



Transmission Dynamics of SARS-CoV-2(COVID-19) with Vaccine Preventive Measures among Nigerian Populace Using Mathematical Modeling

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Authors' contributions

This work was carried out in collaboration between both authors. Author OTO designed the activity of medicinal plants on infectious diseases for prevention, controlling and treatment of infectious diseases, author AOB designed the materials and methods using first-order non-linear ordinary differential equations and numerical simulations of mathematical modeling, authors MA and GMC helps to proof read the manuscript for constrictive criticism. All authors read and approved the final manuscript.

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ABSTRACT

SARS-CoV-2(COVID-19) is a very infectious symptomatic and asymptomatic infection caused by Severe Acute Respiratory Syndrome Corona virus 2 (SAR-COV-2). COVID-19 is caused by the SARS-CoV-2 virus, which spreads between people, mainly when an infected person is in close contact with another uninfected person. In this research work, a mathematical model for the novel

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COVID 19 viral infectious disease which is ravaging the world today including Nigeria was considered. The mathematical model consists of four different compartments namely, susceptible, infected, vaccine and recovered under convex incident rate. The formulation of the mathematical model and some qualitative aspects for the model including the basic reproductive number of the model, existence of equilibria and its stability results by using various tools of nonlinear analysis were done. On the basis of simulations using Matlab Software package. Dynamical behaviour were observed in the mathematical model due to the vaccine of susceptible and infected individuals or one of those two classes of individuals. The x – rays features of this paper is to formulate and analyse a mathematical model that extends and complements the ones in the literature by incorporating the vaccination class denoted by $V(t)$. Mathematical models are widely used to examine, explain, and state the relevance of the use of vaccines to prevent the spread of the dynamics of infectious diseases (COVID 19) transmission among the Nigerian populace.

Keywords: *Mathematical model; vaccine; SARS-CoV-2(COVID-19); MATLAB; ordinary differential equations; basic reproductive number; simulation.*

1. INTRODUCTION

Coronavirus 2 disease 2019 (COVID-19) is a very Contagious symptomatic infection caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). It has an animal based origin from Bats, Betacoronavirus and Sarbecoronavirus together with two bat-derived strains, it is ravaging infection which has destroyed millions of life worldwide.

The transmission dynamics of SARS-CoV- 2 (CO VID 19) infection will be discussed in details in this section of the research work. COVID-19 is caused by the SARS-COV-2virus, which spreads between people, mainly when an infected person is in close proximity/ contact with another un infected person. The virus can spread from an infected person's mouth, nose in small liquid droplets when they coughing, sneezing, speaking, breathing heavily. These droplets are different sizes, ranging from larger „respiratory droplets“ to smaller „aerosols“. Person to person can be infected with COVID-19 when the virus gets into their mouth, nose or eyes, which is more likely to happen when people are in direct or close contact (less than 1 metre apart) with an infected person [1].

Aerosol transmission can occur in particularly in indoor, crowded places and inadequately ventilated spaces, where infected person(s) spend long periods of time with others, such as restaurants, choir practices, fitness classes, nightclubs, offices, places of worship etc [2].

The virus can also spread after infected people sneeze or cough on, or touch surfaces, or objects, such as tables, doorknobs and handrails. Other people may become infected by

touching these contaminated surfaces, then touching their eyes, noses or mouths [3].

However, transmission of SARS-CoV-2 occurs primarily between people through direct, indirect, or close contact with infected people through infected secretions such as saliva and respiratory secretions, or through their respiratory droplets, which are expelled when an infected person coughs, sneezes, talks [4].

The exposure to SARS-CoV-2(COVID-19) infection can either be symptomatic (with symptoms) or asymptomatic (without symptoms), studies has shown that they are more deadly during the asymptomatic level. Whether or not they have symptoms, infected people can be contagious and the virus can spread from them to other people (horizontal transmission). Infected people appear to be most infectious just before they develop symptoms (namely 2 days before they develop symptoms) and early in their illness(onset of infection). People who develop severe disease can be infectious for longer [5]

It should be noted that while someone who never develops symptoms can transmit the virus to others, it is still not clear how frequently this occurs and more research is needed in this area. The difference is that „asymptomatic“ refers to people who are infected but never develop any symptoms, while „pre-symptomatic“ refers to infected people who have not yet developed symptoms but go on to develop symptoms later [5].

Moreover, people are in close proximity to one another for long periods of time, may increase the risk of transmission. Indoor locations, where there is poor or no ventilation the possibility of

developing a viral vaccination for the SARS-CoV-2(COVID 19)infection is high and a subject of discussion.

Some people and country have developed theirs while some may be very skeptical about it due to some numerous factors which cannot be discussed during the scope of this research work. But the development of vaccine is inevitable and effort is ongoing to develop veritable vaccine both for prevention and cure of SARS-CoV-2(COVID 19).

Vaccination is a simple, safe, and effective way of protecting people against harmful diseases, before coming into contact with the infection. It uses the body's natural defenses to build resistance to specific infections and makes immune system stronger. Vaccines train the immune system to create antibodies, before exposed to a disease [6].

Mathematical modelling of transmission dynamics of infectious diseases with the presence of vaccines is now ubiquitous [7]. A series of mathematical models has been investigated by many researchers to describe the interactive dynamics of infectious diseases [8]. We proposed here a deterministic ordinary differential equations model that can represent the overall dynamics of the novel coronavirus or SARS CoV- 2.

We divided the total human population into four compartments namely, susceptible individuals, infected individuals, vaccine individuals and recovered individuals. The total size of the population is $N(t) = S(t) + I(t) + V(t) + R(t)$. We construct the deterministic model under convex incidence rate which is assumed to be a convex function with respect to the infective class due to the host population.

The benefits of using convex incidence rate is that it corresponds to an increased rate of infection because of two exposures over a small time period: a single contact produces infection at the rate $\beta_1(t)S(t)$ [9]. while the new infective individuals arise from double exposures with $\beta_2(t)S(t)$. It produces further chance that, the recovered individual again may catch infection due to the wave of the vaccine.

Here, we remark that the function

$$\Phi(I, S) = \beta I(t)S(t)(1+\tau I(t))$$

Where both β and

τ are positive constants [10]. The flow chart or the transmission diagram of the mathematical model is shown in Fig. 1.

The dynamics of the population are described by the following differential equations

$$\frac{dS}{dt} = \Lambda - \beta S(t)I(t)(1+d(t)) + \alpha R(t) - (\mu + \omega)S(t); \quad (1)$$

$$\frac{dI}{dt} = \beta S(t)I(t)(1+d(t)) - (a + \mu + \delta - b)I(t); \quad (2)$$

$$\frac{dV}{dt} = (a + \delta - b)I(t) + \omega S(t) - (\mu + \gamma)V(t); \quad (3)$$

$$\frac{dR}{dt} = \gamma V(t) - (\mu + \alpha)R(t). \quad (4)$$

Hellewell et al. [11] showed that (for most instances) the spread of COVID-19 can be effectively contained in 3 months if contact-tracing and isolation are highly effective. Furthermore, using another stochastic model to study the COVID-19 trajectory in the Wuhan city of China from January to February, 2020, Kucharski et al. [12] showed that a reduction in COVID-19 transmission can be achieve when travel restrictions are implemented. Using a model for assessing the effect of mass influenza vaccination on the spread of COVID-19 and other influenza-like pathogens co-circulating during an influenza season, Li et al. (2020) showed that increasing influenza vaccine uptake (or enhancing the public health interventions) would facilitate the management of outbreaks of respiratory pathogens circulating during the peak flu season [10,13,14]. Recently, Iboi et al. [15] developed a mathematical model to determine whether or not a hypothetical imperfect vaccine can lead to the elimination of COVID-19 in the United States. Their study showed that such elimination is feasible, using the hypothetical vaccine with assumed efficacy of 80%, if the vaccine coverage is high enough to achieve herd immunity. In particular, the vaccine coverage needed to achieve herd immunity in US is 90%, while the computed herd threshold for the states of New York and the state of Florida are 84% and 85%, respectively.

2. EQUILIBRIUM STATES OF THE MATHEMATICAL MODEL

The mathematical model (1) – (4) exhibits two states of equilibrium i.e disease free equilibrium and the endemic equilibrium such that [16-18]

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dV}{dt} = \frac{dR}{dt} = 0 .$$

$$\Lambda - \beta S(t)I(t)(1+d(t)) + \alpha R(t) - (\mu + \omega)S(t) = 0; \quad (5)$$

$$\beta S(t)I(t)(1+d(t)) - (a + \mu + \delta - b)I(t) = 0; \quad (6)$$

$$(a + \delta - b)I(t) + \alpha S(t) - (\mu + \gamma)V(t) = 0; \quad (7)$$

$$\gamma V(t) - (\mu + \alpha)R(t) = 0. \quad (8)$$

3. RESULTS

(i) Disease free equilibrium (DFE) i.e. in the absence of any infection ($I = 0$), then the disease free equilibrium points are

$$H_0(S^*, I^*, V^*, R^*) = \left(\frac{\Lambda}{\mu + \omega}, 0, 0, 0 \right) \quad (9)$$

(ii) The Endemic Equilibrium state (EE) i.e. in the presence of the infection ($I \neq 0$), we obtain;;

$$S^* = \frac{\Lambda \alpha \beta (1 + d^*) + \alpha [(a + \mu + \delta - b)(\mu + \omega) - \Lambda \beta + \beta I^* (a + \mu + \delta - b - \Lambda \tau) + (a + \mu + \delta - b) \beta d^{*2}]}{\alpha \beta (1 + d^*) (\beta I^* + \beta d^{*2} + \mu + \omega)} \quad (10)$$

$$V^* = \frac{(\mu + \alpha) [(a + \mu + \delta - b)(\mu + \omega) - \Lambda \beta + \beta I^* (a + \mu + \delta - b - \Lambda \tau) + (a + \mu + \delta - b) \beta d^{*2}]}{\alpha \beta \gamma (1 + \tau I^*)} \quad (11)$$

Table1. Definitions of parameters and variables with hypothetical values

Parameters	Definition	Hypothetical values	Source
A	Rate of net inflow of susceptible individuals	0.1132	Assumed
β	Contact rate of transmission of the corona virus	0.02	Assumed
a	Rate of prevention of the infected individual of spreading the corona virus via vaccine	0.0205	Assumed
τ	Rate of infection of the corona virus in the country	0.326	Assumed
α	Rate of recovered individuals lose immunity	0.0001	Assumed
μ	Natural death rate for all the individuals	0.2	[19]
ω	Rate at which susceptible individuals are vaccine	0.09	Assumed
δ	Death rate due to corona virus infection	0.0124	Assumed
b	Rate at which infected individuals are isolated by the government officials	0.1	Assumed
γ	Rate at which infected individuals are vaccine	0.5	1Assumed
S_0	Initial fractional value of the Susceptible Individuals	0.9951	Estimated
I_0	Initial fractional value of the Infected Individuals	0.0006634	Estimated
V_0	Initial fractional value of the Vaccinated Individuals	0.003949	Estimated
R_0	Initial fractional value of the Recovered Individuals	0.0003159	Estimated
N_0	Initial fractional value of the population	1.0000	Estimated

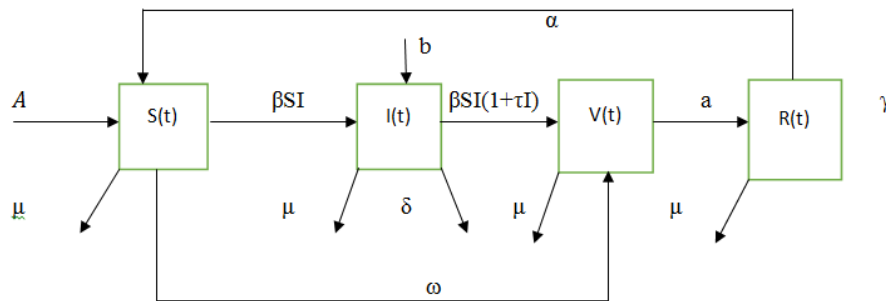


Fig 1. Transmission diagram of the mathematical model

$$R^* = \frac{(a + \mu + \delta - b)(\mu + \omega) - \Lambda\beta + \beta I^*}{\alpha\beta(1 + \tau I^*)} \quad (12)$$

For I* is the positive root of the equation $AI^* + BI^* + CI^* + DI^* + E = 0$ where;

$$A = \alpha\beta\tau\gamma\beta\tau (a + \delta - b) - \beta\tau(\mu + \alpha)(\mu + \gamma)\beta\tau \quad (13)$$

$$(a + \mu + \delta - b)$$

$$B = \alpha\beta\tau\gamma\beta(a + \delta - b) + \alpha\beta\gamma\beta(a + \delta - b) + \beta\tau(\mu + \alpha)(\mu + \gamma)\alpha\beta\tau - \beta(\mu + \alpha)(\mu + \gamma)$$

$$\beta\tau(a + \mu + \delta - b) - \beta\tau(\mu + \alpha)(\mu + \gamma)\beta(a + \mu + \delta - b)$$

$$C = \alpha\beta\tau(\mu + \alpha)(a + \delta - b) + \alpha\beta\gamma\beta(a + \delta - b) + \beta(\mu + \alpha)(\mu + \gamma)\alpha\beta\tau - \beta\tau(\mu + \alpha)(\mu + \gamma)\beta\tau$$

$$\gamma\omega\alpha\beta\tau(a + \mu + \delta - b) - \beta(\mu + \alpha)(\mu + \gamma)\beta\tau(a + \mu + \delta - b)$$

$$D = \beta(\mu + \alpha)(\mu + \gamma)(\mu + \alpha)(a + \mu + \delta - b) - \beta(\mu + \alpha)(\mu + \gamma)\beta\tau(a + \mu + \delta - b)$$

$$(\mu + \alpha)(\mu + \gamma)\alpha\beta\tau\gamma\omega\alpha\beta\tau(a + \mu + \delta - b) + \gamma\omega\alpha\alpha\beta\tau$$

$$E = (\mu + \omega)(\mu + \alpha)(\mu + \gamma)(\mu + \omega)(a + \mu + \delta - b) + \gamma\omega\alpha\Lambda\beta - \Lambda\beta(\mu + \omega)(\mu + \alpha)(\mu + \gamma) - \gamma\omega\alpha(\mu + \omega)(a + \mu + \delta - b)$$

In which $S^* > 0$, $V^* > 0$ and $R^* > 0$ for $I^* > 0$. With these values for S^* , V^* , I^* and R^* , the positivity and uniqueness of the endemic equilibrium are guaranteed if and only $R_0 > 1$ where R_0 is the basic reproductive number of the mathematical model of equations (1) – (4) given in the form;

$$F = \begin{pmatrix} \beta S_0 & 0 \\ a + \delta - b & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} a + \mu + \delta - b & 0 \\ 0 & \mu + \gamma \end{pmatrix} \quad (14)$$

Hence, for the mathematical model (1) – (4), by simple calculation, we have,

$$FV^{-1} = \begin{pmatrix} \frac{\beta S_0}{a + \mu + \delta - b} & 0 \\ \frac{\beta S_0}{a + \mu + \delta - b} & 0 \end{pmatrix}$$

Where

$$S_0 = \frac{\Lambda}{(\mu + \omega)} \quad (15)$$

Hence, the basic reproduction number R_0 (13, 14) is given in the form;

$$R_0 = \frac{\beta\Lambda}{(\mu + \omega)(a + \mu + \delta - b)} \quad (16)$$

3.1 Stability Analysis of the Disease Free Equilibrium (DFE)

Theorem 1: The Disease Free equilibrium point $H_0(S^*, I^*, V^*, R^*) = \left(\frac{\Lambda}{\mu + \omega}, 0, 0, 0\right)$ exists and is locally asymptotically stable for $R_0 < 1$, otherwise unstable.

Proof: To determine the local stability of the disease free equilibrium point, we compute the Jacobian matrix of the mathematical model equations (1) – (4) around the DFE H_0 is given by

$$J(H_0) = \begin{pmatrix} -(\mu + \omega) & -\beta S_0 & 0 & \alpha \\ 0 & \beta S_0 - (a + \mu + \delta - b) & 0 & 0 \\ \omega & a + \delta - b & -(\mu + \gamma) & 0 \\ 0 & 0 & \gamma & -(\mu + \alpha) \end{pmatrix} \quad (17)$$

The Characteristic equation of $J(H_0)$ corresponding to the eigenvalue λ is $\det(J(H_0) - \lambda I_4) = 0$ (16,17,18,19). From the characteristic equation, the eigenvalues are given in the following forms:

$$\lambda_1 = -\mu, \quad (18)$$

$$\lambda_2 = -a - \mu - \delta + b + \beta \left(\frac{\Lambda}{\mu + \omega} \right), \quad (19)$$

$$\lambda_3 = \frac{1}{2}\alpha - \frac{1}{2}\gamma - \mu - \frac{1}{2}\omega + \frac{1}{2}\sqrt{\alpha^2 - 2\alpha\gamma - 2\alpha\omega + \gamma^2 - 2\gamma\omega + \omega^2} \quad (20)$$

$$\lambda_4 = \frac{1}{2}\alpha - \frac{1}{2}\gamma - \mu - \frac{1}{2}\omega - \frac{1}{2}\sqrt{\alpha^2 - 2\alpha\gamma - 2\alpha\omega + \gamma^2 - 2\gamma\omega + \omega^2} \quad (21)$$

From the above eigen values, the eigenvalues are real and negative since all the parameters are positive. Hence, disease free equilibrium point (DFE) is locally asymptotically stable if $R_0 < 1$ and unstable for $R_0 > 1$.

3.2 Numerical Simulations

Numerical simulations [20,21] were carried out to graphically explain the importance of vaccines as protective measure to reduce the spread of covid-19 in the world generally. In order to support the analytical results, graphical representations showing the time graphs of combination of two state variables are provided. Fig. 1 is the diagram showing the dynamics of the fractions of the infected population and the vaccine population. As the number of vaccine population increases with time due to the outbreak of the infectious disease, it got to a time that the number begins to reduce such that the

effect of the vaccine has been felt by the population that has been infected with the infectious disease.

Fig. 2 shows the graph of the fractions of the susceptible individuals and the vaccine populations. Observe that the proportion of the susceptible population reduces as the vaccines are given to the population in order to protect the individuals in contacting or spreading the SARS-CoV-2(COVID – 19) in the society.

Fig. 3 shows that as the proportion of the infected individuals reduces, the proportion of the recovered individuals increases. This is due to the effect of the vaccine that has been received by the susceptibles before contacting the covid 19 thereby giving room to receive proper treatment from the health officials and this makes the individuals to recover at faster rate from the infectious disease.

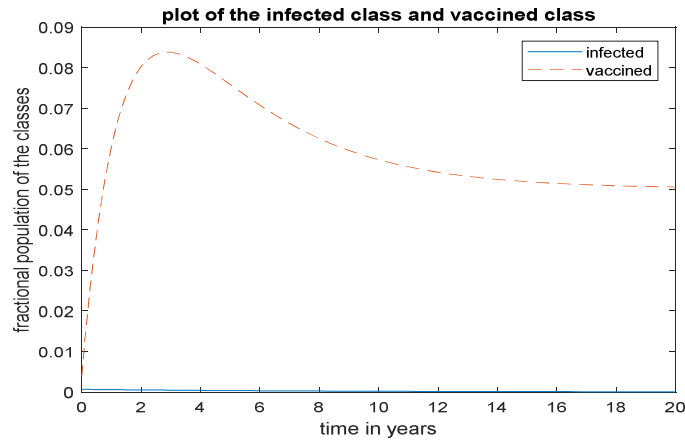


Fig. 2. Plot of the infected class and the vaccine class

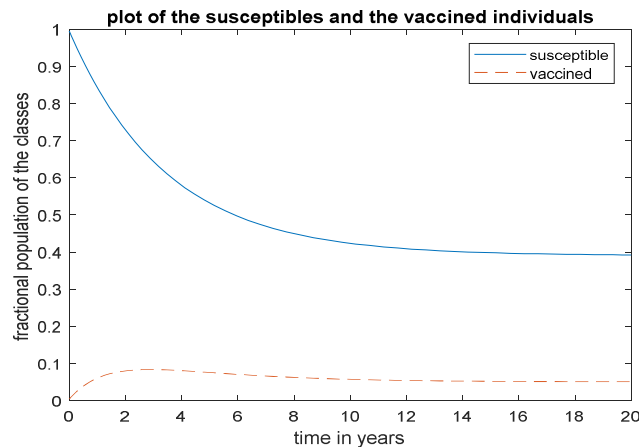


Fig.3.Plot of the susceptible and the vaccine individuals



Fig.4.Plot of the infected and recovered classes

4. DISCUSSION

SARS-CoV-2(COVID 19) pandemic, vaccination continues to be critically important. The pandemic has caused a decline in the number of children receiving routine immunizations, which could lead to an increase in illness and death from preventable diseases. WHO has urged countries to ensure that essential immunization and health services continue, despite the challenges posed by COVID-19 [22].

Many studies have focused on characterizing the heterogeneity of Susceptibility of SARS-CoV-2(COVID 19) in terms of demographics, with clear evidence of higher mortality in men and older individuals. The adaptive immune system, including both B and T cells, has recently been recognized to play a critical role in providing preexisting immunity to SARS-CoV-2 [23,24].

Studies have highlighted mechanisms that protect against severe symptoms but have not revealed factors that predispose to mortality. Consequently, acquired immune responses to prior infections may account for a large percentage of the variability in disease presentation, although questions remain about additional determinants of disease, such as preexisting comorbidities. Host genetic risk factors have also emerged as a potential explanation for clinical heterogeneity and additionally offer the potential for understanding molecular pathways for tailored therapeutic intervention [25].

Small-scale studies have implicated the type I interferon (IFN) pathway as protective against SARS-CoV-2 [26].

SARS-CoV-2 [26].The type I IFN pathway plays a crucial role in mediating innate immune responses to viral infections. This family of cytokines is comprised of 13 IFN- α subtypes, IFN- β , IFN- ω , IFN- κ , and IFN- ϵ , which all signal through the heterodimeric IFN I receptor, composed of IFN- α/β receptor 1 (IFNAR1) and IFNAR2. In host cells, type I IFNs are expressed at low amounts, poised to combat infections. Upon infection, they are rapidly produced by immune cells, such as macrophages and dendritic cells, to limit the spread of pathogens [27].

In addition, type I IFNs induce the expression of several hundred interferon stimulated genes that can further limit pathogen replication through various mechanisms. However, this typically protective immune response can, when over activated, lead to autoimmune diseases. Conversely, loss-of-function variants in genes encoding members of the type I IFN pathway lead to severe immunodeficiencies characterized by life-threatening viral infections. Recently, multiple studies demonstrated that impaired type 1 IFN responses may be a hallmark of severe COVID-19 [28,29] but why this pathway was suppressed remained unclear.

Casanova et al. [30] identified neutralizing auto-antibodies as another potential cause of severe COVID-19. Auto-antibodies recognize and thereby may inhibit host proteins; they are a hallmark of many autoimmune diseases and are thought to be a contributor to autoimmune pathophysiology.

Neutralizing auto-antibodies against type I IFNs, mostly IFN- $\alpha 2$ and IFN- ω , were found in up to

13.7% (135/987) of patients with life-threatening COVID-19 and were shown to neutralize activation of the pathway *in vitro*. By contrast, these auto-antibodies were not present in 663 patients with asymptomatic or mild COVID-19 and were only found in 0.33% (4/1227) healthy individuals not exposed to SARS-CoV-2. The presence of neutralizing auto-antibodies correlated with low serum IFN- α concentrations. Auto-antibodies against type I IFNs were also detected in blood samples of some patients obtained before SARS-CoV-2 infection, indicating that their production was not triggered by the virus in those patients. Notably, inactivating auto-antibodies were identified primarily in males (94%) and may be a cause of the higher male-specific disease mortalities [30,31].

What remains unknown are the contributions of genetic variation outside of the type I IFN pathway for defense against SARS-CoV-2 infection. Additionally, although Zhang et al. [31] focused on rare germline variation, the roles of common single-nucleotide polymorphisms (SNPs) and acquired somatic mutations in immune cells, which accumulate with age, need to be investigated. Further comprehensive genetic studies could also help provide insights into the potential contribution of deleterious variation in the severe SARS-CoV-2-associated multisystem inflammatory syndrome in children [31,32].

Although the studies of Zhang et al. [31] and Bastard et al. [33] illuminates the importance of pathways responsible for clearing infections, it is also possible that proinflammatory variants may either reduce or enhance disease severity. Why some patients who carry pathogenic variants in innate immune genes, such as IFN-related genes, remain asymptomatic until their exposure to a specific pathogen is likely explained by the presence of other genetic modifying alleles or epigenetic factors.

The recovery time of most people who are infected with SARS-CoV-2(COVID-19) infection will have only mild illness. Mild COVID-19 cases still can make feel nausea. Scientists and researchers are constantly tracking infections and recoveries. But they have data only on confirmed cases; it is difficult to count people that are infected with COVID-19. Experts also don't have information about the outcome of every infection. However, early estimates predict that the overall COVID-19 recovery rate is between 97% and 99.75% [32].

The post-recovery time of SARS-CoV-2(COVID-19) infection is stated here, Not all patient who were infected with SARS-CoV-2(COVID-19) infection will notice symptoms. If noticed, it may show up 2 to 14 days after your infection. And those symptoms can vary from one person to the next. One of the most common signs is a fever, which for most adults is 100.4 F or higher. It means your body is trying to fight off an invader. About 50% of people who become ill have a dry cough. The kind that does not bring up any mucus or phlegm. But about a third day a cough with mucus. The patient might feel very tired. Less commonly, the throat may be sore and head might ache. muscles and joints could hurt, and might get chills, nausea, vomiting, or diarrhea [33]. Some people who had COVID-19 said they had trouble taking deep breaths and felt like they had a tight band wrapped around their chest. Others have likened the illness to a bad cold. Loss of smell and taste have been reported in many cases. Some patients have skin rashes and darkened toes, called "COVID toes" and short of breath [33].

The pre-recovery time for SARS-CoV-2(COVID-19) infection suggested that it could take 2 weeks for the body to get over a illness, or up to 6 weeks for severe or critical cases. Newer data show that recovery varies for different people, depending on age and overall health. Fatigue, headache, and trouble breathing were the symptoms most likely to linger [34,35].

Recovery time After Severe Illness of SARS-CoV- 2(COVID-19) infections, small percentage of people who have the new coronavirus need to stay in the hospital to get help in breathing. It may depend age and your overall health. This might last 2 weeks or more. Some people who have severe COVID-19 get a complication called Acute respiratory distress syndrome (ARDS), which can damage the lungs and make it very hard to breathe.severely ill need treatment in an intensive care unit (ICU). Many patients who spend time in the ICU lose weight and strength [36,19,37].

Recovery Outlook, Scientists are still looking at how a person's immune system responds to SARS-CoV-2(COVID-19) infection and whether they can infect the virus again after you recover. One early study on monkeys found that they didn't get infected a second time. Other research says that some people might lose their antibodies over a couple of months. But the patent may have the virus in your body for weeks, it's a good idea to keep following official

advice on washing your hands, keeping surfaces clean and staying home when possible [38,39].

There's no treatment for SARS-CoV-2 (COVID-19), expect the prevention through the vaccine, although if people have to stay in the hospital, some medicines may shorten their recovery. Eat healthy foods, Drinks lots of fluids and Lower your fever. Take acetaminophen or ibuprofen if you have a temperature or body aches. Be careful not to take more than a total of 3,000 milligrams every 24 hours. That includes acetaminophen alone as well as in medications like cold and flu pills and syrups [40,41].

There are many factors that affect a vaccine's coverage rate of SARS-CoV-2(COVID-19). Some African country are facing availability difficulty ranging from production out-put to distribution, all because of the outburst in population density, SARS-CoV-2(COVID-19) crisis, production capacity is being tested on an unprecedented scale, with vaccine makers turning to dozens of third-party manufacturers in order to fulfill orders for hundreds of millions of doses [15,11].

Another vaccine coverage challenges is distribution, African like Nigeria finds it difficult to distribute the vaccine from one state to another. It costs a country like Nigeria a whopping sum of 10(ten) billion naira to distributes the vaccine in its 36 states from rural or remote areas, several other SARS-CoV- 2(COVID-19) vaccines pose broader cold-chain challenges: the Pfizer-BioNTech vaccine must be stored at -70°C (-94°F), meaning warehouses, trucks and planes, and points of care all require ultra-cold freezers. Oxford/ AstraZeneca COVID-19 vaccine (AZD 1222). can be stored and transported at normal refrigerated temps of 2 degrees to 8 degrees Celsius (36 degrees to 46 degrees Fahrenheit) for at least six months and can be administered in "existing healthcare settings," giving the shot a major logistics leg up over a leading mRNA-based competitor that requires ultra-cold storage [12].

Furthermore, Because of the education level of African and Nigerian populace, our healthcare giver should give public awareness and gain the public understanding on vaccine's coverage rate of SARS-CoV-2(COVID-19) Health officials can build trust in their communities through clear and transparent communication about vaccines. This includes information about their effectiveness, any expected side effects, and when to return for booster shots [42].

Health officials must be aware of the Complexity of the vaccine series, to achieve total eradication of SARS-CoV-2(COVID-19). -Sometimes two or more doses are recommended for maximum protection, with weeks or even months between doses. The Moderna and Pfizer-BioNTech vaccines both require two doses, and individuals will have to receive the same vaccine for both doses.

5. CONCLUSION

In conclusion, SARS-CoV-2(COVID-19) is one of the greatest challenges of the twenty first century. It was AIDS-HIV, that was the limiting factor in the middle of 80s and 90s, but we still find a way to combat AIDS -HIV, SARS-CoV-2(COVID-19) will not be an exception. The world is beaming the scientific search light of the prevention, cure and treatment of SARS-CoV-2(COVID-19) and 99% success has been achieved, this gives rise to the vaccine production and distribution all over the world. The limitation factors that we have in the third world country like Nigeria is acquiring and storage of the vaccine, knowing fully-well that, we may not have the state of the art facility in our research centers and hospitals for the latest developed vaccine, but efforts are ongoing to develop our health care facilities to contain the world latest invention. The mathematical model produced an asymptotically stable population such that the importance of using vaccines to protect and prevent the spread of the SARS-CoV-2(COVID-19) has been shown.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Antia R, Bergstrom CT, Pilyugin SS, Kaech SM, Ahmed R. Models of CD8+ responses: What is the antigen-independent proliferation program. *J Theor Biol.* 2003 ;221(4): 585–98. pmid:12713942

2. CDC; 2012.
Available:<https://www.cdc.gov/vaccines/vpd/vpd-vac-basics.html>
3. CDC; 2018.
Available:www.cdc.gov/vaccines/hcp/conversations/understanding-vacc-work.html
4. History of Vaccines; 2018. Available: www.historyofvaccines.org/content/articles/different-types-vaccines
5. The Immunisation Advisory Centre; 2020. Available:<https://www.immune.org.nz/vaccines/vaccine-development/types-vaccines>
6. Vaccines; 2020.
Available:<https://www.vaccines.gov/basics/types>
7. Uahimd R Din. Study of transmission dynamics of novel COVID-19 by using mathematical model. *Advances in Difference Equations*; 2020.
Available:<https://doi.org/10.1186/s13662-020-02783-x>.
8. Anirudh A. Mathematical modelling and the transmission dynamics in predicting the Covid-19—what next in combating the pandemic. 2020;S2468-0427(20)30018-X. Available:<https://doi.org/10.1016/j.idm.2020.06.002>
9. Kermack WO, Mckendrick AG. A contribution to the mathematical theory of epidemics, *Proceedings of the Royal Society of London*. 1927;115:700 - 721.
10. Hethcote HW. The mathematics of infectious diseases. *SIAM Review*. 2020; 42:599 – 653.
11. Hellewell J, Abbott S, Gimma A, Bosse NI, Jarvis CI, Russell TW, Munday JD, Kucharski AJ, Edmunds WJ, Sun F, et al. Feasibility of controlling COVID-19 outbreaks by isolation of cases and contacts, *The Lancet Global Health*; 2020
Kucharski AJ, Russell TW, Diamond C, Liu Y, Edmunds J, Funk S, Eggo RM, Sun F, Jit M, Munday JD, et al. Early dynamics of transmission and control of COVID-19: A mathematical modelling study. *The Lancet Infectious Diseases*; 2020.
12. Kucharski AJ, Russell TW, Diamond C, Liu Y, Edmunds J, Funk S, Eggo RM, Sun F, Jit M, Munday JD, et al. Early dynamics of transmission and control of COVID-19: A mathematical modelling study. *The Lancet Infectious Diseases*; 2020
13. Nigeria Centre for Disease Control. Coronavirus Covid -19 Microsite; 2020.
14. World Health Organization. Question and answer: Influenza and Covid – 19 – Similarities and Differences; 2020.
15. Iboi E, Ngonghala CN, Gumel AB. Will an imperfect vaccine curtail the covid-19 pandemic in the us? 2020.
16. Diekmann O, Heesterbeek JAP, Metz JAJ. On the definition and computation of basic reproductive ratio RO in models for infectious diseases in heterogeneous populations. *Journal of Math Bio*. 1990;28: 365–382..
17. Hefferman JM, Smith RJ, Wahl LM. Perspectives on the basic reproductive ratio. *J R Soc Interface*. 2005;2:281– 293.
18. World Health Organization. Report of the WHO China Joint Mission on Coronavirus Disease 2019 (COVID-19); 2020.
19. Osuntokun OT, Binuyo AO. Optimising medicinal plants values grown in Nigeria for prevention, controlling and treatment of infectious diseases, determinant factors of infant mortality using mathematical modelling protégé. *Journal of Materials Science Research and Reviews*. 2021;7(1):15-30.
Available: [https:// www.j ournaljmsrr.com/index.php /JMSRR /article /view/30170](https://www.journaljmsrr.com/index.php/JMSRR/article/view/30170)
20. Rao V. Dukkupati. MATLAB An introduction with applications, Published by New Age International (P) Limited Publishers, New Delhi; Applied Numerical Methods with MATLAB for engineers and Scientists; 2010. ISBN:978-81-224-2920- 6 Steve C. Chapra.,
21. Steve C. Chapra. Applied Numerical Methods with MATLAB for engineers and Scientists, third edition, published by McGraw-Hill; 2012. third edition, published by McGraw-Hill; 2012.
22. Daniel Deborah O. Mathematical model for the transmission of COVID-19 with nonlinear forces of infection and the need for prevention measure in Nigeria. *Journal of Infectious Diseases and Epidemiology*. 2020;6(5). ISSN: 2474-3658
23. Oran DP, Topol EJ. Prevalence of asymptomatic SARS-CoV-2 infection: A narrative review. *Ann Intern Med*; 2020. Available:<https://doi.org/10.7326/M20-3012>
24. PiuSamui. A mathematical model for COVID-19 transmission dynamics with a case study of India. *Journal of Chaos, Solitons and Fractals, Nonlinear Science*

- and Nonequilibrium and complex phenol mena; 2020.
Available:<https://doi.org/10.1016/j.chaos.2020.110173>.
25. Cheng HY, Jian SW, Liu DP, Ng TC, Huang WT, Lin HH. Contact tracing assessment of COVID-19 transmission dynamics in taiwan and risk at different exposure periods before and after symptom onset. *JAMA Intern Med*; 2020. Available:<https://doi.org/10.1001/jamainternmed.2020.2020>
 26. Payne DC, Smith-Jeffcoat SE, Nowak G. SARS-CoV-2 Infections and Serologic Responses from a Sample of U.S. Navy Service Members - USS Theodore Roosevelt, April 2020. *MMWR Morb Mortal Wkly Rep*; 2020. Available:<https://dx.doi.org/10.15585/mmwr.mm6923e4>
 27. Morens DM, Fauci AS. Emerging pandemic diseases: How we got to COVID-19. *Cell*. 2020;182:1077–1092 DOI: 10.1016/j.cell.2020.08.021; PMID: 32846157
 28. Zhang Q. Human genetics of life-threatening influenza pneumonitis. *Hum. Genet*. 2020;139:941–948. DOI: 10.1007/s00439-019-02108-3; pmid: 32025908
 29. Pérez de Diego R. Human TRAF3 adaptor molecule deficiency leads to impaired Toll-like receptor 3 response and susceptibility to herpes simplex encephalitis. *Immunity*. 2010;33, 400–411. DOI: 10.1016/j.immuni.2010.08.014; PMID: 20832341
 30. Casanova JL, Su HC. COVID Human genetic effort, a global effort to define the human genetics of protective immunity to SARS-CoV-2 infection. *Cell*. 2020;181:1194–1199. DOI: 10.1016/j.cell.2020.05.016; pmid: 32405102
 31. Zhang SY. TLR3 deficiency in patients with herpes simplex encephalitis. *Science*. 2007;317: 1522–1527. DOI: 10.1126/science.1139522; pmid: 17872438
 32. Thomsen MM. Identification of an IRF3 variant and defective antiviral interferon responses in a patient with severe influenza. *Eur. J. Immunol*. 2019;49:2111–2114 (2019). DOI: 10.1002/eji.201848083; PMID: 31250433
 33. Bastard P. Auto-antibodies against type I IFNs in patients with life-threatening COVID-19. *Science*; 2020. DOI: 10.1126/science.abd4585.
 34. Andreano, Nicastrì E, Paciello I, Pileri P, Manganaro N, Piccini G, Manenti A, Pantano E, Kabanova A, Troisi M. Identification of neutralizing human monoclonal antibodies from Italian COVID-19 convalescent patients; 2020. bioRxiv, 10.1101/2020.05.05.078154
 35. Baum A, Fulton BO, Wloga E, Copin R, Pascal KE, Russo V, Giordano S, Lanza K, Negron N, Ni M. Antibody cocktail to SARS-CoV-2 spike protein prevents rapid mutational escape seen with individual antibodies. *Science*; 2020. DOI: 10.1126/science.abd0831
 36. Braun J, Loyal L, Frensch M, Wendisch D, Georg P, Kurth F, Hippenstiel S, Dingeldey M, Kruse B, Fauchere F. Presence of SARS-CoV-2 reactive T cells in COVID-19 patients and healthy donors *Med Rxiv*; 2020. DOI: 10.1101/2020.04.17.2006440
 37. Olaposi Omotuyi, Oyekanmi Nash, Basiru O. Ajiboye, Victor O. Olumekun, ET AL. *Aframomum melegueta* secondary metabolites exhibit polypharmacology against SARS-CoV-2 drug targets: in vitro validation of furin inhibition, *Phytotherapy Research*. 2020;1–12. Wiley online library.com/journal/ptr, DOI: 10.1002/ptr.6843
 38. Oludare Temitope Osuntokun. Synergistic efficacy of *Aframomum melegueta* [Roscoe] K. Schum and *Spondias mombin* (Linn), A predictive treatment of SARS-CoV-2 (COVID -19) Infection, *Journal of Bioscience & Biomedical Engineering (J B & Bio Engine*. 2020;1(2):1-8. Available :www.uniscience.pub.com DOI: doi.org/10.47485/2693-2504.1013
 39. Brouwer PJM, Caniels TG, vander Straten K, Snitselaar JL, Aldon Y, Bangaru S, et al. Potent neutralizing antibodies from COVID-19 patients define multiple targets of vulnerability. *Science*; 2020. DOI: 10.1126/science.abc5902
 40. Hoffmann M, Kleine Weber H, Schroeder S, Krüger N, Herrler T, Erichsen S, et al. SARS-CoV-2 Cell entry depends on ACE2 and TMPRSS2 and is blocked by a clinically proven protease inhibitor. *Cell*. 2020;181:271–280.e8
 41. Siddiqi HK, Mehra MR. COVID-19 Illness in native and immunosuppressed states: a clinical-therapeutic staging proposal. *J Heart Lung Transplant*; 2020. DOI: 10.1016/j.healun.

42. Qian L, Biao T, Nicola LB, Yanni X, Wu J. Modeling the impact of mass influenza vaccination and public health interventions on covid-19 epidemics with limited detection capability. *Mathematical Bio sciences*; 2020. Available:<https://doi.org/10.1016/j.mbs.2020.108378>

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