A GLOBAL ASYMPTOMATIC STABILITY OF COVID-19 DIABETES COMPLICATION FREE EQUILIBRIUM

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Abstract:
In this paper, a Mathematical modelling of COVID-19 incorporating the comorbidity of Diabetes was established base on the accompanying assumptions, a global asymptotic stability of the same model was developed by applying the theorem of Castillo-Chavez by fixing a point $E^0 = (X^*, 0)$ *to be globally asymptotically stable equilibrium of the system, provided* $R_0 < 1$ *that and the two set conditions are satisfied. It is very clear that* $\hat{G}(X,Z) < 0$ *so the conditions are not met. Hence,* E^0 *may not be globally asymptotically stable when* $R_0 < 1$.

Keywords: *Asymptotic, COVID-19, Diabetes Complication, Equilibrium and Stability*

Introduction

COVID-19 is transmitted from human-to-human through direct contact with contaminated objects or surfaces and through inhalation of respiratory droplets from both symptomatic and infectious humans. There is also limited data that the virus can be exhaled through normal breathing. The incubation period of COVID-19 ranges from 2 to 14 days, and most infections (over 80%) show mild or no clinical symptoms of the disease (Ngonghala*et al.,* 2020b; Gumel, 2020a).

COVID-19 is transmitted from human-to-human via direct contact with contaminated surfaces and through the inhalation of respiratory droplets from infected individuals (Bai *et al.,* 2020). virus that has caused COVID-19 to be a lethal disease, a disease which targets the human respiratory system, started as an outbreak of pneumonia of unidentified cause? It swiftly became an overwhelming pandemic, thinning out to every country on earth, and wreaking a brutal public health and socio-economic burden globally and the patients were linked to a seafood and wet animal market in Wuhan, Hubei Province, China, an ongoing corona virus disease 2019 (COVID-19) pandemic has decimated the world as we know it (WHO, 2021). SARS-COV2, a highly virulent virus that has caused COVID-19 to be a lethal disease, began as an outbreak of pneumonia of unknown origin in the Chinese city of Wuhan in December of 2019, caused by the novel corona virus, SARS-COV2, a highly virulent virus that has caused COVID-19 to be a fatal disease, a disease that attacks the human respiratory system (Rothana and Byrareddy, 2020; Gumel *et al.,* 2020; Branswell, 2020; Dong *et al.,* 2020; Roda *et al.,* 2020; Sun and Wang, 2020; Contreras *et al.,* 2020; Li *et al.,* 2020; Zhu *et al.,*2020). It is reported that the virus might be bat origin (Zhou *et al.,* 2020). And the virus's spread could be linked to a Hunan Seafood Wholesale Market (Huang *et al.,* 2020; Zhu *et al.,* 2020b). By January 22, 2020, a total of 571 cases of COVID-19 were reported in 25 provinces in China (Rothana and Byrareddy, 2020; Lu, 2020). On January 30, 2020, about 7734 cases have been confirmed in China, with 90 cases reported in about 13 countries (Rothana and Byrareddy, 2020) Including the United States, India, Canada, France, Germany and the United Arab Emirates.

People with certain underlying medical conditions may have a higher risk of severe illness from COVID-19. These conditions include diabetes, heart problems, obesity, and chronic kidney disease. Specifically, the available evidence suggests that people with type 2 diabetes have a higher risk of severe illness from COVID-19, according to the Centers for Disease Control and Prevention (CDC). People with type 1 or gestational diabetes may also have an increased risk, but the data is less conclusive (CDC,2021; IDF,2021).

In general, infections are more serious in people with diabetes. One reason is that diabetes affects the way the immune system works, making it harder for the body to fight viruses. Also, diabetes causes high blood sugar levels, and the International Diabetes Federation observes that the novel corona virus may thrive in an environment of elevated blood glucose. Diabetes also keeps the body in a low-level state of inflammation, which makes its healing response to any infection slower. High blood sugar levels combined with a persistent state of inflammation make it much more difficult for people with diabetes to recover from illnesses such as COVID-19 (IDF, 2021).

Akinwande *et al.* (2022) in their research Mathematical model of COVID-19 transmission dynamics incorporating booster vaccine program and environmental contamination, developed an SIR-type model that captured both human-to-human and environment-to-human-to-environment transmissions which allows the recruitment of corona viruses in the environment in the midst of booster vaccine program.

Okuonghae and Omame (2020) in their work, examines the impact of various non-pharmaceutical control measures on the population dynamics of the novel corona virus disease 2019 (COVID-19) in Lagos, Nigeria, using an appropriately formulated mathematical model Omame *et al.* (2022) presented and analyzed the fractional optimal control model for COVID-19 and diabetes co-dynamics, using the Atangana-Baleanu derivative. They established the positivity and boundedness of the solutions as was shown by the method of Laplace transform. The existence and uniqueness of the solutions of the model equation were established using Banach fixed point Theorem and Leray–Schauder alternative Theorem.

Kouidere *et al.* (2021) Pontryagin's maximum principle was used, to characterize the optimal controls and the optimality system as solved by an iterative method and some numerical simulations were performed to verify the theoretical analysis using MATLAB

In this paper a model was formulated for COVID-19 incorporating the comorbidity of diabetes, the global asymptotic stability was established using Castillo-Chavez theorem.

Model Formulation

The model equations were formulated using the first order ordinary differential equation, with the total human population at time t,denoted by N. the total population is further divided into fourteen (14) compartment, which are Susceptible to COVID-19(S_i), Diabetic without complication and also susceptible to COVID-19(S_d), Exposed to COVID-19(E_i), COVID-19 infectious (I_i), Quarantine for COVID-19(Q_i), Isolated for COVID-19(P_i), Recovered from COVID-19(R_i), Diabetics without complication and also COVID-19Exposed (E_d), Diabetics without complication and also COVID-19Infectious(I_d), Quarantine for COVID-19 and also diabetes without complication(Q_d), Isolated for COVID-19and diabetes without complication(P_d), Recovered from COVID-19but having diabetes without complication (R_d), Diabetics with complication and also COVID-19Infectious (I_c), Isolated for COVID-19and diabetes with complication (P_c).

A recruitment to the susceptible to COVID-19 class is at a constant rate β_i ,The COVID-19 susceptible individuals may acquire infection after effective contacts $\eta_{i1}, \eta_{i2}, \eta_{i3}, \eta_{i4}$ with Exposed to COVID-19, COVID-19infectious, Quarantine for COVID-19, and Isolated for COVID-19, the quarantined are recruited through a contact with the exposed class at rate $\sigma_i$and return to the susceptible at a rateξ_i.

The quarantined class become COVID-19 infectious at a rate ρ_i at a rate τ_i, COVID-19infectious is recruited to isolated for COVID-19 and subsequently γ_i, is the rate at which isolated become recovered thus some of the recruited will become susceptible while some will be recruited to the class of Diabetic without complication and also susceptible to COVID-19, again recruitment to the diabetic without complication and also susceptible to COVID-19class at a constant rate β_d .

The COVID-19 susceptible but diabetic without complication individuals may acquire infection after effective contacts $\eta_{d1}, \eta_{d2}, \eta_{d3}, \eta_{d4}, \eta_{d5}, \eta_{d6}$ $\alpha_d$with Diabetics without complication and also COVID-19Exposed, Diabetics without complication and also COVID-19Infectious, Quarantine for COVID-19and also diabetes without complication Isolated for COVID-19and diabetes without complication, Diabetics with complication and also COVID-19 Infectious, Isolated for COVID-19 and diabetes with complication, the quarantined are recruited through a contact with the exposed class at rate $\sigma_d$and return to the susceptible at a rate ξ_d. the quarantine class become COVID-19 infectious at a rate ρ_d, and a recruitment to COVID-19 infectious but diabetic with complication which move back to the susceptible class without complication and some to isolated with COVID-19 but diabetic with complication, at a rate τ_dCOVID-19 infectious is recruited to isolated for COVID-19 and subsequently γ_d.

All individuals who are alive may die naturally at a rate μ similarly infectious and isolated individuals experience an additional death burden occasioned by COVID-19, diabetes without complication and diabetes with complication at their respective rates $\delta_1, \delta_2, \delta_3$.

The force of infection is given by;

$$\alpha_i = m_i\left(\frac{\eta_{i1}E_i+\eta_{i2}Q_i+\eta_{i3}I_i+\eta_{i4}P_i}{N}\right) \quad (1)$$

$$\alpha_d = m_d\left(\frac{\eta_{d1}E_d+\eta_{d2}Q_d+\eta_{d3}I_d+\eta_{d4}P_d+\eta_{d5}I_c+\eta_{d6}P_c}{N}\right) \quad (2)$$

Where m_i, m_d the number of contacts made between the susceptible to COVID-19, Diabetic without complication and also susceptible to COVID-19, and the agents of virus transmission.

Assumptions of the Model

The models are formulated base on the following Assumptions:

i. The diabetes disease infections can either be without complication (acute) or with complication (chronic).
ii. There can be death due to complications from COVID-19.
iii. There can be death due to complications from acute diabetes.
iv. There can be death due to complications from chronic diabetes.
v. There is no recovery from COVID-19 when there is diabetes with complication.
vi. If the person that is co-infected with both COVID-19 and diabetes is effectively treated for diabetes, the person moves into the class of diabetes without complication but still COVID-19 infected.
vii. The people with diabetes are more likely to have more severe symptoms and complications when infected with COVID-19.
viii. We assume that persons who already have diabetes-complication are likely to have worse outcomes if they contract COVID-19.
ix. After effective treatment of COVID-19, persons from the recovered class may move back to diabetic without complications but susceptible to COVID-19 class after a while.

Based on the assumptions, the equations governing the co-infection dynamics is given as,

$$\frac{dS_i}{dt} = \beta_i - \alpha_i S_i + \eta_i R_i + \xi_i Q_i - \mu S_i \quad (3)$$

$$\frac{dS_d}{dt} = \beta_d - \alpha_d S_d + \omega I_c + \theta R_i + \eta_d R_d + \xi_d Q_d - \mu S_d \quad (4)$$

$\frac{dE_i}{dt} = \alpha_i S_i - A_{11} E_i$ (5)

$\frac{dQ_i}{dt} = \sigma_i E_i - A_2 Q_i$ (6)

$\frac{dI_i}{dt} = \rho_i Q_i - A_1 I_i$ (7)

$\frac{dP_i}{dt} = \tau_i I_i - A_3 P_i$ (8)

$\frac{dE_d}{dt} = \alpha_d S_d - A_{12} E_d$ (9)

$\frac{dQ_d}{dt} = \sigma_d E_d - A_5 Q_d$ (10)

$\frac{dI_d}{dt} = \rho_d Q_d - A_4 I_d$ (11)

$\frac{dP_d}{dt} = \tau_d I_d - A_6 P_d$ (12)

$\frac{dI_c}{dt} = (1-\alpha) I_d - A_7 I_c$ (13)

$\frac{dP_c}{dt} = \tau_c I_c - A_8 P_c$ (14)

$\frac{dR_i}{dt} = \gamma_i P_i - A_9 R_i$ (15)

$\frac{dR_d}{dt} = \gamma_d P_d - A_{10} R_d$ (16)

Were

$\tau_i + \delta_1 + \mu = A_1,\ \rho_i + \xi_i + \mu = A_2, \gamma_i + \delta_1 + \mu = A_3,\ 1 - \alpha + \tau_d + \delta_2 + \mu = A_4$
$\rho_d + \xi_d + \mu = A_5, \gamma_d + \delta_2 + \mu = A_6, \omega + \tau_c + \delta_3 + \mu = A_7,\ \delta_3 + \mu = A_8, \eta_i + \theta + \mu = A_9, \eta_d + \mu = A_{10},$
$\sigma_i + \mu = A_{11}, \sigma_d + \mu = A_{12}$

Results and Discussion

To establish the global stability of the COVID-19 Diabetes Free Equilibrium, we apply the theorem by Castillo-Chavez *et al.* (2002). We re-write the model as

$\frac{dX}{dt} = H(X,Z)$ (17)

$\frac{dZ}{dt} = G(X,Z), G(X,0) = 0$ (18)

Where $X = (S_i, R_i, S_d, R_d)$ (19)

And $Z = (E_i, Q_i, I_i, P_i, E_d, Q_d, I_d, P_d, I_c, P_c)$ (20)

with the components X denoting the uninfected population and components Z denoting the infected population.

The COVID-19- diabetes complication-free equilibrium is now denoted as

$E^0 = (X^*, 0);\ \ X^* = \left(\frac{\beta_i}{\mu}, 0, \frac{\beta_d}{\mu}, o\right)$ (21)

The following conditions must be satisfied to guarantee global asymptotical stability:

i. $\frac{dX}{dt} = H(X,0);\ X^*$ is globally asymptotically stable

ii. $G(X,Z) = PZ - \hat{G}(X,Z),\ \hat{G}(X,Z) \geq 0$for $(X,Z) \in \Omega,$ (22)

where $P = D_Z G(X^*, 0)$ is a M-matrix (the off-diagonal elements of P are non-negative) and Ω is the region where the model makes biological sense.

If the system satisfies the condition above, then the theorem below holds.

Theorem 1: The fixed point $E^0 = (X^*, 0)$ is globally asymptotically stable equilibrium of the system provided $R_0 < 1$that and the conditions in (22) are satisfied.

Proof: From the model system, we have

$$H(X,0) = \begin{pmatrix} \beta_i - \alpha_i S_i + \eta_i R_i - \mu S_i \\ -(\theta + \eta_i + \mu) R_i \\ \beta_d - \alpha_d S_d + \theta R_i + \eta_d R_d - \mu S_d \\ -(\eta_d + \mu) R_d \end{pmatrix} \quad (23)$$

$G(X,Z) = PZ - \hat{G}(X,Z)$

$$P = \begin{bmatrix} -c_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \sigma_i Q_i & -c_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \rho_i I_i & -c_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_i P_i & -c_4 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -c_5 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -c_6 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \rho_d I_d & -c_7 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \tau_d I_d & -c_8 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & (1-\alpha) I_c & 0 & -c_9 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \tau_c I_c & -c_{10} \end{bmatrix} \quad (24)$$

Where $c_1 = \sigma_i + \mu$, $c_2 = \xi_i + \mu$, $c_3 = \tau_i + \delta_1 + \mu$, $c_4 = \delta_1 + \gamma_i + \mu$ $c_5 = \sigma_d + \mu$, $c_6 = \xi_d + \rho_d + \mu$, $c_7 = \tau_d + \delta_2 + \mu$, $c_8 = \gamma_d + \delta_2 + \mu$, $c_9 = \tau_c + \delta_3 + \mu$, $c_{10} = \delta_3 + \mu$

$$P \times Z = \begin{bmatrix} -c_1 E_i \\ E_i Q_i \sigma_i - Q_i c_2 \\ I_i Q_i \rho_i - I_i c_3 \\ I_i P_i \tau_i - P_i c_4 \\ -c_5 E_d \\ -c_6 Q_d \\ I_d Q_d \rho_d - I_d c_7 \\ I_d^2 \tau_d - P_d c_8 \\ (1-\alpha) I_c I_d - c_9 I_c \\ I_c^2 \tau_c - P_c c_{10} \end{bmatrix} \quad (25)$$

$$G(X,Y) = \begin{bmatrix} (S_i \alpha_i - c_1) E_i \\ E_i Q_i \sigma_i - Q_i c_2 \\ I_i Q_i \rho_i - I_i c_3 \\ I_i P_i \tau_i - P_i c_4 \\ (S_d \alpha_d - c_5) E_d \\ -c_6 Q_d \\ I_d Q_d \rho_d - I_d c_7 \\ I_d^2 \tau_d - P_d c_8 \\ (1-\alpha) I_c I_d - c_9 I_c \\ I_c^2 \tau_c - P_c c_{10} \end{bmatrix} \quad (26)$$

$$\hat{G}(X,Z)=\begin{vmatrix} \left(-c_1+\alpha_i S_i'\right)E_i-\left(S_i\alpha_i-c_1\right)E_i \\ -E_i Q_i \sigma_i \\ -I_i Q_i \rho_i \\ -I_i P_i \tau_i \\ \left(-c_5+\alpha_d S_d'\right)E_d-\left(S_d\alpha_d-c_5\right)E_d \\ 0 \\ -I_d Q_d \rho_d \\ -I_d^2 \tau_d \\ -(1-\alpha) I_c I_d \\ -I_c^2 \tau_c \end{vmatrix} \quad (27)$$

From (27), it is clear that $\hat{G}(X,Z)<0$ so the conditions are not met. Hence, E^0 may not be globally asymptotically stable when $R_0<1$.

(Rothana and Byrareddy, 2020; Gumel *et al.*, 2020; Branswell, 2020; Dong *et al.*, 2020; Roda *et al.*, 2020; Sun and Wang, 2020; Contreras *et al.*, 2020; Li *et al.*, 2020; Zhu *et al.*,2020). (WHO, 2021). (Bai *et al.*, 2020). (Ngonghala*et al.*, 2020b). (Zhou *et al.*, 2020) (Huang *et al.*, 2020; Zhu *et al.*, 2020b). (Rothana and Byrareddy, 2020; Lu, 2020). (Rothana and Byrareddy, 2020) (CDC,2021; IDF,2021). Akinwande *et al.* (2022) Okuonghae and Omame (2020) Omame *et al.* (2022) Kouidere *et al.* (2021) Castillo-Chavez *et al.* (2002).

Conclusion

The Global Asymptotic Stability may not be stable when $R_0<1$ but may be stable otherwise, since the two Castillo-Chavez theorem conditions are not met, Hence, E^0 may not be globally asymptotically stable when $R_0<1$.

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