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Nonlinear Modeling and Stability Analysis of Glucose–Insulin Homeostasis

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Abstract

This study develops a nonlinear dynamic model for glucose-insulin regulation that includes glucose utilization, saturated insulin secretion, insulin clearance, and an auxiliary remote-effect variable capturing the delayed regulatory response. The proposed framework enables comprehensive investigation of glucose homeostasis from a dynamical systems perspective, emphasizing equilibrium structure, stability properties, and parameter-induced transitions. The positivity and bounds of the solution were established ensuring the physical acceptability . Analytical characterization of the equilibrium points reveals that the steady-state of glucose concentration satisfies a non-linear algebraic relationship, allowing parameter-dependent shifts in the metabolic regime. Local and global stability were investigated by Jacobian matrix analysis and the Lyapunov-cascade approach, showing that the system admits a unique globally asymptotically stable equilibrium under physically relevant conditions. Numerical simulations confirm the analytical results and show that variations in physiological parameters including external glucose input, insulin clearance rate, insulin secretion gain, and net insulin effectiveness consistently displace the equilibrium from normoglycemic to pathological hyperglycemic states without inducing oscillatory dynamics. It is important to mention that the transition to type 2 diabetes emerges as spontaneous equilibrium shifts and weakening of stability rather than bifurcation-driven oscillations as an mathematical simplification aiming to focus on the stability and equilibrium behavior of the system.These findings give a mathematically rigorous and physiologically interpretable explanation of metabolic flexibility and its gradual breakdown, providing a unifying framework for understanding glucose homeostasis and its impairment under adverse conditions.

Key words: Glucose–insulin dynamics, Equilibrium geometry, Global asymptotic stability, Lyapunov methods, Phase-plane analysis, Type-2 diabetes modeling.

1. Introduction

The glucose–insulin regulatory process can be modeled as a nonlinear dynamical system characterized by coupled feedback interactions and time-dependent behavior[1,2]. The dynamic relationship between glucose and insulin concentrations determines the stability of the system and its equilibrium states[3,4]. Mathematical modeling has been recognized as an efficient method to uncover non-linear interactions between glucose, insulin and regulatory control mechanisms[5,6]. Earlier mathematical models, including minimal models and compartment models of glucose/insulin homeostasis, have been helpful in understanding glucose tolerance and insulin sensitivity[7-9]. However, most existing mathematical models are mainly for the analysis of steady-state phenomena or short-term dynamics, which are not sufficient to identify nonlinear dynamic control mechanisms of glucose/insulin homeostasis, especially threshold phenomena, multi-stability and qualitative transitions[10-12].Dynamical systems theory has provided an

alternative approach, where the geometry of the phase plane, equilibrium, and stability of the glucose/insulin regulatory control process are of major interest. The role of saddle equilibria has been acknowledged as an important mechanism with separatix-induced transitions[13,14]. The main object of the current study is to develop and analyze a nonlinear dynamical model of glucose–insulin regulation that captures key physiological mechanisms. Where the study aims to investigate the equilibrium structure, establish the local and global stability conditions in addition to that examine how variations in physiological parameters influence transitions between normal and pathological metabolic states. The structured of this manuscript as follows: The proposed mathematical model is introduced in Section 2. The analytical results, including local and global stability analysis, are presented in Section 3. Numerical simulations and their interpretations are discussed in Section 4 followed by concluding remarks in Section 5.

2. Mathematical Modeling of Glucose–Insulin Dynamics

The glucose insulin regulatory system is given by the following nonlinear differential equations:

$$\begin{aligned} \frac{d\,G(t)}{dt} &= -k_1 G(t) + k_2 I(t) + D(t) - U(G,I) \\ \frac{d\,I(t)}{dt} &= -k_3 I(t) + f(G) - H(t) \\ \frac{d\,R(t)}{dt} &= \alpha_1 G(t) - \alpha_2 R(t) \end{aligned} \qquad \textbf{(1)}$$

where

- $G(t)$ represents the blood glucose concentration at time t.
- $I(t)$ represent the insulin concentration.
- $R(t)$ denotes a regulatory or remove insulin effect variable.
- $D(t)$ is the glucose input from meals or digestion .
- $U(G,I)$ represents the rate of glucose utilization by body tissues.
- $f(G)$ is the insulin secretion function in responses to glucose.
- $H(t)$ represents insulin clearance .
- k_1 Basal glucose removal rate constant independent of insulin action.
- k_2 Glucose production or release coefficient associated with insulin dynamics.
- k_3 Insulin degradation or clearance rate constant.
- α_1, α_2 are regulatory parameters governing the dynamics of $R(t)$.

We adopt the following physiologically motivated functional forms:

$U(G,I) = mG + nI, \quad f(G) = \frac{a\,G}{G+b} \quad m,n,a,b > 0,$ such that $n > k_2$. Substituting these expressions into system (1), the model reduces to

$$\begin{aligned} \frac{d\,G(t)}{dt} &= -(k_1 + m)G(t) + (k_2 - n)I(t) + D(t) \\ \frac{d\,I(t)}{dt} &= -k_3 I(t) + \frac{a\,G}{G+b} - H(t) \\ \frac{d\,R(t)}{dt} &= \alpha_1 G(t) - \alpha_2 R(t) \end{aligned} \qquad \textbf{(2)}$$

The above formulation represents a tractable and expressive model for the analysis of equilibrium and stability in the glucose insulin regulatory system, both under physiological and pathological conditions.

2.1 Positivity and Boundedness of Solutions

2.1.1Theorem If all parameters of the **System 2** are positive and that $D(t), H(t) \geq 0$, are bounded for all $t \geq 0$. then , for any initial condition in $\mathcal{R}^3 \geq 0$, the corresponding solution remains non-negative and bounded for all $t \geq 0$.

Proof : The non-negative orthant is forward invariant since the vector field does not point outward on its boundary. Moreover, the boundedness of the saturation function $0 \leq \frac{aG}{G+b} \leq a$,

implies that, $I(t) \leq k$, where $k = \max\{I(0), \frac{a}{k_3}\}$, and hence $I(t)$ is bounded . By comparison arguments, $G(t)$ is also bounded, and the linear R-dynamics driven by bounded $G(t)$ ensures bouneded $R(t)$.

2.2 Equilibrium Points Analysis

Let (G^*, I^*, R^*) denote an equilibrium points of system (2) .and set all the time derivatives to zero. From the third equation, the regulatory variable satisfies , $R^* = \frac{\alpha_1}{\alpha_2} G^*$. From the Insulin dynamics, the steady-state Insulin concentration is given by, $I^* = \frac{1}{k_3}(\frac{a\,G^*}{G^*+b} - H)$. Substitution this expression into the glucose balance equation yields a scalar equilibrium condition for G^*, $-(k_1+m)G^* + \frac{(k_2-n)}{k_3}(\frac{a\,G^*}{G^*+b} - H) + D = 0$, defining $= \frac{(k_2-n)}{k_3}$, and multiplying by (G^*+b), this condition can be written as the quadratic polynomial

$(k_1+m){G^*}^2 + \big((k_1+m)b - (D-\tau H) - \tau a\big)G^* + (D-\tau H)b = 0$, or equivalently ,

$\alpha G^2 + \beta G + \gamma = 0$

where $\alpha = (k_1+m)$, $\beta = \big((k_1+m)b - (D-\tau H) - \tau a\big)$ and $\gamma = (D-\tau H)b$,the equilibrium glucose concentrations are given by: $G^*_{1,2} = \frac{-\beta \pm \sqrt{\beta^2 - 4\alpha\gamma}}{2\alpha}$,provided that the discriminant satisfies $\beta^2 - 4\alpha\gamma > 0$, the product of the roots is $G^*_1 G^*_2 = \frac{\gamma}{\alpha} < 0$. Since > 0 , the sign of $G^*_1 G^*_2$ depends on γ if $\gamma < 0$ implying $G^*_1 G^*_2 < 0$. Consequently, one equilibrium glucose is positive while the other is negative. From a physiological standpoint , only the positive equilibrium $G^* > 0$ is biologically meaningful[15-17].

Special interest is the unforced (basal) equilibrium, corresponding to the absence of external inputs, $D = 0$ and $H = 0$. In this case, the equilibrium condition simplifies, and the system admits a single positive steady state representing the fasting glucose- insulin balance. Notably, under basal conditions the constant forcing term vanishes , eliminating the possibility of multi-stability and saddle-node bifurcations, and yielding a unique homeostatic equilibrium governed solely by intrinsic physiological parameters[18,19].

3. Local and Global Stability Analysis

The objective of this section is to determine the conditions under which the system returns to equilibrium after perturbations. In local asymptotic stability investigates the behavior of the system in a neighborhood of the equilibrium point using linearization techniques and in global asymptotic stability, which guarantees convergence to the equilibrium for all admissible initial conditions using a Lyapunov-based approach

3.1 Local asymptotic stability of the equilibrium

3.1.1.Theorem Then (G^*, I^*, R^*) is locally asymptotically stable if and only if $\theta f'(G^*) < \alpha k_3$, where $\theta = (k_2 - n)$.

Proof: Linearizing of system (2) at (G^*, I^*, R^*) yields the Jacobion

$$J(G^*) = \begin{bmatrix} -\alpha & \theta & 0 \\ f'(G^*) & -k_3 & 0 \\ \alpha_1 & 0 & -\alpha_2 \end{bmatrix}$$

Because R does not feed back into the (G, I)- subsystem, the Jacobian matrix is block lower-triangular, hence its spectrum consists of the eigenvalue $(-\alpha_2)$ and the eigenvalues of the (G, I)-subsystem matrix[20,21].

$$A = \begin{bmatrix} -\alpha & \theta \\ f'(G^*) & -k_3 \end{bmatrix}$$

Therefore, local asymptotic stability is equivalent to $\alpha_2 > 0$ and Hurwitz stability of the 2×2 matrix A. since $tr(A) < 0$, always hold for sufficient condition is $det(A)>0$, which completes the proof.

3.2. Global asymptotic stability via a Lyapunav-cascade argument

3.2.1. Theorem:Assume that $D(t) \equiv D$ and $H(t) \equiv H$ are constant and that the system admits an equilibrium (G^*, I^*, R^*). Let the error variables be $g(t) = G(t) - G^*$ and $i(t) = I(t) - I^*$. If the parameter condition $\frac{a}{b}\theta < \alpha k_3$, holds then the equilibrium (G^*, I^*, R^*) is globally asymptotically stable.

Proof: Subtracting the equilibrium equations from the system yields the incremental dynamics $g' = -\alpha g + \theta i$ and $i' = -ik_3 + \Delta f(G)$, where $\Delta f(G) = f(G) - f(G^*)$. Since f is globally Lipschitz on $G > 0$ with constant $L = sup_{G>0} f'(G) = \frac{a}{b}$, it follows that $|\Delta f(G)| \leq L|g|$. Consider the Lyapunov function $V(g, i) = \frac{1}{2}g^2 + \frac{p}{2}i^2, p > 0$. Its derivative along trajectories satisfies. $V' \leq -\alpha g^2 - pk_3 i^2 + (|\theta| + p\,L)|g||i|$. By young's inequality the mixed term can be absorbed into the negative quadratic terms provided $\theta|L| < k_3\alpha$. Under this condition there exist constants $c_1, c_2 > 0$ such that $V' \leq -c_1 g^2 - c_2 i^2$, implying that V is positive definite and radially unbounded with a strictly negative definite derivative. Hence $g(t) \to 0$ and $(t) \to 0$, globally as $t \to \infty$. The R −dynamics is a stable linear filter driven by G and therefore converges $R^* = \frac{\alpha_1}{\alpha_2}G^*$, completing the proof [22-25].

4. Numerical Results and Discussion

In this section the theoretical findings obtained in the previous analysis are numerically simulated to illustrate the results. The objective of this numerical simulation is to elucidate the role of variations in physical parameters on the equilibrium structure, stability properties, and geometric organization of the system. Particular emphasis is placed on comparing healthy and type-2 diabetic configurations, highlighting how pathological parameter variations distort nonlinear geometry, alter convergence patterns, and undermine metabolic flexibility. The presented data collectively demonstrate consistency between analytical stability results and numerical dynamics, offering a clear dynamic interpretation of glucose homeostasis and its progressive breakdown under adverse physiological conditions.

In **Figure 1** the healthy state glucose-insulin phase plane, in which the nullclines intersect to form locally stable equilibrium. The trajectories of the system converge to the equilibrium point over a wide range of initial conditions. This reflects the effective role of insulin in regulating glucose levels, strong metabolic stability, and well-regulated glucose homeostasis.

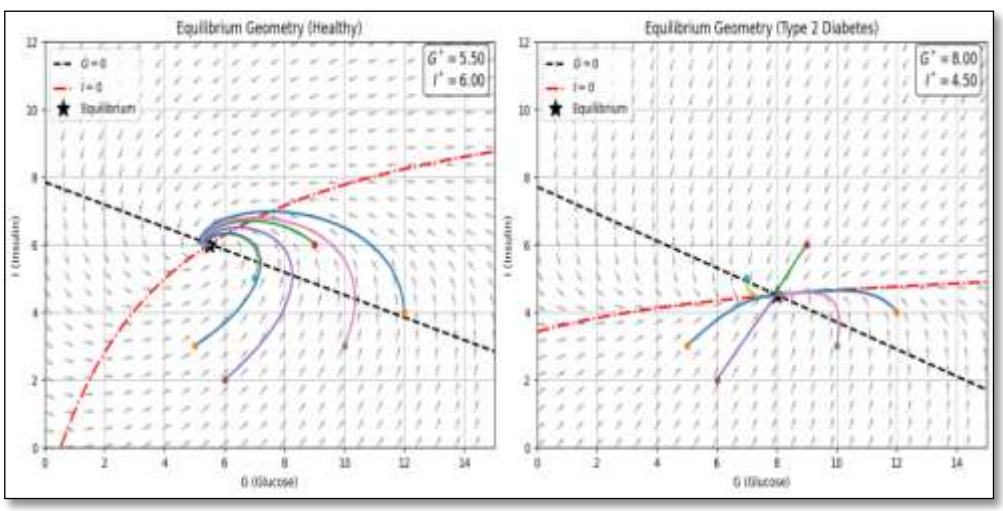

Figure 1. Glucose–insulin phase-plane showing a unique equilibrium that shifts from normoglycemic (healthy) to hyperglycemic (Type-2 diabetes) conditions

The vector field reflects the stabilizing role of feedback mechanisms on glucose levels, which respond to disturbances in glucose levels. In addition to that this figure showed type-2 diabetic

state, in which the glucose-insulin balance is shifted to higher glucose levels. Distortion of the nonlinear geometry results in a weakened stabilization role of the feedback mechanism on glucose levels. The trajectories gradually converge to equilibrium, indicating weak metabolic stability. Physiologically, such geometric rearrangement of the system explains the loss of metabolic robustness observed in type-2 diabetes, where small perturbations can induce a prolonged hyperglycemic regime, even when balance is nominally present.

Figure 2 present phase-plane analysis of steady state glucose-insulin balance for healthy and type-2 diabetes states. In both cases, all trajectories are seen to converge to a stable node, indicating local asymptotic stability via the Jacobian matrix eigenvalues. Physiologically, in a healthy state, glucose concentrations are low and insulin concentrations are high, whereas in a type-2 diabetic state, a stable equilibrium is observed, but glucose concentrations are high due to the ineffectiveness of insulin.

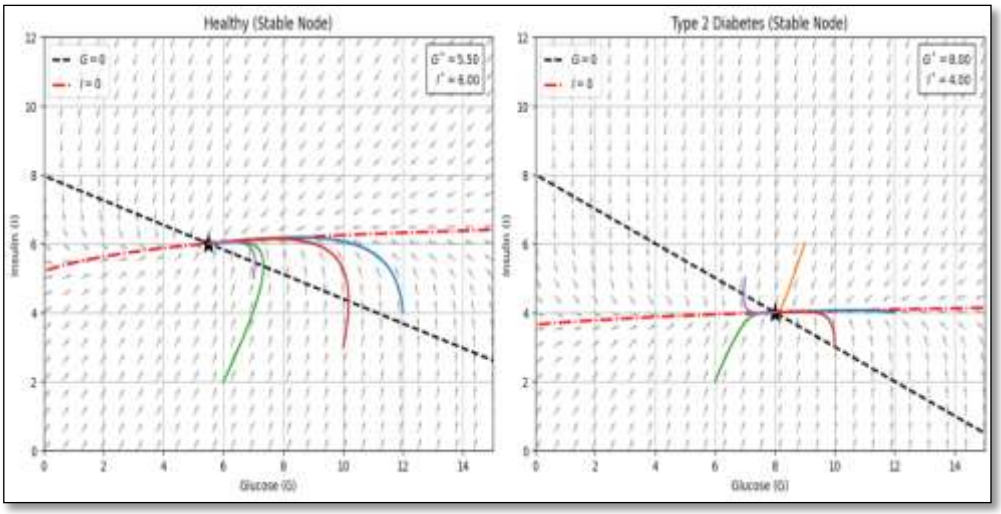

Figure 2. Comparative Phase-Plane Dynamics of Healthy and Type-2 Diabetic Glucose–Insulin System

The phase-plane diagrams in **Figure 3** present fixed-focus equilibria for healthy and type-2 diabetes states. In both cases, the trajectory is seen to spiral into a stable equilibrium point, where glucose and insulin concentrations intersect. Physiologically, the healthy state is seen transitioning to a lower glucose set-point with higher insulin concentrations, whereas the type-2 diabetes state sees a stable equilibrium but with higher glucose concentrations due to ineffectiveness of insulin

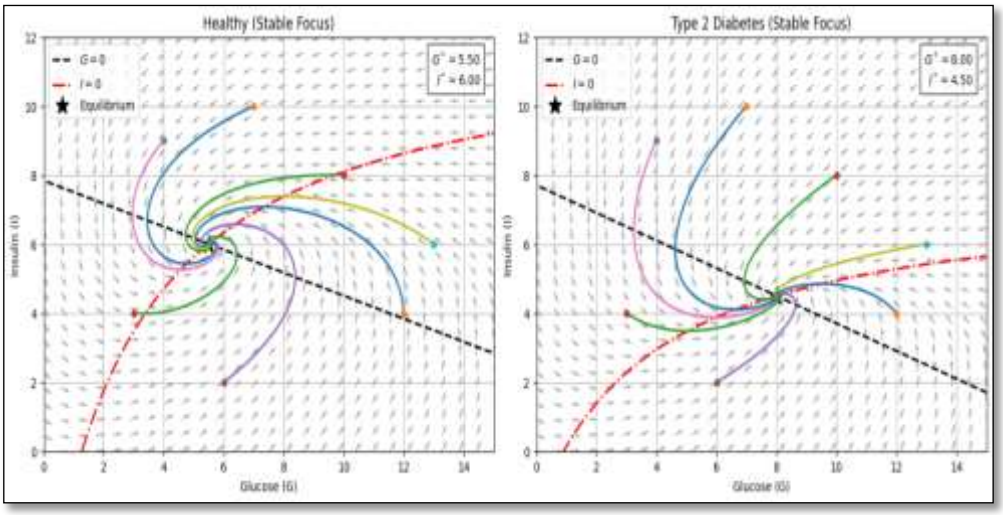

Figure 3. Glucose–Insulin Phase-Plane: Stable Focus in Health and Diabetes

In **Figure 4** the global asymptotic stability of the glucose-insulin regulatory system. **Figure 4a** shows the time evolution of the system states under different initial conditions. It is clear that all system states converge to a single equilibrium point. This is true regardless of the initial condition applied to the system. This shows that this system has a global basin of attraction. **Figure 4b** shows the error dynamics corresponding to the glucose-insulin system. It is clear that the errors converge to zero over time. This indicates that the attractiveness of this system is on a global scale. This is true because there are no oscillations in the error dynamics. **Figure 4c** shows the time evolution of the Lyapunov function V(t). It is clear that the Lyapunov function strictly decreases as time evolves. This demonstrates that the glucose-insulin system has a global attraction zone. This is true because the Lyapunov function strictly goes to zero as time evolves. This verifies that the chosen Lyapunov function satisfies the necessary conditions. In particular, the fact that the Lyapunov function strictly reduces to zero as time this establishes that the system has a global basin of attraction. This is true because as shown above, the system states converge to a single equilibrium point. Furthermore, the fact that the Lyapunov function strictly reduces to zero as time evolves confirms the fact that the system has a global basin of attraction.

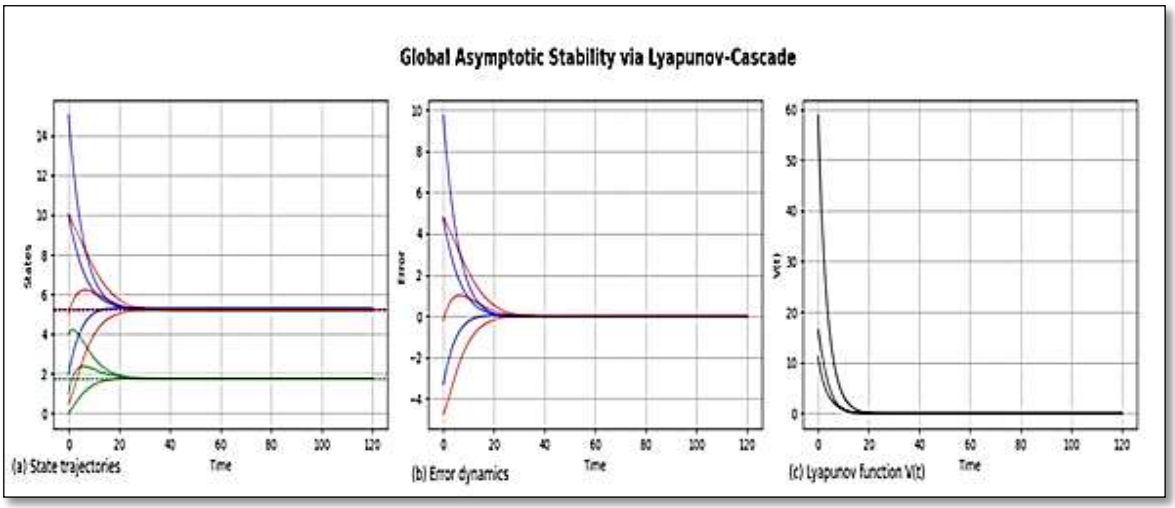

Figure 4. Global asymptotic stability of the glucose–insulin system shown by convergence of states, decay of errors, and monotonic decrease of a Lyapunov function.

Figure 5 presents the dependence of glucose concentration on the input parameters. Smoothly and monotonically increasing curves of equilibrium with respect to input parameters indicate that the system shifts to higher glucose levels with increased input. This does not cause dynamic instability. Healthy, pre-diabetic and type-2 diabetic regimes are classified on the basis of glucose levels. The difference between pre-diabetic and type-2 diabetic regimes is due to the application of different diagnostic criteria on the continuum curve. This implies that the transition to type 2 diabetes is a gradual change in a pathological homeostatic state.

Figure 6 presents the effect of insulin clearance parameters on steady-state glucose levels and the associated glucose-insulin phase-plane dynamics. The upper left panel presents the smoothly and monotonically increasing curve of the steady-state glucose level with respect to the input parameters. This means that the system shifts to higher glucose levels with increased insulin clearance. Healthy, pre-diabetic and type-2 diabetic regimes are classified on the basis of glucose levels. The phase-plane diagram shows that the system has a unique equilibrium point for all arrangements. This occurs due to the intersection of glucose and insulin nullclines. This shows that the system is asymptotically stable. The unique balance point shifts to higher glucose and lower insulin levels as insulin clearance increases. This implies that the system shifts to a pathological homeostatic state with increased insulin clearance. This suggests that the transition to type 2 diabetes is a gradual change in a pathological homeostatic state.

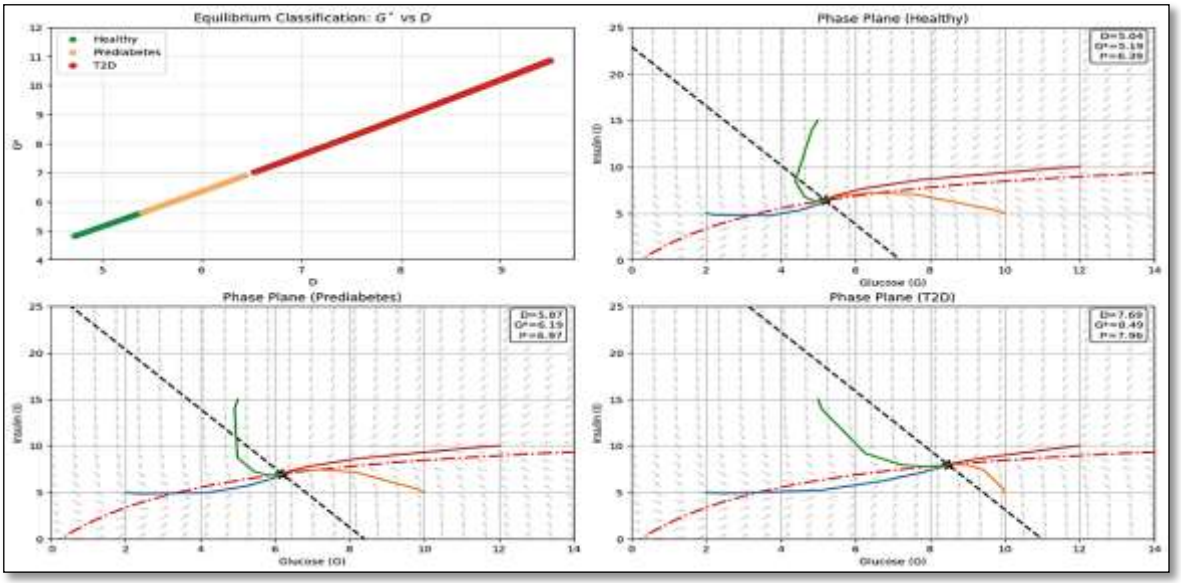

Figure 5. Equilibrium Glucose Level as a Function of External Glucose Input

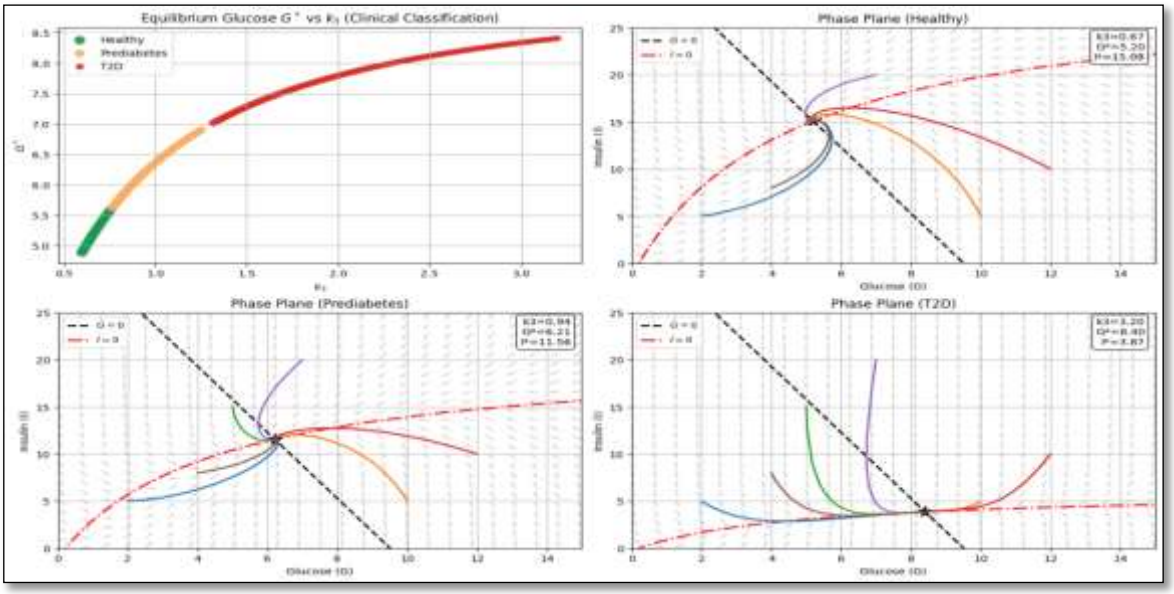

Figure 6. Effect of Insulin Clearance on Glucose Homeostatic Equilibrium

Figure 7 examines the effect of the insulin secretion gain parameter on the equilibrium glucose level and the associated glucose-insulin phase plane dynamics. The upper left panel of **Figure 7** shows a monotonic decrease with respect to the parameter, meaning that the increase in pancreatic insulin secretion results in a decrease in the steady-state glucose level of the system. The various clinical settings of healthy, pre-diabetic and type-2 diabetic states are represented using standard ranges for these states. Phase plane plots show that for all values, the system has a unique equilibrium point determined by the intersection of the glucose and insulin lines. As depletion occurs, the insulin nullcline moves downward, resulting in decreased insulin effect and shifting the balance point horizontally toward higher glucose concentrations. The trajectories of the system converge to the equilibrium point in all regimes, confirming the asymptotic stability of the system dynamics. Physiologically, these results imply that a reduction in the body's ability to secrete insulin results in a stable but pathological hyperglycemic state and not a loss of system stability.

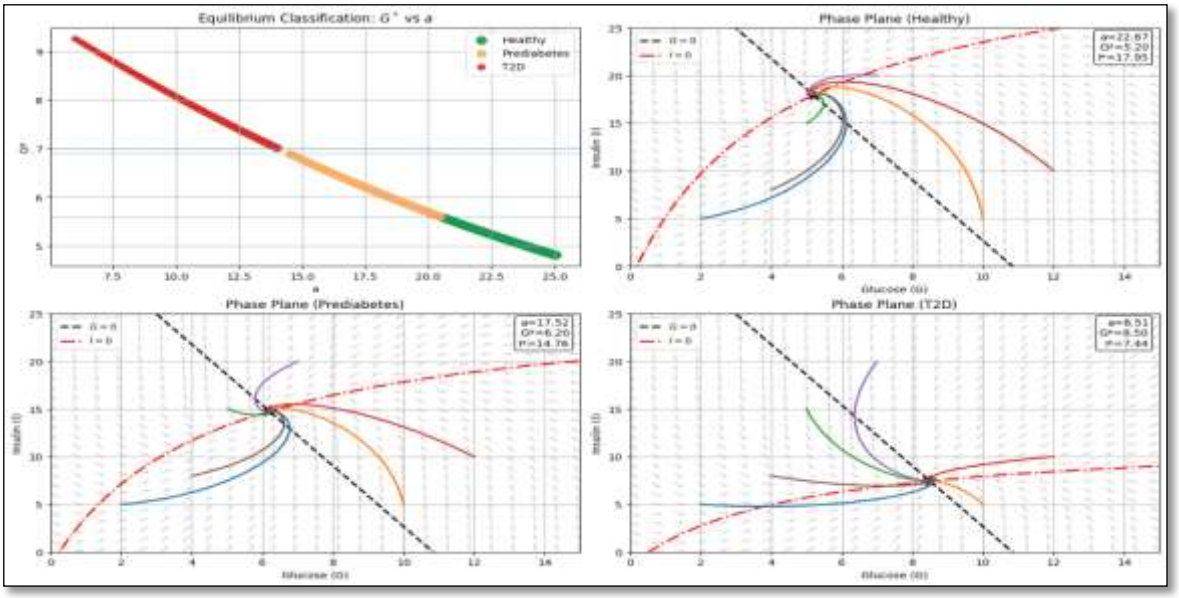

Figure 7. Effect of Insulin Secretion Gain on Glucose Homeostatic Equilibrium

Figure 8 examines the effect of parameters on the equilibrium glucose level and glucose/insulin phase plane dynamics. As glucose input approaches zero, a smooth and monotonous increase is seen in the upper left panel. This suggests that decreased insulin effectiveness has a tendency to increase steady-state glucose concentrations. Healthy individuals, prediabetic individuals, and type-2 diabetic patients are identified using standard diagnostic criteria. Phase plane plots show that the system has a unique equilibrium point that is determined by the intersection of the glucose and insulin line for all regimes. As the parameter approaches zero, the glucose nullcline shifts, moving the equilibrium point toward higher glucose concentrations while maintaining asymptotic stability. This suggests that from a physiological perspective, progression toward a state of reduced insulin effectiveness continually drives the system toward a stable hyperglycemic state.

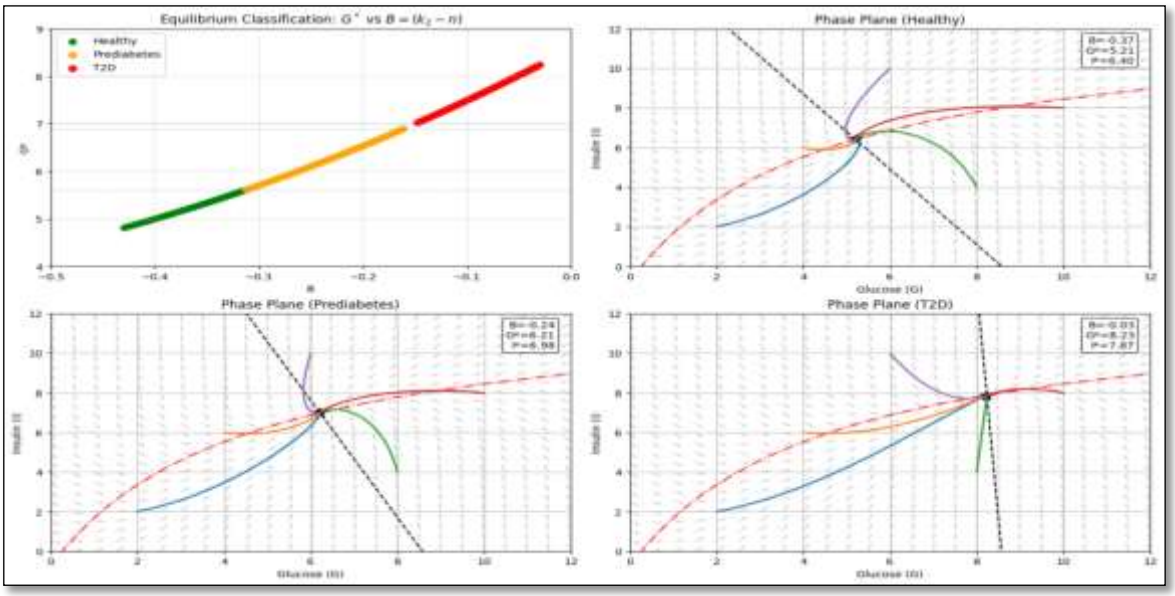

Figure 8. Effect of Net Insulin Effectiveness on Glucose Homeostatic Equilibrium

5. Conclusion

The present work has provided a new perspective on glucose/insulin regulation, showing that progress toward different regimes of metabolic health and disease is determined by equilibrium geometry and stability structure rather than oscillatory instability. The model has shown that different regimes of glucose/insulin regulation, both physiological and pathological, correspond to a class of structurally stable equilibria that are organized along parameter-dependent manifolds. Progression to type-2 diabetes is characterized by a continual displacement of stable homeostatic equilibrium with a concomitant loss of regulatory flexibility. Systematically varying glucose input, insulin clearance, insulin secretion gain, and insulin effectiveness revealed that the phase plane geometry is reshaped by shifting attractor points and basins of attraction while maintaining stability. Hyperglycemia has thus been shown to be a persistent yet maladaptive stable equilibrium state that explains the chronic self-perpetuating nature of type-2 diabetes.

From a physiological perspective, these findings suggest that the deterioration of metabolic control is not necessarily associated with dynamic instability, but rather with a gradual reorganization of regulatory set-points driven by parameter variations. This interpretation is consistent with clinical observations where type-2 diabetes develops progressively without pronounced oscillatory behavior in glucose–insulin dynamics. Moreover, in contrast to several existing models that emphasize oscillatory or bifurcation-driven transitions, the present framework highlights equilibrium displacement as the dominant mechanism underlying disease progression, thereby offering a complementary and unifying interpretation. This adds value by bridging mathematical stability theory with clinically observed gradual metabolic deterioration, and provides a clearer conceptual understanding of how stable yet pathological states can emerge and persist in glucose homeostasis.

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Conflict of Interest

The authors declare that they have no conflicts of interest.

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