

Analysis of Alpha and Gamma Variants of Corona Virus through WX1X2YW Model

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ABSTRACT

A WX1X2YW (Susceptible, Alpha Infectious, Gamma Infectious and Recovered) model has been developed to mimic coronavirus transmission in human populations in order to investigate the impact of two variations, such as Alpha and Gamma versions of the corona virus and their recovery during the COVID-19 pandemic. The Alpha and Gamma corona virus types are included in this model in accordance with the way the virus spreads. We determined R0, the threshold value. The Routh-Hurwitz Criterion has demonstrated that if the threshold number is less than one, the coronavirus has local steadiness at the infection free equilibrium point, and if the basic reproduction number is more than one, the endemic equilibrium state is locally stable. The Lyapunov Krasovshii approach produces global stability. Ranga-Kutta 4-5th numerical techniques are used to solve and simulate the system of differential equations, which sheds light on how the two coronavirus genotypes assault human society. This strategy may help direct the creation of defenses against the spread of the many coronavirus strains in human populations.

Keywords: Alpha and Gamma variants of Corona Virus; Routh-Hurwitz Criterion; Local and Global Stability; Threshold number. Lyapunov Krasovshii's method.

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1. INTRODUCTION

The SARS-CoV-2 virus, a novel coronavirus initially discovered in Wuhan, China, in December 2019, is the cause of COVID-19 (Coronavirus Disease 2019) [1]. Along with SARS-CoV (2002 outbreak) and MERS-CoV (2012 outbreak), it is a member of the Corona viridae family [2]. Aerosols, surface contact, and respiratory droplets are the main ways that SARS-CoV-2 spreads. Fever, coughing, exhaustion, and loss of taste or smell are common symptoms of COVID-19, while shortness of breath, pneumonia, and multi-organ failure are severe symptoms [3, 4].

Millions of deaths, lockdowns, economic crises, and unprecedented worldwide vaccination campaigns resulted from the pandemic that the World Health Organization labelled a Public Health Emergency of International Concern (PHEIC) in January 2020 and a pandemic in March 2020. The WHO has designated the several corona virus variations as either Variants of Interest (VOI) or Variants of Concern (VOC). The following are the several corona virus variations [5, 6].

Alpha Variant (B.1.1.7): In September 2020, it was first created in the United Kingdom [7]. This variant's main characteristics include being highly transmissible (40–70% faster spread) and having the highest infection rates in the USA and Europe in early 2021 [8, 9].

Beta Variant (B.1.351): In October 2020, the beta variant originated in South Africa [10]. Strong immune escape caused by the E484K mutation is a key characteristic of this variation [11]. This viral strain infects both European and African nations.

In November 2020, the Gamma Variant (P.1) was created in Manaus, Brazil, and its main characteristics include high transmissibility, immunological escape, and reinfection risk [12].

The second deadly wave occurred in Brazil and its mutations are N501Y, E484K, K417T [13].

Delta Variant (B.1.617.2) originated in India in late 2020 and its main characteristics are extremely transmissible, severe disease, high hospitalization [14]. It had global dominance mainly in India, USA, and UK and its mutations are L452R, P681R, and D614G.

Late in 2021, Omicron Variant (B.1.1.529 and sublineages: BA.1, BA.2, XBB, etc.) was observed in Botswana and South Africa. Although this variety had a lot of mutations and a significant immune escape, the sickness was not as severe as Delta's. Due to recurrent waves with subvariants, the variant dominated the world market starting in late 2021 [15, 16, 17].

Additional Variants of Interest include Mu (B. 1. 621) initially observed in Colambia and Lambda, which was observed in Peru with a substantial immune escape.

We have taken into account the Alpha and Gamma Variants in our WX1X2YW model.

Variant Alpha (B.1.1.7)

The WHO designated it a Variant of Concern (VOC) in December 2020 after it was initially discovered in Kent, UK, in September 2020. It has 17 mutations, some of which are prominent in the spike (S) protein:

- o N501Y: Boosts transmissibility by increasing binding affinity to the ACE2 receptor.
- o P681H: Found next to the fur in the cleavage location, which may facilitate viral entrance.
- o 69–70 deletion: Linked to difficulties in diagnosis and immune evasion.

Because the alpha variation has a greater viral load in infected persons and is thought to be 40-70% more transmissible than the original Wuhan strain, it spreads quickly.

In contrast to previous versions, it caused significant waves in the UK, Europe, and North America in late 2020–2021, which were linked to an increased risk of hospitalization and fatality. Compared to the first wave, younger age groups were said to be more impacted. Some vaccinated and previously infected people had decreased antibody neutralization. Nonetheless, vaccines (such as those made by Pfizer, AstraZeneca, Moderna, and others) continued to be generally effective, particularly in preventing serious illness and death.

Gamma variant was first identified in November 2020 in Manaus, Brazil, Gamma Variant (P.1) spread quickly by early 2021. In January 2021, it was designated as a Variant of Concern (VOC).

It has 17 different amino acids, especially in the spike protein:

N501Y: Boosts binding to ACE2, which is shared by Alpha and Beta.

- o E484K: Reduces antibody neutralization and is strongly associated with immunological escape.
- o K417T: Could aid in avoiding immunological detection

Compared to previous strains, the gamma variation is 1.7–2.4 times more contagious.

Because it may partially circumvent protection from a previous infection with the original strain, it is linked to reinfections. Despite having a high past infection rate, this variant virus was the cause of the second lethal wave in Brazil, overwhelming hospitals in Manaus (indicating the possibility of reinfection). Although it never achieved the same level of worldwide dominance as Alpha or Delta, it did expand to Japan, the United States, and some European nations.

Effectiveness of Vaccines and Immune Escape

- Displayed a notable decrease in neutralizing antibodies from vaccination and spontaneous infection.
- The E484K mutation hindered the effectiveness of certain monoclonal antibody treatments.
- Although breakout infections were more often than Alpha, vaccines continued to offer protection against serious illness.

Methods of Treatment (Similar to Both Variants)

- Supportive care includes anticoagulants, corticosteroids (like dexamethasone), and oxygen therapy.
- Remdesivir, Molnupiravir, and Paxlovid (later developed) are antiviral medications.
- The E484K mutation caused several monoclonal antibodies to lose their efficacy, particularly against Gamma.
- Vaccination: Continued to be the best defense, despite Gamma decreasing the protection it provided.

For the recovery of the patient and analysis of the impact of virus and their variants, we developed new mathematical model WX1X2YW and started following many literature surveys.

Andrew et al [18] first discussed and confirmed the existence of coronavirus 19 in India in their journal. Narasimhan [19] also agreed that the Kerala student was the first to be detected coronavirus positive during their test. In an effort to stop the spread and recovery of people from the coronavirus, the WX1X2YW model is constructed and the model incorporates two

variants of corona viruses such as Alpha Variant and Gamma Variant. The model helps us understand how different public policies affect the total number of instances of COVID-19. Unfortunately, it predicts that with the current measures (which aren't very effective), and without good drug treatments or vaccines available to everyone, SARS-CoV-2 infections might continue for a long time and even become seasonal.

Governments must reopen nations while weighing the virus's effects on the economy, social cohesion, and mental health of its citizens. Some suggested strategies include gradually reopening and keeping a close watch on new cases. The model shows that this approach can be successful if there is good social distancing, partial lockdowns, widespread mask-wearing, and careful monitoring to prevent having to lock down again.

Quarantine means keeping people away from others to prevent the spread of diseases. In recent decades, quarantine has become very effective in stopping the spread of diseases like the Coronavirus, Leprosy, Plague, Smallpox, and Coronavirus etc. [20, 21, 22]. Goh et al. and Kass et al. also studied the behavior of coronavirus by using mathematical models [23, 24]. Kermack and McKendrick [25, 26, 27] first formulated a SIR (Susceptible, Infectious, Recovered) mathematical dynamical model for the transmition of malicious object for the epidemiology during early age of nineties. A new compartment named Exposed instead of Recovered compartment was added by Rechard and Mark in their epidemiological model SEI for the transition of viruses [28]. The SEIR model suggested by Ping Yan [29] assumes that once people recover from the disease, they are permanently immune with a certain probability. However, this assumption doesn't always match real-life situations. To address this restriction, Mishra and Saini [30-31] introduced a SEIRS model that includes immunological phases that are latent and transient. The spread of prevalent diseases can be better explained by this paradigm. Recently, researchers have focused more on combining virus spread models with antivirus measures to study how viruses spread which includes looking at methods like vaccination [32-35] and quarantine [36].

Our goal was to create a WX1X2YW model that included two distinct coronavirus variants without confirming permanent immunity. The infectious class has been split into two sub-compartments for the two coronavirus variants, such as the Alpha Variant for the first compartment and the Gamma Variant for the second. After receiving the appropriate care and immunizations, the vulnerable individuals migrate from the infectious class to the recovered class based on their traits and spreading behaviours. Since there is no lasting immunity, we hypothesized that those who had recovered will become vulnerable and join the susceptible class. The total population size in this model is variable because some babies will be born during this time, some may travel from other countries, and some people may pass away naturally—that is, not as a result of the corona virus attack—and some may die as a result of the corona virus attack.

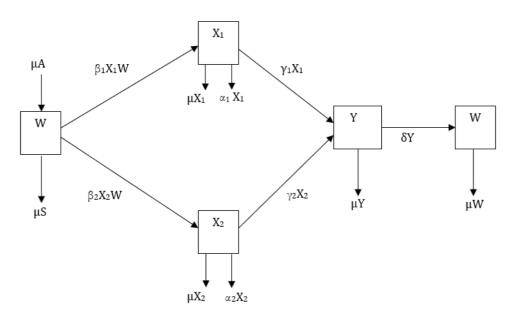


Figure 1: A schematic depiction of the coronavirus's spread throughout human civilization

Consider the system of differential equation for the schematic diagram 1.

$$\frac{dW}{dt} = \mu A - \beta_1 X_1 W - \beta_2 X_2 W - \mu W + \delta Y
\frac{dX_1}{dt} = \beta_1 X_1 W - \mu X_1 - \alpha_1 X_1 - \gamma_1 X_1
\frac{dX_2}{dt} = \beta_2 X_2 W - \mu X_2 - \alpha_2 X_2 - \gamma_2 X_2
\frac{dY}{dt} = \gamma_1 X_1 + \gamma_2 X_2 - \mu Y - \delta Y$$
(1)

Since each person is presumed to be susceptible, let N be the entire population and μ be the inflow rate, or the integration of new individuals due to the entrance of people from other places or the birth of a new infant in the community. Again, μ indicates the death rate which will take place in each compartment that is not caused by a coronavirus infection, such as natural death or death from another cause. β_1 be the infectivity contact rate due to attack of Alpha variant of corona virus and β_2 be again an infectivity contact rate of Gamma variant of corona virus. γ_1 is the rate of transmission of people from Alpha infectious class X_1 to recovered compartment Y, and γ_2 be the rate of transmission of people from Gamma infectious class X_2 to recovered compartment Y. We have seen that there is no permanent immunity again, recovered people will become susceptible by the rate of δ .

Let N = W + X₁ + X₂ + Y
$$\frac{dN}{dt} = \frac{dW}{dt} + \frac{dX_1}{dt} + \frac{dX_2}{dt} + \frac{dY}{dt}$$

$$= \mu A - \beta_1 X_1 W - \beta_2 X_2 W - \mu W + \delta Y + \beta_1 X_1 W - \mu X_1 - \alpha_1 X_1 - \gamma_1 X_1$$

$$+ \beta_2 X_2 W - \mu X_2 - \alpha_2 X_2 - \gamma_2 X_2$$

$$+ \gamma_1 X_1 + \gamma_2 X_2 - \mu Y - \delta Y$$

$$\frac{dN}{dt} = \mu A - \mu N - (\alpha_1 X_1 + \alpha_2 X_2)$$
(2)

Under the equilibrium state, in the absence of different variant of corona viruses, $X_1 = 0$; $X_2 = 0$ then $\frac{dN}{dt} = 0$ from (2) we get $0 = \mu A - \mu N \Rightarrow N = A$

Let us define the solution region D = $\{(W, X_1, X_2, Y) / W \ge 0; X_1 \ge 0; X_2 \ge 0; Y \ge 0; W, X_1, X_2, Y \le A\}$

Solution of the initial value problem starting in D and define by (1) exists and unique on a maximal interval. Since remains bounded in the positively invariant region D, the maximal interval is $[0,\infty)$..

Basic Reproduction Number: - We find the basic reproduction number with the help of next generation method. This number can be explained as the average number of secondary infections enter into the totally susceptible population. Basic reproduction number can be obtained by the linearisation of the system of equations (1).

Here
$$f = \begin{pmatrix} \beta_1 WX \\ \beta_1 WX \end{pmatrix} \text{ and } v = \begin{pmatrix} (\mu + \alpha_1 + \gamma_1)X_1 \\ (\mu + \alpha_2 + \gamma_2)X_2 \end{pmatrix}$$

$$F = \begin{pmatrix} \frac{\partial f}{\partial X_1} & \frac{\partial f}{\partial X_2} \\ \frac{\partial g}{\partial X_1} & \frac{\partial g}{\partial X_2} \end{pmatrix} = \begin{pmatrix} \beta_1 & 0 \\ 0 & \beta_2 \end{pmatrix} As W = 1$$
Since

$$V = \begin{pmatrix} \frac{\partial v_1}{\partial X_1} & \frac{\partial v_1}{\partial X_2} \\ \frac{\partial v_2}{\partial X_1} & \frac{\partial v_2}{\partial X_2} \end{pmatrix} = \begin{pmatrix} (\mu + \alpha_1 + \gamma_1) & 0 \\ 0 & (\mu + \alpha_2 + \gamma_2) \end{pmatrix}$$

$$R_{0} = FV^{-1} = \begin{pmatrix} \frac{\beta_{1}}{(\mu + \alpha_{1} + \gamma_{1})} & 0\\ 0 & \frac{\beta_{2}}{(\mu + \alpha_{2} + \gamma_{2})} \end{pmatrix}$$

Now the Basic Reproduction Number

Since the maximum of the Eigen values from the Eigen values $\frac{\beta_1}{(\mu + \alpha_1 + \gamma_{\partial 1})} \frac{\beta_2}{\text{and}} \frac{\beta_2}{(\mu + \alpha_2 + \gamma_2)}$

So, we take $\frac{\beta_1}{(\mu + \alpha_1 + \gamma_{\partial 1})}$ as the basic reproduction number R_0 since X_1 variant is stronger than X_2 i.e. $\beta_1 > \beta_2$.

Local Stability Analysis for the Corona Virus Free Equilibrium State: - Consider the system of differential equation (1), if R₀ < 1then the corona virus disease free equilibrium state is locally asymptotically stable in the region D, otherwise it is unstable.

Proof:- To show the local stability for the disease-free equilibrium state, we consider the Jacobian matrix

$$J = \begin{bmatrix} -\mu_1 & -\beta_1 A & -\beta_2 A & \delta \\ 0 & \beta_1 A - \mu - \gamma_1 - \alpha_1 & 0 & 0 \\ 0 & 0 & \beta_2 A - \mu - \alpha_2 - \gamma_2 & 0 \\ 0 & \gamma_1 & \gamma_2 & -(\mu + \delta) \end{bmatrix}$$

As per the basic reproduction numbers $\beta_1 A < (\mu + \alpha_1 + \gamma_1)$; $\beta_2 A < (\mu + \alpha_2 + \gamma_2)$ then all the diagonal elements are negatives this implies that the system is locally asymptotically stable.

For the Endemic Equilibrium State D* (W*, X₁*, X₂*, Y*)

We have
$$N = W^* + X_1^* + X_2^* + Y^*$$

$$\frac{dW^*}{dt} = 0 = \frac{dX_1^*}{dt} = \frac{dX_2^*}{dt} = \frac{dY^*}{dt}$$

$$W^* = \frac{(\mu + \alpha_1 + \gamma_1)}{\beta_1} = \frac{(\mu + \alpha_2 + \gamma_2)}{\beta_2}$$

$$X_{1}^{*} = \{(\mu + \delta)N\beta_{1} - (\mu + \alpha_{1} + \gamma_{1})(\mu + \delta) - (\beta_{1}(\mu + \delta) + \beta_{1}\gamma_{2})X_{2}\}\frac{1}{\{\beta_{1}(\mu + \delta) + \beta_{1}\gamma_{21}\}},$$

$$Y^* = \frac{1}{(\mu + \delta)} \{ \gamma_1(\mu + \delta) N \beta_1 - (\mu + \alpha_1 + \gamma_1)(\mu + \delta) - \frac{1}{(\mu + \delta)^2} \}$$

$$(\beta_1(\mu+\delta)+\beta_1\gamma_2)X_2^*)\frac{1}{(\beta_1(\mu+\delta)+\beta_1\gamma_1}+\gamma_2X_2$$

Global Stability of the system of equation (1)

As per Krosovshii's Method which suggest a simple form of Lyapunov function:-

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

$$f(x) = \begin{pmatrix} \mu A - \beta_1 X_1 W - \beta_2 X_2 W - \mu W + \delta Y \\ \beta_1 X_1 W - \mu X_1 - \alpha_1 X_1 - \gamma_1 X_1 \\ \beta_2 X_2 W - \mu X_2 - \alpha_2 X_2 - \gamma_2 X_2 \\ \gamma_1 X_1 + \gamma_2 X_2 - \mu Y - \delta Y \end{pmatrix}$$

Where

If the matrix $F = A + A^T$ is a negative definite in a neighbourhood of D, then the equilibrium pt at the origin is asymptotically stable.

Here

$$A = \begin{bmatrix} \frac{\partial f_1}{\partial W} & \frac{\partial f_1}{\partial X_1} & \frac{\partial f_1}{\partial X_2} & \frac{\partial f_1}{\partial Y} \\ \frac{\partial f_2}{\partial W} & \frac{\partial f_2}{\partial X_1} & \frac{\partial f_2}{\partial X_2} & \frac{\partial f_2}{\partial Y} \\ \frac{\partial f_3}{\partial W} & \frac{\partial f_3}{\partial X_1} & \frac{\partial f_3}{\partial X_2} & \frac{\partial f_3}{\partial Y} \\ \frac{\partial f_4}{\partial W} & \frac{\partial f_4}{\partial X_1} & \frac{\partial f_4}{\partial X_2} & \frac{\partial f_4}{\partial Y} \end{bmatrix}$$

$$= \begin{bmatrix} -\beta(X_1 + X_2) - \mu & -\beta_1 W & -\beta_2 W & \delta \\ \beta_1 X_1 & \beta_1 W - \mu - \alpha_1 - \gamma_1 & 0 & 0 \\ \beta_2 X_2 & 0 & \beta_2 W - \mu - \alpha_2 - \gamma_2 & 0 \\ \delta & 0 & 0 & -(\mu + \delta) \end{bmatrix}$$

$$A^{T} = \begin{bmatrix} -\beta(X_{1} + X_{2}) - \mu & \beta_{1}W & \beta_{2}W & 0 \\ -\beta_{1}X_{1} & \beta_{1}W - \mu - \alpha_{1} - \gamma_{1} & 0 & 0 \\ -\beta_{2}X_{2} & 0 & \beta_{2}W - \mu - \alpha_{2} - \gamma_{2} & 0 \\ \delta & 0 & 0 & -(\mu + \delta) \end{bmatrix}$$

According to the Krasovsshii's method, we have to show that $F = A + A^T$ is negative definite in a neighbourhood of D, then the equilibrium state is asymptotically stable.

Therefore $F = A + A^T$

$$= \begin{bmatrix} -2\beta_{1}(X_{1} + X_{2}) - 2\mu & \beta_{1}X_{1} & \beta_{2}X_{2} & \delta \\ 0 & 2(\beta_{1}W - \mu - \alpha_{1} - \gamma_{1}) & 0 & \gamma_{1} \\ \beta_{2}X_{2} - \beta_{1}W & 0 & 2(\beta_{2}W - \mu - \alpha_{2} - \gamma_{2}) & \gamma_{2} \\ \delta & \gamma_{1} & -\gamma_{2} & -2(\mu + \delta) \end{bmatrix}$$

As the diagonal elements are negative for the basic reproduction number of $F = A + A^{T}$ then the matrix F is negative definite in the neighbourhood of D. Therefore, the system of equation is asymptotically stable at the equilibrium point by Krosovshii's method.

$$V(x) = f^*(x)f(x) \tag{4}$$

$$f(x) = \begin{bmatrix} \mu A - \beta_1 X_1 W - \beta_2 X_2 W - \mu W + \delta Y \\ \beta_1 X_1 W - (\mu + \alpha_1 + \gamma_1) X_1 \\ \beta_2 W X_2 - (\mu + \alpha_2 + \gamma_2) \\ \gamma_1 X_1 + \gamma_2 X_2 - (\mu + \delta) Y \end{bmatrix}$$

Since

And

$$f^*(x) = \begin{bmatrix} \mu A - \beta_1 X_1 W - \beta_2 X_2 W - \mu W + \delta Y & \beta_1 X_1 W - (\mu + \alpha_1 + \gamma_1) X_1 & \beta_2 X_2 W - (\mu + \alpha_2 + \gamma_2) X_2 & \gamma_1 X_1 + \gamma_2 X_2 - (\mu + \delta) Y \end{bmatrix}$$

$$V(x) = \frac{\{\mu A - \beta_1 X_1 W - \beta_2 X_2 W - \mu W + \delta Y\}^2 + \{\beta_1 X_1 W - (\mu + \alpha_1 + \gamma_1) X_1\}^2 + \{\beta_2 W X_2 - (\mu + \alpha_2 + \gamma_2)\}^2 + \{\gamma_1 X_1 + \gamma_2 X_2 - (\mu + \delta) Y\}^2}$$

Therefore

Since V(x) is positive definite for all values of W, X_1, X_2 , and Y. $V(x) \to \infty$ as $||x|| \to \infty$ then the system of differential equation is globally asymptotically stable if we consider one equilibrium point and if we consider both the equilibrium point then by Lyapunov theorem, the system of equation (1) is globally asymptotically unstable.

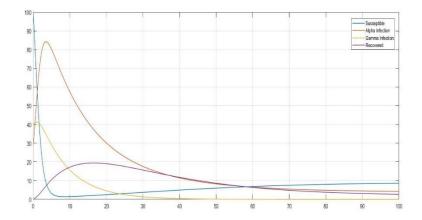


Figure 2: Time-dependent dynamic behavior of susceptible, infection through alpha variant, gamma variant, and recovered in lacs.

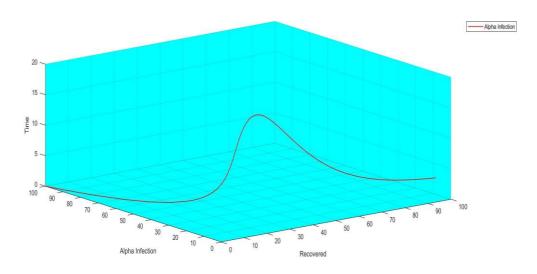


Figure 3: Time-dependent dynamic behaviour of the coronavirus of alpha variant and recovered individuals.

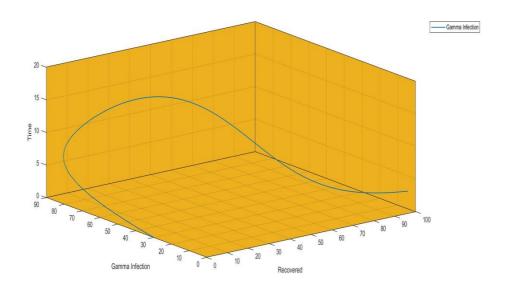


Figure 4: Time-dependent dynamic behaviour of the coronavirus of gamma variant and recovered individuals.

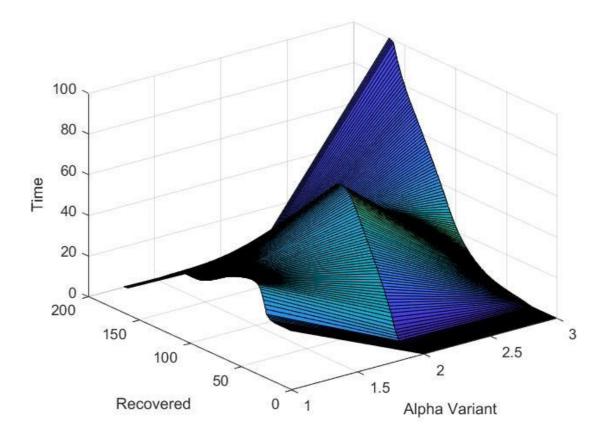


Figure 5: The alpha variant coronavirus's time-dependent dynamic behavior in recovered patients with the help of surface graph.

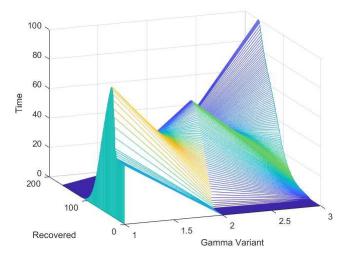


Figure 6: Time-dependent dynamic behaviour of the coronavirus of gamma variant and recovered individuals.

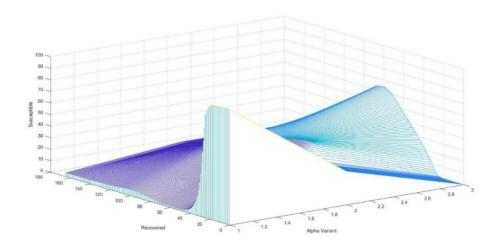


Figure 4: Time-dependent dynamic behaviour of the coronavirus of alpha variant and recovered individuals.

Susceptible	Alpha Infection	Gamma Infection	Recovered	Time
100	29	40	0	0
99.998325	29.0010363	40.00012756	5.02E-05	4.37E-05
99.99664999	29.00207263	40.00025511	0.000100478	8.74E-05
99.99497497	29.00310897	40.00038266	0.000150719	0.000131055
99.99329993	29.00414532	40.0005102	0.000200961	0.00017474
99.98492454	29.00932738	40.00114782	0.000452191	0.000393165
99.97654882	29.01450989	40.00178531	0.000703452	0.00061159
99.96817278	29.01969284	40.00242266	0.000954745	0.000830015
99.9597964	29.02487625	40.00305987	0.00120607	0.00104844

Table 1: Time series values for the above graphs

99.91790956	29.05079997	40.00624391	0.002463168	0.002140564
99.87601449	29.07673488	40.00942453	0.003721058	0.003232689
99.8341112	29.10268096	40.01260175	0.004979739	0.004324813
99.7921997	29.12863819	40.01577555	0.006239213	0.005416938
99.58251965	29.25859117	40.03159336	0.012548471	0.01087756
99.37263675	29.38882092	40.04732567	0.018877574	0.016338183
99.16255296	29.51932569	40.06297229	0.025226555	0.021798806
98.95227022	29.6501037	40.07853302	0.031595452	0.027259428
97.89794186	30.30802938	40.15504173	0.063739858	0.054562542
96.83894246	30.97251088	40.22937513	0.096387215	0.081865655
95.7755345	31.64330785	40.3015106	0.129541546	0.109168769
94.70799017	32.32016957	40.37142644	0.163206666	0.136471882
89.3182426	35.78613318	40.68701941	0.339311286	0.272987449
83.87120917	39.36042525	40.94441497	0.528625048	0.409503016
78.40942298	43.00234471	41.1420824	0.731342253	0.546018583
72.97809619	46.66796078	41.27923697	0.947481787	0.68253415

Table2: Parametric values for the above graphs.

Parameters	Values
A	15.15
μ	0.05
β_1	0.009
β_1	0.002
γι	0.03
γ ₂	0.007
α_1	0.02
α_2	0.07
δ	0.01
W(t=0)	100
$X_1(t=0)$	29
$X_2(t=0)$	40
Y(t=0)	0

2. DISCUSSION

For figure 2, susceptible starts very high (~100) and declines rapidly in the beginning (as people get infected). It stabilizes at a low value and then slowly increases again after ~40 time units. This rebound suggests secondary susceptibility or model assumptions (reinfection/waning immunity). Alpha Infection (Orange) rises sharply early on, and peaking around time ≈ 5 at ~85 cases. After the peak, it steadily declines, reaches near zero by time ≈ 60 . Gamma appears shortly after Alpha, peaking lower (~40) around time $\approx 2-3$ which declines faster than Alpha, nearly gone by time ≈ 25 . This suggests Gamma was a weaker but earlier co-circulating variant. Recovered starts at zero and rises gradually which peaked around time 20. After that, it slowly declines, suggesting recovery is not permanent (possible reinfection or loss of immunity built into the model).

In figure 3, the graph represents the epidemic trajectory of Alpha variant infections over time. Infections rise sharply, peak, and then decline as more individuals recover, leaving the population dominated by recovered individuals in the long run.

For figure 4, During the initial time period between 0 to 3 Gamma infections increase rapidly and recovered individuals are very few, which indicates the outbreak is in its growth phase. But during the time period 5 to 10 Gamma infections reach their highest levels (≈ 80) and recoveries begin to rise, showing that many infected individuals start transitioning to the recovered state. Declining Phase starts in between 10-20 (Time ≈ 10 -20), Gamma infections steadily decrease and recovered individuals increase continuously and dominate by the end of the timeline (≈ 90 + recovered).

In figure 5, the surface shows how recovery numbers change over time for different Alpha Variant intensities. At lower Alpha values ($\approx 1-1.5$), the epidemic spread is weaker this implies recoveries remain moderate and stable. At higher Alpha values ($\approx 2-3$), the epidemic intensifies sharply: this leads to a steep rise in recovery happens, which indicates many individuals were infected and then recovered quickly. This corresponds to a strong outbreak with a large infected population that eventually transitions to recover. The sharp peak around Alpha $\approx 2-2.5$ shows the critical point of maximum outbreak impact. After the peak, recoveries stabilize as time progresses (epidemic burnout).

In figure 6, and 7 the surface remains flat for the alpha and gamma intensity in between 1 and 1.5, this means outbreaks are mild and spread slowly which leads to lower recovery. But at time 2 sharp peak recoveries appear. The surface shows very steep peak for the alpha and gamma intensity in between time 2.5 to 3. This represents explosive outbreaks that infect a large portion of the population quickly.

3. CONCLUSION

Following the coronavirus pandemic's second wave augmentation, the globe began to face significant difficulties that are altering the way civilizations function. We have developed a mathematical model, WX1X2YW, that incorporates two corona virus variations, such as Alpha and Gamma variations, in order to better comprehend the virus's impacts. This model demonstrates that the virus-free state stabilizes both locally and globally when the fundamental reproduction number, R0, falls below 1 and all model parameters are real and negative. Our belief in recuperation techniques like immunization and therapy is increased by this consistency.

However, the stability of this virus-free condition becomes unstable if certain of the model parameters are not negative. We solve and simulate the system of differential equations using sophisticated numerical techniques known as Runga-Kutta fourth and fifth-order methods in order to tackle this challenging problem. The data from tables I and II was used to construct graphs 2 through 7, which illustrate the changes over time in the susceptible, alpha infectious, gamma infectious, and recovered populations. By analyzing the aforementioned graphs, we may better understand the behavior of the virus and use them as a guide to create vaccinations that work.

Alpha (orange) became the major driver of the epidemic, infecting more individuals than Gamma. Gamma (yellow) peaked quickly but was less impactful and faded sooner. The unusual behaviour of Susceptible and Recovered curves (increasing again after decline) suggests waning immunity or reinfection is allowed in the model as per the figure 2. And long-term equilibrium shows a small balance between Susceptible and Recovered, with very few active infections. The graph shows co-circulation of Alpha and Gamma variants, with Alpha driving the larger outbreak. Gamma peaked earlier but had a weaker impact. Recovery is temporary in this system, leading to fluctuations in susceptibility over time. Ultimately, infections decline to near zero, leaving a mix of recovered and susceptible individuals.

The surface plot of figure 5 illustrates the relationship between Alpha variant infectiousness, recoveries, and time. A threshold around Alpha ≈ 2 marks the transition from mild outbreaks to severe epidemics with rapid, large-scale recoveries. Essentially, it visualizes how increasing transmissibility drives more intense epidemic waves.

The Gamma variant graph 6 shows that increasing transmissibility intensifies outbreaks, with a critical threshold near Gamma ≈ 2 , beyond which infections spread explosively, causing rapid and large-scale recoveries before stabilizing.

The Alpha variant graph 7 reveals a slower and less severe epidemic dynamic compared to Gamma, with outbreaks intensifying at transmissibility ~2 but stabilizing earlier and with fewer overall recoveries.

The stability of the virus-free state, which depends on R_0 and the parameters, gives us hope for overcoming the pandemic. The numerical methods used highlight the importance of precise predictions and management. The graphs provide a detailed view of the pandemic's progression and are crucial for developing strong defenses and effective vaccines for the future.

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