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Mathematical recipe for curbing coronavirus (COVID-19) transmission dynamics

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1. Introduction

Coronavirus (COVID-19) is a Latin word meaning “Crown.” The name was coined by June Almeida and David Tyrell who was the first scientist to study human coronavirus in (1930) when an acute respiratory infection of domesticated chickens showed disease symptoms and was investigated to be caused by Bronchi’s virus (IBV) as reported in Animal by Ref. [1]. According to Arthur Shalks and Hawn (1931), a new finding on the respiratory infection of chickens in North Dakota was reported to be a viral disease. Human coronaviruses also known as COVID-19 are large pleomorphic spherical particles with bulbous surface projection, with a coverage diameter of 120 nm (12 μm). The diameter of the envelop is about 80 nm (0.8 μm) within the envelop is a nucleotides of protein. Coronavirus contains a positive-sense single stranded RNA genome [2]. The genome size ranges from 26.4 to 31.7 kielbasas [3]. Infection begins when the viral spike glycol proteins attaches to its complementary host cell receptors in parasitic kind of relationship. Depending on the host cell protease available cleavage and activation that allows the virus to enter the host cell by endocytosis or direct fusion of viral envelop with the host membrane, on penetrating the host cell, the viral particles are coated as the genome enters the cytoplasm. Viruses propagate through their host cells [4]. Revealed that, COVID-19 is transmitted quite efficiently, and an average infected person can...
spread the disease to two, three, or more persons with an exponential increase, respectively. It also stated that there are strong evidence that coronavirus can be transmitted by people who are ill or mildly ill as well as those who are asymptomatic patients. Coronavirus is such a threat that it can kill healthy adults or elderly people with health-related challenges even if it’s mild. The data so far suggest that COVID-19 has a case fatality risk around 1%; this rate may make it more severe than the typical seasonal influenza, many times than expected. Putting it somewhere between the then 1957 influenza pandemic at (0.6%) and the 1918 influenza pandemic at (2%) [4]. Hence, the need for all possible measures to be taken as response to this prevailing novel pandemic such as vaccination to curtail or minimize it massive wide spread around the world, so that stability can be achieved while saving lives. Thus, in Ref. [7], an enhance model for computer viruses Counter measures have been proposed. The proposed model employs a systematic transition stages of development and provides counter measures for viruses when infected else there will be reoccurrence (reinfection) and proposed an algorithm for viruses Counter measures. In addition, the Novel coronavirus varies significantly in terms of risk factor. Some viruses can kill more than 30% of those infected such as MERS-COV while some are relatively harmless such as those that cause common cold [6].

More so, the findings in Ref. [8] report that COVID-19 viral infection shows symptoms such as fever, sore throat as in common cold in severe cases damage of the respiratory tract [8]. The human coronavirus discovered in 2003, SARS-COV, which causes the severe acute respiratory syndrome (SARS) has a unique pathogenesis and can lead to both lower and upper respiratory infection [9] with possible acute kidney injury, most especially on the elderly individuals [38].

Coronavirus now widely known as COVID-19 spread more rapidly due to increase commercial activities in Wuhan. Wuhan is a large city connecting the North, South, East, and West of China mainly through railways and a major international airport. The availability of connecting flights, as well as the timing of the outbreak during the Chinese festive season (Lunar), also known as New Year and the massive rail transit located within and outside the city of Wuhan enabled the virus to pierce throughout China, and subsequently, around the world [24], respectively.

The outbreak took place precisely around December 2019, when a unique pneumonia case now coronavirus case was established, the outbreak was trace to a novel strain according to Refs. [10,11]. It was later renamed as SARS-COV2 by the international committee taxonomy of viral. After about a year of China outbreak, the virus was later introduced in Lagos Nigeria on the 27th February 2020 by an Italian Citizen that work in Nigeria as reported by the Nigerian Center for disease control (NCDC) [12,13] from where it was spread to almost the 34 out of 36 states of Nigeria including the federal capital territory (FCT-Abuja) with the total report of 5959 confirmed cases, 1594 discharged, and 182 deaths in Nigeria as at May 18th 8:00 a.m. GMT + 1 NCDC Report [13]. The rate of coverage from the period of introduction brought about the interest of this finding.
Several other research is ongoing on the novel COVID-19, some existing studies presented examples related to the dynamics of infectious disease [14]. Different kinds of models where employed, essentially considering nonlinear governing equations [14–16] described the dynamics of coronavirus infection in human, establishing interaction among human cells and the virus. The findings in Ref. [17] developed a mathematical model for calculating the transmission ability of the virus considering a simplified version of the bats-hosts-reservoir-people transmission model, defined as a reservoir-people model. Results follow the general trend of the initial propagation. While [18] also estimated the characteristics of the epidemiologic time distribution, exploiting some pattern trends of transmission propagation and interspecies jumping [19]. The authors in Ref. [20] proposed a dynamic ways of preventing virus spread using quarantine technique [21] exploiting the pattern of human-to-human transmission of novel coronavirus in Wuhan, China. A reproductive number that defines the infectious propagation and the individual variation in the number of secondary cases was established. Uncertainty quantification tools were employed to define the transmission patterns. Susceptible-exposed-infectious-recovered (SEIR) models are an interesting approach to deal with the mathematical modeling of coronavirus transmission. And Refs. [22,23] investigated Wuhan—China case, evaluating now casting and forecasting domestic and international spread outbreak, respectively.

2. Materials and methods

The population is divided into four (4) compartments which are namely as follows: Susceptible Humans (S_H), Exposed Humans (E_H), Infected Humans (I_H), and Recovered Humans (R_H). The model parameters to be incorporated are also systematically presented as follows:

The recruitment rate of human due to natural birth rate and immigration is given as \((1−P) \Pi N\).

The rate at which exposed humans get infected is also given as \((\varepsilon E)\), while the rate at which recovered humans becomes susceptible is \((\delta R)\) and the natural death rate of human is \((\mu S)\) as well as the death rate due to infection of the novel coronavirus or COVID-19 is \((\delta + \mu) I\), in addition, the rate at which a susceptible human becomes exposed is given as \([BSI]\).

To be more precise, our proposed model for this finding is presented in Section 3 as follows:

3. Proposed model

In our proposed model, vital parameters are presented into compartment (Fig. 28.1), each of which is presented as follows:

The infection compartment is \((\sigma +\mu) I\), and death for recovered human is \((\mu R)\) (Fig. 28.2).
These are all represented in Fig. 28.1, while the square represents the different compartments, the arrows represent the state transition between the compartments, respectively (Fig. 28.3).

\[ N = S + E + I + R \]  

(28.1)

\[ \frac{dS}{dt} = (1 - p)\pi N - \frac{\beta SI}{N} - \mu S + \delta R \]  

(28.2)

\[ \frac{dE}{dt} = \frac{\beta SI}{N} - (\epsilon + \mu)E \]  

(28.3)
\[
\frac{dI}{dt} = eE - (\Gamma + \sigma + \mu)I \\
\frac{dR}{dt} = P\pi N + \Gamma T - (\mu + \delta)R
\] (28.4) (28.5)

Such that
\[
\frac{dN}{dt} = (\pi - N)N - \sigma I
\] (28.6)

To dimensionalize Eqs. (28.2)–(28.5) above
\[
s = \frac{S}{N}, \quad e = \frac{E}{N}, \quad i = \frac{I}{N}, \quad r = \frac{R}{N}
\] (28.7)

From Eq. (28.7) above
\[
S = sN
\]
\[
\frac{dS}{dt} = \frac{dsN}{dt} = \frac{sdN}{dt} + \frac{Nds}{dt}
\] (28.8)

\[
\frac{ds}{dt} = \frac{I}{N} \left[ \frac{ds}{dt} - \frac{sdN}{dt} \right]
\]
Substituting Eqs. (28.2) and (28.6) into Eq. (28.8), we have:

\[
\frac{ds}{dt} = \frac{I}{N} \left[ (I - P)\pi n - \frac{\beta s I}{N} - \mu s + \delta R - s(\pi N - \mu N - \sigma I) \right]
\]

\[
\frac{ds}{dt} = (I - P)\pi - \beta si - \mu s + \delta r - \frac{s\pi N}{N} + NS + \sigma i
\]

(28.9)

It can be seen from Eq. (28.7) that

\[
\frac{de}{dt} = \frac{I}{N} \left[ \frac{dE}{dt} - \frac{edN}{dt} \right]
\]

(28.10)

Substituting Eqs. (28.3) and (28.6) into Eq. (28.10), we have

\[
\frac{de}{dt} = \frac{I}{N} \left[ \frac{dE}{dt} - \frac{edN}{dt} \right]
\]

\[
\frac{de}{dt} = \frac{I}{N} \left[ \frac{\beta s I}{N} - (e + \mu)E - e(\pi N - N - \sigma I) \right]
\]

\[
\frac{de}{dt} = \beta si - (e + \mu)e - e\pi + NE + \sigma ei
\]

(28.11)

From Eq. (28.7)

\[
\frac{di}{dt} = \frac{I}{N} \left[ \frac{df}{dt} - \frac{idN}{dt} \right]
\]

(28.12)

Substituting Eqs. (28.4) and (28.6) into Eq. (28.12), we obtained

\[
\frac{di}{dt} = e e - (r + \sigma + \mu)I - \pi i + \mu i + \sigma i^2
\]

\[
\frac{di}{dt} = e e - (r + \sigma + \pi)I + \sigma - i^2
\]

(28.13)

From Eq. (28.7)

\[
\frac{dr}{dt} = \frac{I}{N} \left[ \frac{dR}{dt} - \frac{rdN}{dt} \right]
\]

(28.14)

Substituting Eqs. (28.5) and (28.6) into Eq. (28.14), we have

\[
\frac{dr}{dt} = \frac{I}{N} \left[ P \pi N + rI - (\mu + \delta)R - r(\pi N - \mu N - \sigma I) \right]
\]

\[
\frac{dr}{dt} = P\pi + r - (\mu + \delta)r - r\pi + \mu r + \sigma ri
\]
\[
\frac{dr}{dt} = P\pi + ri - (\delta + \pi)r + \sigma \Gamma i 
\]  
(28.15)

Now considering Eqs. (28.9), (28.11), (28.13), and (28.15) as follows:

\[
\begin{align*}
\frac{ds}{dt} &= (I - P)\pi - (\beta - \sigma)si + \delta r - \pi s \\
\frac{de}{dt} &= (\beta s - \sigma e)r - (\varepsilon + \pi)e \\
\frac{di}{dt} &= \sigma i^2 + \varepsilon e - (\Gamma + \sigma + \pi)i \\
\frac{dr}{dt} &= \sigma ri + P\pi + ri - (\delta + \pi)r \\
\end{align*}
\]  
(28.16)

The above Eq. (28.16) can be written in this form as:

\[
\frac{ds}{dt} = (I - P)\pi - (\beta - \sigma)si + \delta r - \pi s 
\]  
(28.17)

\[
\frac{de}{dt} = (\beta s + \delta e)i - k_1e  
\]  
(28.18)

\[
\frac{di}{dt} = \sigma i^2 + \varepsilon e - k_2i  
\]  
(28.19)

\[
\frac{dr}{dt} = \sigma ri + P\pi + ri - k_3r  
\]  
(28.20)

Were \(k_1 = \varepsilon + \pi\), \(k_2 = +\sigma + \pi\), \(k_3 = \delta + \pi\) and \(s + eti + r = 1\)

4. Existence and uniqueness of solution of the model

From Eqs. (28.17)—(28.20), we find \(f_1\), \(f_2\), \(f_3\), and \(f_4\), respectively, as follows:

\[
F_1 = (I - P)\pi - (\beta - \sigma)si + \delta r - \pi s 
\]  
(28.21)

\[
F_2 = (\beta s + \delta e)^2i - k_1i  
\]  
(28.22)

\[
F_3 = \sigma i^2 + \varepsilon e - k_2i  
\]  
(28.23)

\[
F_4 = \sigma ri + P\pi + \Gamma i - k_3r  
\]  
(28.24)

From Eq. (28.21), we achieve

\[
\left| \frac{\partial f_1}{\partial s} \right| = |-(P - \sigma + \pi)| < \infty, \quad \left| \frac{\partial f_1}{\partial e} \right| = 0 < \infty
\]

\[
\left| \frac{\partial f_1}{\partial i} \right| = |-(\beta - \sigma)s| < \infty, \quad \left| \frac{\partial f_1}{\partial r} \right| = |\vartheta| < \infty
\]
From Eq. (28.22), we obtained
\[
\left| \frac{\partial f_2}{ds} \right| = |(\beta i) < \infty|, \left| \frac{\partial f}{de} \right| = |\Gamma i - k_1| < \infty, \left| \frac{\partial f_2}{dt} \right| = |\sigma e| < \infty, \left| \frac{\partial f_2}{dr} \right| = 0 < \infty
\]
And from Eq. (28.23), we have
\[
\left| \frac{\partial f_3}{ds} \right| = \left| \frac{\partial f_3}{dr} \right| = 0, \left| \frac{\partial f_4}{de} \right| = e < \infty, \left| \frac{\partial f_4}{dr} \right| = 0 < \infty
\]
\[
\left| \frac{\partial f_4}{ds} \right| = \left| \frac{\partial f_4}{de} \right| = 0 < \infty, \left| \frac{\partial f_4}{dr} \right| = |\sigma i - k_3| < \infty
\]

5. Stability analysis (positivity solution)

\[
\frac{ds}{dt} = (I - P)\pi(\beta - \sigma)si + \delta r - \pi s
\]
\[
\frac{ds}{dt} \leq (I - P)\pi - \pi s
\]
\[
\frac{ds}{dt} + + \pi s \leq (I - P)\pi
\]
\[
I.F = \int e^\pi dt = e^{\pi t}
\]
\[
\therefore S.I.F \leq \int (I - P)\pi ifdt + C
\]
\[
S(t)e^{\pi t} \leq \int (I - P)\pi e^{\pi t}
\]
\[
S(t) \leq \frac{(I - P)\pi e^{\pi t}}{e^{\pi t}} + \frac{C}{e^{\pi t}}
\]
\[
S(t) \leq (I - P) + Ce^{-\pi t}
\]

When \( t = 0 \)
\[
S(0) \leq (I - P) + C
\]
\[
\Rightarrow S(0) \leq (I - P) \leq C
\]

Substitute the value of \( C \) in Eq. (28.25)
\[
\frac{S(t) - (I - P)}{e^{-\pi t}} \leq C
\]
\[
\frac{S(t) - (I - P)}{e^{-\pi t}} \leq S(0) - (I - P)
\]
\[
S(t) - (I - P) \leq [S(0) - (I - P)]e^{-\pi t}
\]
\[
S(t) \leq (I - P) + [S(0) - (I - P)]e^{-\pi t}
\]

When \( t \to \infty \)
\[
S(t) \leq (I - P) + [S(0)]
\]
\[
\leq S(t) \leq (I - P) \leq I
\]
\[
\frac{de}{dt} \geq -k_1 e \text{ by separation of variables}
\]
\[
\int \frac{de}{dt} \geq \int k_1 dt
\]
Line \( \geq -k_1 t + C \)
\[
e \geq e^{-k_1 t} + C
\]
\[
e \geq Ae^{-kt}
\]
\[
A \leq e(0)
\]
\[
\therefore e \geq e(0) e^{-kt}
\]
\( t \to \infty \) \( e(t) \geq 0 \) Similarly
\[
\frac{di}{dt} = \sigma_i^2 + e^{-k_1 i}
\]
\[
\frac{di}{dt} \geq -k_2 i
\]
\[
\int \frac{di}{T} \geq \int -k_2 dt
\]
Lin \( i \) \( \geq -k_2 dt \)
\[
i \geq e^{-k_2 t} + C
\]
\[
i \geq Be^{-k_2 t}
\]
When \( t = 0 \)
\[
i \geq i(0)
\]
\[
i(0) \geq B
\]
\[
\therefore i \geq i(0)e^{-k_2 t}
\]
When \( t \to \infty \)
\[
I(t) \geq 0
\]
\[
\frac{dr}{dt} + k_3 r
\]
\[
\frac{dr}{dt} + k_3 r \geq P \pi
\]
\[
I.F = e^{k_3 t}
\]
\[
re^{k_3 t} \geq \int P \pi e^{k_3 t} + C
\]
\[ r \geq \frac{P\pi}{k_3} e^{k_3 t} + \frac{C}{e^{k_3 t}} \]

\[ r \geq \frac{P\pi}{k_3} + Ce^{-k_3 t} \text{ when } t = 0 \]  

(28.26)

\[ r(0) \geq \frac{P\pi}{k_3} + C \]

\[ \therefore C \leq r(0) - \frac{P\pi}{k_3} \]

Substituting C in Eq. (28.26), we have

\[ r \geq \frac{P\pi}{k_3} + \left[ r(0) - \frac{P\pi}{k_3} \right] e^{-k_3 t} \text{ when } t \to \infty \quad r \geq \frac{P\pi}{k_3} \geq 0 \]  

(28.27)

6. Model equilibrium point

At equilibrium point, this is the state at which there is no infection in the population.

\[ \frac{ds}{dt} = \frac{de}{dt} = \frac{di}{dt} = \frac{dr}{dt} = 0 \]

From Eq. (28.19), we have

\[ e = \frac{[k_2 - \sigma]i}{e} \]

Substituting Eq. (28.27) into Eq. (28.18), we have

\[ \beta s - \sigma \left[ \frac{k_2 - \sigma]i}{e} - k_1 \frac{k_2 - \sigma]i}{e} \right] = 0 \]

\[ \left[ \beta s + \sigma k_2 i - \sigma^2 i^2 - k_1 k_2 \right] i = 0 \]  

(28.28)

From Eq. (28.27), we have two equilibrium points.

\[ i.e \ i = 0, \ \beta s + \sigma (k_2 + k_1) i - \sigma^2 i^2 - k_1 k_2 \neq 0 = > \text{DFE} \]  

(28.29a)

\[ i \neq 0, \ \beta s + \sigma (k_2 + k_1) i - \sigma^2 i^2 - k_1 k_2 = 0 \Rightarrow \epsilon \epsilon \]  

(28.29b)

For D. F. E.

From Eq. (28.27), we have \( e = 0 \), and from Eq. (28.20), we have \( r = \frac{P\pi}{k_3} \)

From Eq. (28.17), we have \( s = \frac{(I - P)\pi + \delta r}{\pi} = \frac{(I - P)\pi + \delta p\pi}{k_3 \pi} \)

Let \( \epsilon_o \) denote the COVID-19 disease free equilibrium

\[ \text{Thus } \epsilon_o = (s_x, I_x, i_x, r_x) = \left( \frac{k_3 (I - P)\pi + \delta p\pi}{k_3 \pi}, 0, 0, \frac{P\pi}{k_3} \right) \]  

(28.30)
7. Results

7.1 Effective reproduction number (R₀)

One of the most important concerns about any infectious disease is its ability to invade a population. The basic reproductive number R₀ is a measure of the potential for disease spread in a population, and ideas that mathematical thinking has brought to epidemic theory. It represents the average number of secondary cases generated by an infected individual if introduced into a susceptible population with no immunity to the disease in the absence of interventions to control the infection. If R₀ < 1, then on average, an infected individual produces less than one newly infected individual over the course of its infection period. In this case, the infection may die out in the long run. Conversely, if R₀ > 1, each infected individual produces one more infected individuals, the infection will be able to spread in a population. A large value of R₀ may indicate the possibility or a major epidemic outbreak (Fig. 28.4).

Using the novel next generation operator technique as applied by Refs. [23,26], we optimize the effective reproduction number R₀ of our model which is the spectral radius of the next generation matrix as follows: (Fig. 28.5).

\[
F = \begin{bmatrix} 0 & \beta s \\ 0 & 0 \end{bmatrix}
\]

\[
V = \begin{bmatrix} k_1 & 0 \\ -\varepsilon & k_2 \end{bmatrix}
\]

\[
V^{-1} = \frac{1}{k_1 k_2} \begin{bmatrix} k_2 & 0 \\ -\varepsilon & k_1 \end{bmatrix}
\]

\[
V^{-1} = \begin{bmatrix} \frac{1}{k_1} & 0 \\ \varepsilon & 1 \\ k_1 k_2 & k_2 \end{bmatrix}
\]

\[
F V^{-1} = \begin{bmatrix} \frac{\beta s \varepsilon}{k_1 k_2} & \frac{\beta \varepsilon}{k_2} \\ 0 & 0 \end{bmatrix}
\]

\[
R_0 = p(FV^{-1}) = \frac{\beta s \varepsilon}{k_1 k_2} = \frac{\beta \varepsilon k_5 (I - P)\pi + \delta P \pi}{k_1 k_2 k_3 \pi}
\]

\[
R_0 = \frac{\beta \varepsilon k_3 (I - P) + \delta P}{k_1 k_2 k_3 \pi}
\]
FIGURE 28.4 Graph of infectious humans against treatment rate. It shows that the population of infected humans decreases as results of vaccination.

FIGURE 28.5 Graph of removed/recovered individuals increases as the treatments increases. It is observed that population of the recovered humans increases as the treatment for the infected humans increases.
\[ R_0 = \frac{\beta e k_3 (1 - P) + \delta P}{k_1 k_2 k_3} \]  

(28.31)

Therefore, since \( R_0 < 1 \), the infected persons can be wiped out because one infected individual will produce less than one infected individual.

### 7.2 Existence of endemic equilibrium

The equilibrium state with the presence of infection is known as endemic equilibrium state which is shown as follows: (Fig. 28.6).

\[ \square \neq 0. \]

That implies that, there is infection in the population or existence of the diseases in the community or population.

\[ \beta e \delta + \sigma (k_2 + k_1)i - \sigma i^2 - k_1 k_2 = 0 \]

From Eq. (28.17), we make \( S \) the subject

\[ S = \frac{(I - P)\pi + \delta r}{\beta - \sigma + \pi} \]  

(28.32)

From Eq. (28.20), make \( r \) the subject of the formula

\[ r = \frac{p\pi + ri}{k_3 - \sigma i} \]  

(28.33)

Substituting Eq. (28.33) into Eq. (28.32), we have

![Graph of totally infected humans or individuals against time for different contact rate. It is observed the population of the infected humans decline as a result of vaccine.](image)

**FIGURE 28.6** Graph of totally infected humans or individuals against time for different contact rate. It is observed the population of the infected humans decline as a result of vaccine.
\[
S = \frac{(I - P)\pi + \delta}{\beta - r + \pi} \left[ \frac{p\pi + ri}{k_3 - \sigma i} \right]
\]

Substitute Eq. (28.34) into Eq. (28.29b), we obtained as follows:

\[
\beta e \left[ \frac{\pi[k_3(I - P) + \delta p]}{k_3[\beta - \sigma + \pi] - \sigma[\beta - \sigma + \pi]} \right] + \sigma(k_2 + k_1)i - \sigma^2i^2 - k_1k_2 = 0
\]

let \( A = k_3[\beta - \sigma + \pi] \) and \( B = \sigma[\beta - \sigma + \pi] \)

\[
\beta e[k_3(I - P) + \delta p] + [\delta r - \pi(I - P)\sigma]i + [A - Bi] [k_2 + k_1]\sigma i - \sigma^2i^2[A - Bi] - k_1k_2[A - B] = 0
\]

\[
\beta e[k_3(I - P) + \delta p] + [\delta r - \pi(I - P)\sigma]i + A\sigma[k_2 + k_1]i - Br[k_2 + k_1]i^2 - A\sigma^2i^2 + \beta\sigma i^2 - k_1k_2A
\]

\[
+ \beta k_1k_2\delta
\]

\[
\beta\sigma^2i^2 - i^2\sigma(p[k_1 + k_2] + A\sigma) + i[\beta e(\delta r - \pi(I - P)\sigma) + A\sigma[k_2 + k_1] + \beta k_1k_2 + \beta e\pi][k_3(I - P) + \delta p]
\]

\[
- k_1k_2A
\]

\[
a_3 = \beta\sigma^2 \Rightarrow \sigma^3[\beta - \sigma + \pi]
\]

\[
a_2 = -\sigma^2[\beta k_2 + k_1] + A\sigma \Rightarrow -\sigma^2[B(k_2 + k_2 + k_3)]
\]

\[
a_1 = \beta e[r - \pi(I - P)\sigma] + A\sigma[k_2 + k_1] + \beta k_1k_2
\]

\[
a_0 = \beta e\pi[k_3(I - P) + \delta p] - k_1k_2k_3[\beta - \sigma + \pi]
\]

\[
a_0 = k_1k_2k_3[Ro - I] - k_1k_2k_3[\beta - \sigma]
\]
7.3 Local stability of the model

This shows the rate at which the infection becomes stable at a given time

\[
(P_0) = \begin{pmatrix}
S & e & i & r \\
-\pi & 0 & -r & 0 \\
0 & -k_1 & \beta s & 0 \\
0 & E & -k_2 & 0 \\
0 & 0 & r & -k_3
\end{pmatrix}
\]

7.4 The characteristics equation is given as follows

\[ (\lambda + \pi)(\lambda + k_3)[\lambda^2 + b_1 \lambda + b_0] = 0 \]

When \( b_1 = k_1 + k_2 \) and \( b_0 = k_1 k_2 - \beta \epsilon \sigma r \)

\[
\Rightarrow k_1 k_3 \left[ 1 - \frac{\beta \epsilon \sigma r}{k_1 k_2} \right] = k_1 k_2 [1 - R_0].
\]

7.5 Numerical simulation results and discussion

We have presented numerical solutions of the model for different initial population sizes using the various values of the parameters stated in Table 28.1 and to validate that these solutions are in agreement with the qualitative behaviors of the model obtained in Section 2 in general. We choose different initial population sizes which are presented as follows: (Table 28.2).

| Parameters | Symbols | Hypothetical values | References |
|------------|---------|---------------------|------------|
| Rate of vaccine coverage | \( P \) | (0,1) | Usman et al. [34] |
| Recruitment rate | \( \Pi \) | 0.1 | Yano [35] |
| Disease transmission coefficient | \( \beta \) | 0.8 | Ibrahim and Ismail [36] |
| Natural death rate | \( \mu \) | 0.02 | Ibrahim et al. [37] |
| Rate of loss of vaccine induced immunity | \( \delta \) | (0.1) | Usman et al. [34] |
| Progression rate from E to I class | \( \epsilon \) | 0.1 | Yano [35] |
| Recovery rate | | 0.03 | Makinde [32] |
| Disease induced death rate | \( \sigma \) | 0.09 | Rahman and Zou [33] |
8. Discussion

The occurrence, spreading and control of infectious disease using mathematical models have aid in the world of health sciences. The findings from this research shows that coronavirus or COVID-19 disease spread will continue to increase if the number of susceptible human or expose human increases, which shows that the practical act of social distancing as contact rate is limited; a similarly finding was reported by Ref. [24]. The results further proved that vaccines will be more effective in curing than the treatment, although the importance of treatment for the already infected cannot be over emphasize; since, the population of infected will decrease due to vaccination as well as the recovered increases when treated as reported in Refs. [25,26]. Although the later side effects of treatments on the recovered humans may become an issue which is in accordance with the findings of [27,29,30], as such the decline of infected persons as a result of vaccination as shown in this findings is significant against time and is in alliance with the finding of [31,39] using mathematical modeling approach.

9. Conclusion

We have proposed a mathematical model as a recipe to curtail the transmission dynamic of the novel coronavirus (COVID-19) in Nigeria. The disease free equilibrium state (E₀), endemic equilibrium state as well as the effecting reproduction number (R₀) was obtained. The simulation results depicted in Figs. 3.1–3.5 and the proposed analytical model, publicized that for the disease to be totally eradicated, R₀ < 1. This is hardly possible, as to vaccinate 75% of both infected humans and susceptible humans as well as 75% prevention practices is rarely possible in any population. However, the recipe for the disease menace is to curb the disease to a bearable minimum by vaccinating more of infected humans. Future work will try to address the use and inferences of internet of things (IoT) technologies for mapping the spread of infection, with active measures needed to be taken to curb the unpredictable risk of continuing outbreaks of infectious disease as the novel COVID-19.

| Case | S₀ | e₀ | i₀ | r₀ | X | B | c | δ | P | R₀ | Comments |
|------|----|----|----|----|---|---|---|---|---|----|-----------|
| 1    | 0.5| 0.25| 0.1| 0.15| 0.1| 0.8| 0.1| 0.03| 0.09| 0.01| 0.25| 1.4050 E₁ | Stable (no disease eradication) |
| 2    | 0.5| 0.25| 0.1| 0.15| 0.1| 0.8| 0.1| 0.03| 0.09| 0.01| 0.5  | 0.9917 E₃ | Stable (disease eradication)   |
| 3    | 0.5| 0.25| 0.1| 0.15| 0.1| 0.8| 0.1| 0.03| 0.09| 0.01| 0.75 | 0.5785 E₃ | Stable (disease eradication)   |
| 4    | 0.5| 0.25| 0.1| 0.15| 0.1| 0.8| 0.1| 0.03| 0.09| 0.01| 0.9  | 0.3306 E₃ | Stable (disease eradication)   |

E₀, Disease Free Equilibrium; E₁, Endemic Equilibrium.

Table 28.2  Table of values of the parameters.
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