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Stability Analysis of a Three-Dimensional Discrete Topp Model

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Abstract Mathematical models of glucose, insulin, and pancreatic beta cell mass dynamics are essential for understanding the physiological basis of type 2 diabetes. This paper investigates the discrete-time dynamics of the Topp model to represent these interactions. We perform a comprehensive analysis of the system's trajectory, examining both local and global behavior. First, we establish the invariance of the positive trajectory and analyze the existence of fixed points. Then, we conduct a complete stability analysis, determining the local and global asymptotic stability of these fixed points. Finally, numerical examples validate the effectiveness and applicability of our theoretical findings. Additionally, we provide biological interpretations of our results.

Keywords Fixed point, periodic point, local stability, global behavior, regular operator

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1. Introduction

To sustain proper human body function, it is crucial to regulate blood glucose levels within the range of $70 - 100 mg/dl$. Insulin, secreted by beta cells in the pancreas, aids in the uptake of glucose by cells and is vital for maintaining blood sugar balance. Elevated blood glucose levels stimulate insulin secretion, which assists in reducing the concentration to a healthy range. As glucose levels drop, insulin secretion progressively diminishes. This mechanism, involving both insulin and glucagon, is essential for maintaining glucose equilibrium and preventing complications associated with diabetes [14, 26, 28].

Mathematical representations of glucose, insulin, and pancreatic beta cell mass dynamics are essential for comprehending the physiological mechanism underlying the onset of type 2 diabetes. Traditionally, type 2 diabetes was thought to arise from insulin insufficiency. The mathematical model introduced by Topp and collaborators

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is significant for examining its progression. Topp's model serves as a cornerstone for investigating the advancement of diabetes [26]. The Topp model is

$$\begin{cases} \frac{dx}{dt} = g_0 - g_1 x - cxy, \\ \frac{dy}{dt} = \frac{s_1 x^2}{s_2 + x^2} z - ky, \\ \frac{dz}{dt} = (-d_0 + r_1 x - r_2 x^2) z. \end{cases} \tag{1.1}$$

In this model, x, y, and z represent the plasma glucose concentration, insulin concentration, and the mass of functional β-cells (capable of appropriate insulin production and secretion) at time t, respectively. g_0 represents the average rate of glucose infusion per day, including hepatic glucose production, primarily from meal ingestion. $g_1 x$ represents insulin-independent glucose uptake, mainly by brain and nerve cells, while cxy depicts insulin-dependent glucose uptake, primarily by fat and muscle cells. The coefficient c represents insulin sensitivity. Insulin secretion from β-cells is assumed to follow a Hill function with a coefficient of 2, triggered by increased glucose levels, where s_1 represents the secretory capacity per β-cell. The insulin clearance rate is denoted by k. The functional β-cell mass is hypothesized to respond to glucose in a parabolic manner: moderate glucose levels promote growth, while high glucose levels exacerbate apoptosis, leading to a decrease in functional β-cell mass. d_0 represents the death rate at zero glucose, and r_1, r_2 are constants [26, 28].

In recent years, the mathematical modeling of the glucose-insulin regulatory system has been extensively studied. As indicated by recent reviews, a large number of authors are using mathematical modeling as pragmatic and theoretical tools to allow scientific understanding and efficient management of diabetes in all its aspects (Al Ali et al., 2025 [1]; Boutayeb and Lamlili, 2025 [4]; Boutayeb et al., 2025 [5]; Lamlili et al., 2025 [16]; Nasir and Mat Daud, 2022 [18]).

Li and Kuang (2007) analyzed the glucose-insulin regulatory system using a delay-based model, investigating its dynamic properties [17]. Al-Hussein et al. (2020) introduced a new time-delay model that captures chaotic behaviors in the system [2]. Subsequently, Al-Hussein, Rahma, and Jafari (2020) further examined Hopf bifurcation and chaos in this model, providing insights into its complex dynamics [3].

Fernández-Carreón et al. (2021) analyzed a fractional-order version of a mathematical model of the glucose-insulin regulatory system [13]. Rao et al. (2023) extended this analysis by considering the effects of the insulin-degrading enzyme and multiple delays, offering a more comprehensive understanding of the system's behavior [19]. Additionally, Zhao et al. (2023) developed a dynamic model based on experimental observations in mice to explore the impact of glucagon-like peptide on glucose-insulin interactions [29].

The original Topp model [26] describes the glucose, insulin, β-cell feedback system using a continuous-time framework with three nonlinear ordinary differential equations. This model revealed the existence of two stable fixed points representing physiological (healthy) and pathological (diabetic) states, and has been widely used to investigate the progression of diabetes through bifurcation analysis and numerical simulations.

Following this foundational work, several studies have investigated extensions of the Topp model, including parameter sensitivity, bifurcation behavior, and the influ-