 = -(\phi_s + \mu_{IS} + \delta_{IS})(\tau + \mu_e)\left[1 - \frac{\beta \wedge x\tau}{\mu(\tau+\mu_e)(\phi_s+\mu_{IS}+\delta_{IS})}\right].$$

Recall $R_1 = \frac{\beta \wedge x\tau}{\mu(\tau+\mu_e)(\phi_s+\mu_{IS}+\delta_{IS})}$ and putting this in λ_7 implies;

$$-(\phi_s + \mu_{IS} + \delta_{IS})(\tau + \mu_e)(1 - R_1) \prec 0.$$

That is $1 - R_1 \prec 0$ and so $R_1 \prec 0$ and this completes the proof.

3.3.2 Global stability analysis

Theorem: *The disease-free equilibrium (DFE),* $E_0 = \left(\frac{\wedge}{\mu}, 0, 0, 0, 0, 0, 0\right)$ *of the system (2) is globally asymptotically stable if* $R_0 \leq 1$.

Proof: Let $S_0 = \frac{\wedge}{\mu}$ and as a result of the combination of the following linear and Lyapunov function as explained in [9];

$$\begin{aligned} L_0 &= L_0(S, E, I_a, I_s, I_p, T, R) \\ &= S - S_0 \ln(S) + E + K_1 I_s + K_2 I_a + K_3 I_p + K_4 T \end{aligned} \tag{19}$$

Attributing the fact that $\wedge = \mu S$, the Lie derivative of L_0 in the direction of the vector field provided by the right-hand-side of equation (2) is;

$$L_0^{\bullet} = \frac{dS}{dt}\left(1 - \frac{S_0}{S}\right) + \frac{dE}{dt} + K_1 \frac{dI_s}{dt} + K_2 \frac{dI_a}{dt} + K_3 \frac{dI_p}{dt} + K_4 \frac{dT}{dt} \tag{20}$$

$$\begin{aligned} &= -\frac{\mu}{S}(S - S_0)^2 + [K_1 x\tau + K_2 y\tau + K_3(1 - (x + y))\tau - (\tau + \mu)]E + [\beta S_0 + K_4\phi_s \\ &- K_1(\phi_s + \mu + \delta)]I_s + [\beta m S_0 + K_4\phi_a - K_2(\phi_a + \mu + c)]I_a + [\beta n S_0 + K_4\phi_p \\ &- K_3(\phi_p + \mu + \delta)]I_p + [\beta b S_0 - K_4(\alpha + \mu + \delta)]T \end{aligned} \tag{21}$$

Let us extract K_1, K_2, K_3, K_4 such that;

$$\begin{bmatrix} \beta S_0 + K_4\phi_s - K_1(\phi_s + \mu + \delta) = 0 \\ \beta m S_0 + K_4\phi_a - K_2(\phi_a + \mu + c) = 0 \\ \beta n S_0 + K_4\phi_p - K_3(\phi_p + \mu + \delta) = 0 \\ \beta b S_0 - K_4(\alpha + \mu + \delta) = 0 \end{bmatrix} \tag{22}$$

Note from the last equation of (22) that;

$$\frac{\beta b S_0}{(\alpha+\mu+\delta)} = K_4 \tag{23}$$

Putting equation (23) in the first equation of (22), we have;

$$\frac{\beta S_0[(\alpha+\mu+\delta)+b\phi_s]}{(\alpha+\mu+\delta)(\phi_s+\mu+\delta)} = K_1 \tag{24}$$

Substituting K_1 and K_4 appropriately gives;

$$\frac{\beta S_0[m(\alpha+\mu+\delta)+b\phi_a]}{(\alpha+\mu+\delta)(\phi_a+\mu+c)} = K_2 \tag{25}$$

$$\frac{\beta S_0[n(\alpha+\mu+\delta)+b\phi_p]}{(\alpha+\mu+\delta)(\phi_p+\mu+\delta)} = K_3 \tag{26}$$

With the values of these K's in mind, equation (21) is rewritten as;

$$\dot{L_0} = -\frac{\mu}{S}(S-S_0)^2 + \left[\frac{x\tau\beta S_0[(\alpha+\mu+\delta)+b\phi_s]}{(\alpha+\mu+\delta)(\phi_s+\mu+\delta)} + \frac{y\tau\beta S_0[m(\alpha+\mu+\delta)+b\phi_a]}{(\alpha+\mu+\delta)(\phi_a+\mu+c)} + \frac{(1-(x+y))\tau\beta S_0[n(\alpha+\mu+\delta)+b\phi_p]}{(\alpha+\mu+\delta)(\phi_p+\mu+\delta)} - (\tau+\mu)\right] \tag{27}$$

This simplifies to;

$$\begin{aligned}\dot{L_0} = &-\frac{\mu}{S}(S-S_0)^2 + \tau b S_0[y(\phi_p+\mu+\delta)(m(\alpha+\mu+\delta)+b\phi_a) \\ &+ (1-(x+y))(\phi_a+\mu+c)(n(\alpha+\mu+\delta)+b\phi)] \\ &+ (\tau+\mu)\left[\frac{x\tau\beta S_0-(\tau+\mu)(\phi_s+\mu+\delta)}{(\phi_s+\mu+\delta)}\right] + \frac{\beta b x\tau\phi_s S_0}{(\alpha+\mu+\delta)(\phi_s+\mu+\delta)}\end{aligned}$$

Recall that $R_1 = \frac{\beta \wedge x\tau}{\mu(\tau+\mu)(\phi_s+\mu+\delta)}$ and $S_0 = \frac{\wedge}{\mu}$, then;

$$\begin{aligned}\dot{L_0} = &-\frac{\mu}{S}(S-S_0)^2 \\ &+ \left[\begin{array}{l}\tau\beta S_0 y(\phi_p+\mu+\delta)(m(\alpha+\mu+\delta))+b\phi_a)+(1-(x+y))(\phi_a+\mu+c) \\ (n(\alpha+\mu+\delta)+b\phi_p)+\frac{\beta b x\tau\phi_s S_0}{(\alpha+\mu+\delta)(\phi_s+\mu+\delta)}+(\tau+\mu)(R_1-1)\end{array}\right] \leq 0\end{aligned} \tag{28}$$

Since it is succinctly shown that the biggest invariant subset contained in;

$$\Psi = \left\{(S,E,I_s,I_a,I_p,T,R) \in \frac{M_0}{\dot{L_0}} = 0\right\} \tag{29}$$

This is the disease-free-state (E_0) we bring the proof to a conclusion by Laselle's invariant principle.

4. Results and Discussion

Table 1

Compartments	Initial Values
Susceptible	4420
Exposed	500
Symptomatic	50
Asymptomatic	30
Presymptomatic	20
Treatment	0
Recovered	0

Table 2

Parameters	Interpretations	Value	source
$a(t)$	Percentage of arrivals that are asymptomatic	0.018-0.28	Calibrated
$e(t)$	Percentage of arrivals that are exposed	0.018-0.28	Calibrated
$p(t)$	Percentage of arrivals that are presymptomatic	0.018-0.28	calibrated
$\wedge$	Per capital recruitment rate	50	Assumed
β	Transmission rate	0.005944	Assumed
b	Infectivity factors for treatment compartment individuals	0-1	[7]
m	Infectivity factors for asymptomatic individuals	0.8	Assumed
n	Infectivity factors for presymptomatic individuals	0-1	Calibrated
α	The recovery rate of the treated individuals	0.05	[7]
τ	Progression rate from latent stage to the infectious stage	0.2	[7]
y	Percentage of exposed individuals in the community that becomes asymptomatic	0-1	Assumed

Contd...

$\delta_s, \delta_p, \delta_T$		0.08, 0.008, 0.0008	[7]
ϕ_a	The hospitalization rate of asymptomatic infectious	0-1	Calibrated
ϕ_s	The hospitalization rate of symptomatic infectious	0-1	Calibrated
ϕ_p	The hospitalization rate of presymptomatic infectious	0-1	Calibrated
x	Percentage of exposed individuals in the community that becomes symptomatic	0-1	[7,23]
c	The recovery rate of asymptomatic individuals	0.0004-0.4	Estimated
μ	Natural Mortality rate	0.005	Estimated

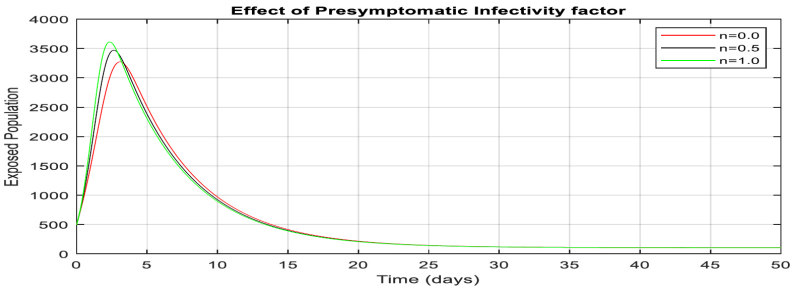


Figure 1a

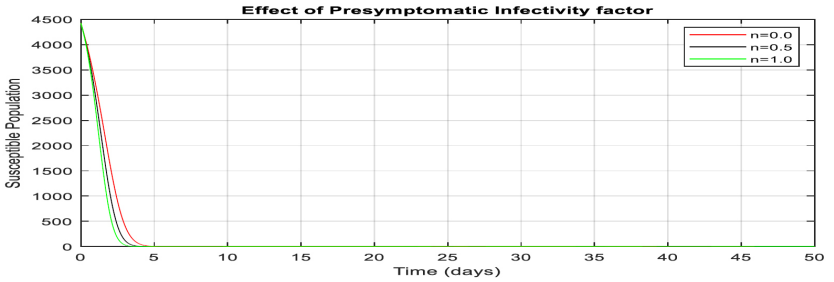


Figure 1b

Alternating the infectivity factor of presymptomatic individuals by increasing it from 0 to 1 [Table 1, Table 2] has a traceable effect on both Susceptible and Exposed populations as shown in **figure 1a and 1b.** It is revealed that if there is no transmission of Covid-19 caused by presymptomatic individuals i.e. "n=0", then the susceptible exposed

curves will be at a steady-state provided that all other conditions are intact and vice versa. The above curves show that keeping "n" at zero would eventually make the disease dies out of the population instead of being a contributing factor for the disease to invade the land for a long time.

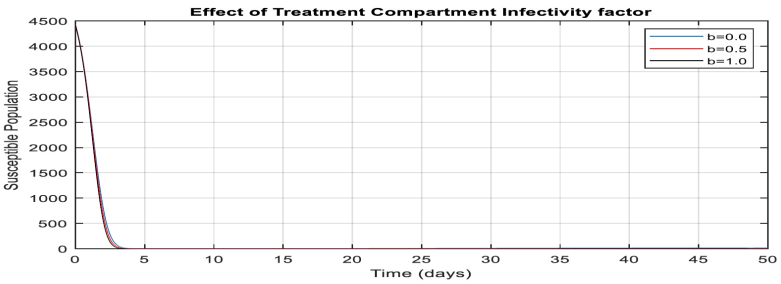


Figure 2a

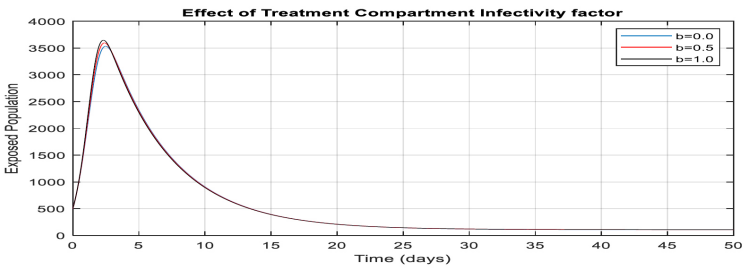


Figure 2b

The traceable effect of negligence even in the hospital or medical centers during the treatment of Covid-19 is shown in **Figure 2a and 2b**. So it is advised that all individuals take note of the preventive measures as stated by NCDC to keep the curve at DFE.

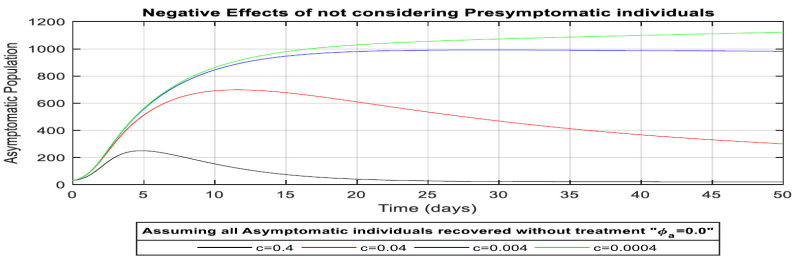


Figure 3a

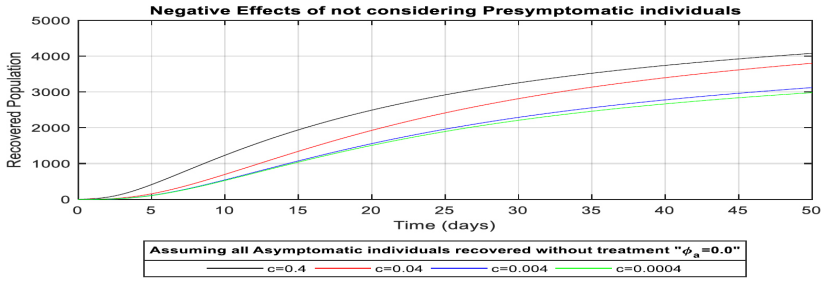


Figure 3b

When the recovery rate of asymptomatic individuals is drastically reduced; the remaining set of infectious individuals cannot be said to be asymptomatic again since they seized to recover themselves. So, this suggests the presence of presymptomatic individuals who needs to be treated by employing Remdesivir, Bamlanivimab & etesevimab, Baricitinib, Casirivimab & Imdevimab, Sotrovimab or Tocilizumab to transcend to the recovered class. So, when treatment is not administered at that stage of discovery, the asymptomatic curves hence approach endemic equilibrium. The likelihood of witnessing the second surge of COVID-19 infections is demonstrated in the above curves **Figures 3a & 3b** if presymptomatic infectious individuals are plausibly not considered in the population density. So it is strongly advised that segmentation from the exposed class should not be narrowly symptomatic and asymptomatic but presymptomatic.

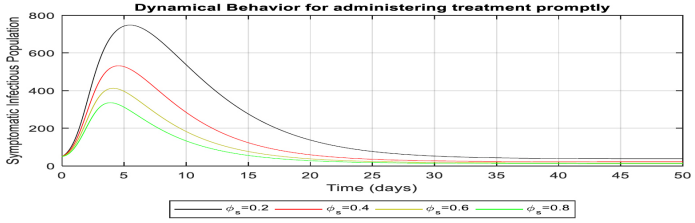


Figure 4a

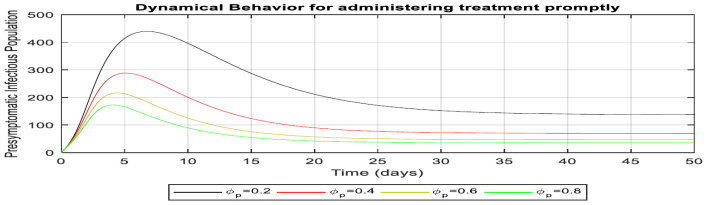


Figure 4b

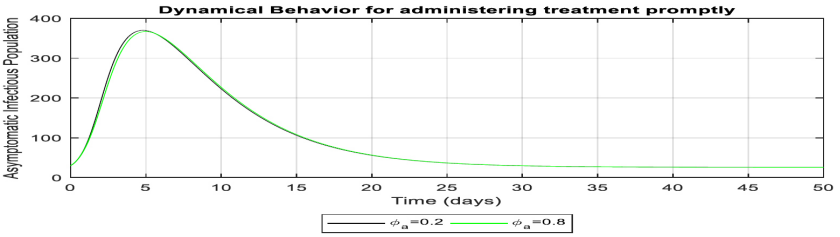


Figure 4c

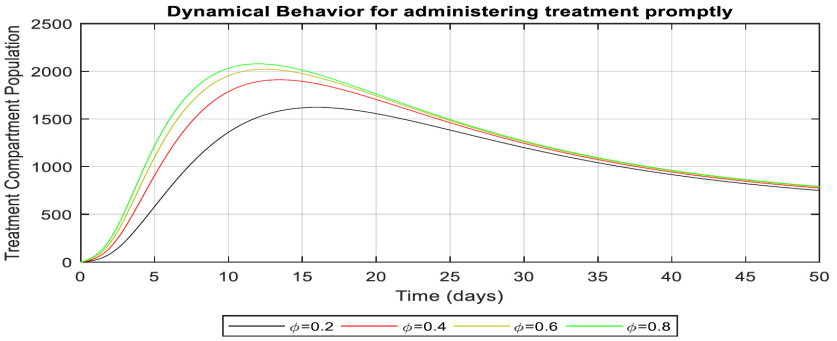


Figure 4d

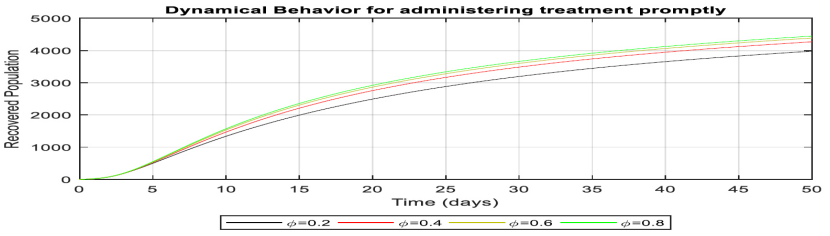


Figure 4e

Figure 4a, 4b, 4c, 4d, and **4e** demonstrates the dynamic behaviors of the population density when the treatment is increased sequentially. When treatment parameters "ϕ" " are doubled progressively with respect to the concerned compartmentalized variables $\phi_s \& \phi_p$, the corresponding effect were shown. This would be underachieved if Covid-19 drugs are not always available in all registered organizations and medical centers. Prompt usage and availability of Remdesivir, Bamlanivimab & etesevimab, Baricitinib, Casirivimab & Imdevimab, Sotrovimab or Tocilizumab in all concerned places across the globe will eventually nullify the presence of

the virus in all affected habitats. Hence, the Disease Free Equilibrium will be achieved which is one of the paramount reasons for this research.

5. Conclusion

In this study, we have carefully examined the horribly disastrous but preventable effects of failing to take presymptomatic infectious individuals in the population density into account. To do this, we proposed an unprecedented $SEI_aI_sI_pTR$ mathematical model for Coronavirus disease -19. We also proved that the system is bounded and positive. An important parameter that determines the level of invasion of infection, the control reproduction number R_0 comprising R_1, R_2, R_3 and R_4 for the population density was also determined to be less than unity. Additionally, it was determined that the model's the persistence (Endemic) and Disease Free Equilibrium point were asymptotically stabled both locally and globally, indicating that the lifespan and transitory of the Covid-19 depending if $R_0 < 1$ or $R_0 > 1$. The disease would vanish in the host population at a time t, according to the numerical solution for the global stability at DFE.

Competing Interest

The authors declared that they have no competing interest

Data Availability

It is affirmed by the authors that the data supporting these findings are available within the paper

Authors' Contribution

The authors contributed equally to the preparation of this manuscript.

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