

Glucose–Insulin Interaction System for Type 1 Diabetes Mellitus Patients Based on an Adaptive Aquila Optimization–Tilt Acceleration



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ABSTRACT: Type-I Diabetes Mellitus (T1DM) requires continuous external insulin regulation, and artificial pancreas (AP) systems offer closed-loop control through glucose sensing, control computation, and micro-insulin dispensing. Glucose-insulin dynamics are nonlinear, time-varying, patient-specific, and affected by delays, meal/exercise disturbances, sensor noise, and parameter uncertainty. Conventional PID and advanced controllers often show limited robustness, slow response, and poor adaptability. This study designs an Aquila Optimization–Tilt Acceleration Derivative with Filter (AO-TADF) controller to regulate insulin delivery and maintain Blood Glucose (BG) within 70–120 mg/dl. A multi-compartment Lehmann glucose–insulin patient model is implemented in SIMULINK. Meal and exercise disturbances are included. Linearization at 81 mg/dl and basal insulin 22.3 mU/min yields state-space matrices. TADF combines tilt, acceleration, and derivative actions with pre-filters, gains, and shaping coefficients. The Aquila Optimizer, mimicking high soaring, contour gliding, low-altitude flight, and prey capture, tunes parameters by minimizing integral time absolute error. The closed-loop AP includes glucose sensing, the AO-TADF controller, and a micro-insulin dispenser. Performance is tested under a 60 g meal at 600 min, exercise at 1300 min, hepatic/peripheral insulin sensitivity variations, and noise. BG stabilized at 81–83.3 mg/dl with no renal glucose excretion. This controller achieved 230 min settling time, 58.2 mU/min insulin infusion, 3.9 mg/dl peak overshoot, 1.0 mg/dl undershoot, zero steady-state error, mean BG 84 mg/dl, and standard deviation 1.5, outperforming PID, fuzzy, sliding mode, LQG, and H_∞ controllers. The AO-TADF controller provides robust, stable, and accurate glycemic regulation, and promising for practical AP implementation, with future validation using clinical simulators and real-time embedded platforms.

KEYWORDS: glucose; insulin; artificial pancreas; optimization; controller.

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I. INTRODUCTION

Diabetes mellitus, as highlighted by the WHO, is one of the most widespread chronic diseases and is primarily caused by pancreatic dysfunction. This impairment leads to insufficient insulin production or reduced insulin sensitivity, thereby disrupting the maintenance of BG within the normal or norm-glycemic range. In individuals with T1DM, the pancreas fails to produce adequate insulin, making external insulin administration essential for survival. Consequently, maintaining BG levels within a safe physiological range remains a complex and critical task. To address this challenge, researchers have focused on developing advanced medical technologies, particularly automated insulin delivery systems such as the AP.

In traditional practice, BG regulation relied on open-loop control strategies, where insulin was administered manually based on periodic glucose measurements. However, such approaches are limited in their ability to respond to dynamic

physiological changes and external disturbances, including meal intake, stress, and physical activity. As a result, patients are often at the risk of experiencing hypoglycemic or hyperglycemic episodes. To overcome these shortcomings, closed-loop control systems have been introduced, enabling continuous monitoring and automated insulin delivery based on real-time sensor data.

A typical AP system comprises three essential components: a glucose sensor, a controller, and a Micro-Insulin Dispenser (MID). The glucose sensor continuously monitors the patient's BG levels and transmits the measured data to the controller. The controller processes this information and generates an appropriate control signal, which is then sent to the MID. The MID delivers the calculated insulin dose into the patient's body, thereby forming a feedback loop that ensures effective glycemic regulation. The integration of the T1DM patient model with the AP system in a closed-loop configuration is illustrated in Fig. 1, where continuous feedback facilitates stable and

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reliable BG control. The nomenclature and abbreviations used throughout this study are systematically listed in APPENDIX A and APPENDIX B, respectively.

Despite significant progress in AP technology, several challenges remain in achieving optimal BG regulation. The Glucose–Insulin (GI) dynamics are inherently nonlinear and time-varying, with considerable inter- and intra-patient variability. These complexities are further compounded by uncertainties in model parameters, delays in glucose sensing, and slow insulin absorption rates following infusion. Additionally, disturbances such as unannounced meal intake and variations in physical activity introduce further unpredictability into the system. The asymmetric response of BG levels increases the likelihood of extreme fluctuations, making it difficult to maintain stable control. These issues have been extensively discussed in literature (Chee et al., 2003; Kamath et al., 2009).

To address these challenges, numerous control strategies have been proposed. The PID controller has been widely used due to its simplicity and ease of implementation (Sutradhar et al., 2002; Frederick et al., 2003). However, PID controllers are limited by fixed gain parameters and their inability to effectively handle nonlinearities, time delays, and uncertainties. As a result, their performance in achieving robust and precise BG regulation is often inadequate.

To overcome these limitations, advanced control techniques such as Fuzzy Logic Control (Ibbini et al., 2006; Panigrahi, et al., 2020), SM Control (Rout et al., 2017; Rout et al., 2018), LQG Control (Rout et al., 2015; Patra et al., 2020; Nanda et al., 2020), H-infinity (H_∞) Control (Chee et al., 2005; Karimpour et al., 2012; Patra et al., 2014), and MP Control (Mishra et al., 2020; Mangi, et al., 2009; Gampetelli et al., 2013) have been investigated. Although these methods offer improved performance compared to PID controllers, they still face challenges related to robustness, stability, and adaptability under varying operating conditions. In many cases, these controllers exhibit limited bandwidth, slower response dynamics, and reduced effectiveness in handling uncertainties and disturbances.

Furthermore, existing control strategies often fail to guarantee stability in the presence of model mismatches and external perturbations. Their inability to provide fast and reliable responses to sudden changes, such as postprandial glucose excursions, highlights the need for more advanced and adaptive control approaches.

In this context, the present study proposes a novel control strategy based on the Aquila Optimization–Tilt AO-TADF controller. The proposed approach integrates an adaptive control framework with the AOA to enhance system performance. AOA is employed to optimally tune the controller parameters, offering advantages such as rapid convergence, improved accuracy, and consistent performance. Additionally, AOA provides strong global and local search capabilities and is relatively simple to implement compared to other optimization techniques.

The AO-TADF controller introduces a filtering mechanism into the TAD structure. This filtering process enables accurate estimation of system states and facilitates rapid convergence toward the equilibrium point. Consequently, the

proposed controller achieves enhanced precision, improved robustness, effective noise attenuation, and superior handling of uncertainties. Moreover, the filtering strategy contributes to the development of a stabilizing control law, ensuring reliable system performance under diverse patient conditions.

Another significant feature of the proposed approach is its adaptability. The AO-TADF controller can effectively respond to disturbances such as meal intake, parameter variations, and physiological changes, thereby maintaining BG levels within the desired normo-glycemic range. Compared to existing control techniques, the proposed method demonstrates improved resilience against both matched and mismatched uncertainties, making it a promising solution for real-time glucose regulation in T1DM patients.

The major contributions of this work can be summarized as follows. First, a numerical patient model based on the glucose–insulin process is developed for effective BG monitoring. Second, an AO-TADF controller is designed to regulate insulin delivery and achieve normo-glycemia. Third, the performance of the proposed controller is evaluated under abnormal and uncertain conditions to demonstrate its robustness and reliability. Finally, a comprehensive comparative analysis is conducted to validate the superiority of the proposed approach over existing control strategies.

The remainder of this paper is organized as follows. Section II describes the GI process in detail. Section III presents the design methodology of the AO-TADF controller. Section IV provides simulation results and numerical analysis. Finally, Section V concludes the study with key findings and observations.

II. THEORETICAL AND CONCEPTUAL FRAMEWORK

Section II presents the clinical background, formulates the nonlinear physiological model, and analyses BG dynamics across various organs in patients with T1DM.

A. Clinical History

Diabetes mellitus, commonly known as hyperglycemia, refers to a group of metabolic disorders characterized by persistently elevated BG levels, typically exceeding 144 mg/dl. This condition arises due to insufficient insulin secretion, impaired insulin action, or a combination of both. In simple terms, diabetes occurs when the body cannot effectively utilize glucose, which is the primary source of energy for cellular functions. Based on insulin production and effectiveness, diabetes is classified into Type I and Type II. In Type I diabetes, the pancreas is unable to produce insulin, whereas in Type II diabetes, insulin is produced but released more slowly and exhibits reduced activity. Under normal conditions, the body regulates glucose levels efficiently despite variations in food intake, fasting, and physical activity (Lehmann et al., 1992; Lehmann et al., 1998). Glucose plays a vital role in sustaining metabolic activities and is mainly derived from carbohydrate-rich foods. During digestion, carbohydrates are broken down in the gastrointestinal tract, and glucose is absorbed into the bloodstream through the Venous Blood (VB). This externally

derived glucose is transported to the liver, where excess amounts are stored as glycogen. When BG levels decline, the liver converts glycogen back into glucose and releases it into the bloodstream, thereby serving as an internal source of glucose. At the cellular level, glucose undergoes oxidation in

the presence of oxygen to produce energy, along with carbon dioxide and water as by-products. Insulin, secreted by pancreatic β -cells, regulates this process by promoting glucose uptake and facilitating its storage as glycogen, ensuring stable and balanced blood glucose levels.

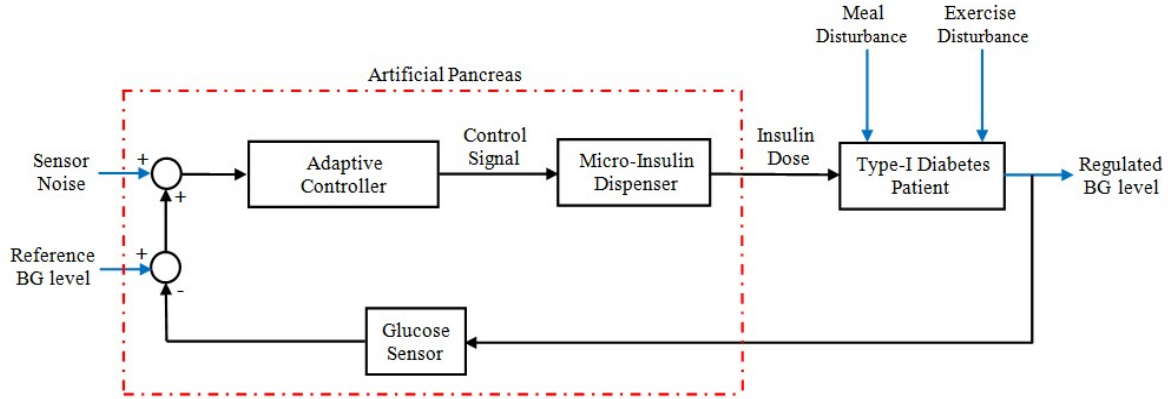


Figure 1 A representative practical depiction of an AP integrated with a T1DM patient.

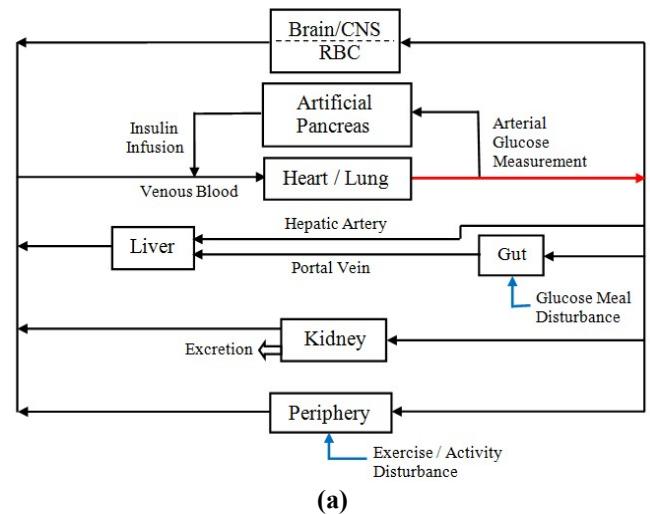
This function becomes especially important during meal intake, when glucose levels rise significantly. Under normal conditions, insulin prevents excessive internal glucose production by the liver and muscles. It also promotes glucose uptake in muscle tissues to satisfy peripheral energy demands. However, in individuals with diabetes, abnormalities in metabolic kinetics impair these processes either partially or completely. As a result, cells fail to utilize glucose efficiently, while the liver continues to produce additional glucose, leading to uncontrolled blood glucose levels. Furthermore, when blood glucose exceeds approximately 162 mg/dl, the kidneys begin to eliminate excess glucose from the vascular system through excretion.

B. Structure of the Patient

Over the past few decades, GI dynamics have been extensively studied to understand how the GI system regulates blood glucose levels, leading to the development of several mathematical models for analysis and control design. Among these, the model proposed by Lehmann et al. (1992; 1998) has gained wide acceptance due to its structural simplicity and its close resemblance to human metabolic behaviour. Owing to these advantages, this model has been widely applied for simulating physiological processes and evaluating different control strategies. In the present study, the same model is adopted to assess the effectiveness of the proposed controller in maintaining desired glucose levels. Fig. 2(a) illustrates the GI system integrated with an AP, consisting of six main compartments: peripheral tissues, kidney, stomach, liver, brain, and heart. These compartments collectively represent the major physiological interactions involved in glucose regulation. The liver plays a central role by producing and releasing glucose, while the gastrointestinal tract facilitates glucose absorption from ingested food.

Continuous monitoring of AG is essential, as it provides real-time input to the AP system. The MID administers insulin into the VB at regular intervals, typically every five

minutes, in synchronization with sensor measurements. The sampling interval is determined by the characteristics of the sensing and actuation mechanisms employed. In this model, meal intake and physical activity are treated as disturbances affecting the stomach and peripheral compartments, respectively. By analysing insulin dynamics, glucose flow, and their interactions, the complete GI process in a diabetic patient is represented. Furthermore, Fig. 2(b) presents a simplified version of the GI model, highlighting key glucose and insulin pathways. Solid arrows indicate glucose transport, while symbols such as $1+$ and $1-$ denote the enhancement and suppression of glucose flow due to insulin action.



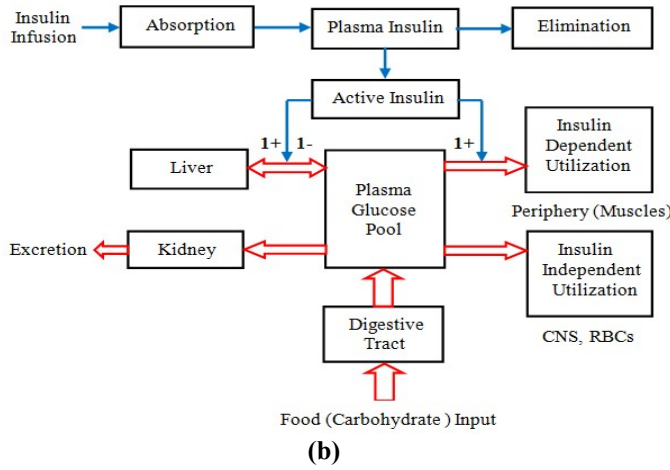


Figure 2 (a) Modeling and representing the GI process's compartments; (b) Patient's structure and the GI interaction.

1) Effects of Insulin

As blood glucose levels increase, the AP delivers an appropriate insulin dose into the VB. The dynamics of insulin action within the VB are described in (Lehmann et al., 1992; Lehmann et al., 1998):

$$\frac{dI_p(t)}{dt} = \frac{I_{abs}(t)}{V_I} - K_e I_p(t) \quad (1)$$

Value of K_e and V_I are expected as 0.123 min^{-1} and 99.4 dl distinctly. The differentiation of $I_a(t)$ in the VB is stated as:

$$\frac{dI_a(t)}{dt} = K_1 I_p(t) - K_2 I_a(t) \quad (2)$$

The value of K_1 and K_2 are interpreted as 0.00042 min^{-1} and 0.021 min^{-1} separately. The $I_{ea}(t)$ and $I_{ep}(t)$ within the VB and is mentioned in Eqs. (3) and (4) accordingly.

$$I_{ea}(t) = S_p (K_2 / K_1) I_a(t) \quad (3)$$

$$I_{ep}(t) = (S_h / I_{basal}) I_p(t) \quad (4)$$

The permissible range for the insulin sensitivity parameters (S_p and S_h) is considered between 0 and 1.0, while the value of the specified basal insulin (I_{basal}) is fixed at 1.0 mU/dl . The dynamic behaviour of insulin actions during the modification process is represented in Eqs. (1)–(4).

2) Digestive System's Glucose Concentration Rate

The “gastric emptying rate” describes the speed at which the Gastric Emptying Subsystem (GES) generates glucose $G_{empt}(t)$, as illustrated in Fig. 3(a, b). The function $G_{empt}(t)$ reaches a maximum value of $V \max_{ge}$ (360 mg/min) and then gradually declines to zero. The overall profile of $G_{empt}(t)$ is governed by the amount of carbohydrate (Ch) intake. As shown in Fig. 3(a), a triangular pattern is observed when the carbohydrate consumption is below the critical threshold Ch_{crit} (10.8 gm). Furthermore, the

value of Ch_{crit} can be determined using Eq. (5). The parameters $Tasc_{ge}$ and $Tdes_{ge}$ are defined in Eq. (6). Fig. 3(b) depicts a trapezoidal profile of $G_{empt}(t)$ when carbohydrate (Ch) intake is equal to or exceeds the critical threshold Ch_{crit} . In this framework, both $Tasc_{ge}$ and $Tdes_{ge}$ are fixed at 30 minutes. The term $T \max_{ge}$ represents the duration over which $G_{empt}(t)$ reaches its maximum value $V \max_{ge}$, as mathematically expressed in Eq. (7).

$$Ch_{crit} = [(Tasc_{ge} + Tdes_{ge}) V \max_{ge}] / 2 \quad (5)$$

$$Tasc_{ge} = Tdes_{ge} = Ch / V \max_{ge} \quad (6)$$

$$T \max_{ge} = [Ch - (1/2) V \max_{ge} (Tasc_{ge} + Tdes_{ge})] / V \max_{ge} \quad (7)$$

The $G_{empt}(t)$ with Ch intake more than Ch_{crit} is categorized as:

$$\begin{aligned} G_{empt}(t) &= (V \max_{ge} / Tasc_{ge}) t ; \text{ for } t < Tasc_{ge} , \\ &= V \max_{ge} ; \text{ for } Tasc_{ge} < t \leq Tasc_{ge} + T \max_{ge} , \\ &= V \max_{ge} - (V \max_{ge} / Tdes_{ge})(t - Tasc_{ge} - T \max_{ge}) ; \\ &\text{ for } Tasc_{ge} + T \max_{ge} \leq t < T \max_{ge} + Tasc_{ge} + Tdes_{ge} , \text{ and} \\ &= 0 ; \text{ for } t \text{ otherwise.} \end{aligned} \quad (8)$$

Here, t denotes the elapsed time since the previous meal intake. The profiles of $G_{empt}(t)$, in both triangular and trapezoidal forms, are identified and utilized for computational analysis, as described in Eq. (8). A first-order filter is employed to estimate the rate of glucose appearance in the gut based on the input signal $G_{empt}(t)$.

$$\frac{dG_{gut}(t)}{dt} = G_{empt}(t) - G_{in}(t) \quad (9)$$

$$G_{in}(t) = K_{gabs} G_{gut}(t) \quad (10)$$

The parameter K_{gabs} is assumed to be 0.017 min^{-1} . Fig. 3(c) illustrates the rate at which the stomach absorbs glucose for carbohydrate (Ch) intakes of 10 gm and 60 gm . This behavior is derived from the application of $G_{empt}(t)$ and is modeled using a first-order filter, as clearly represented in Fig. 3(a) and (b) for the two respective cases. The adjustable parameters defined in Eqs. (5)–(8) are analyzed and implemented for the GES model. In addition, the dynamic conditions of the stomach, described by Eqs. (9)–(10), are also considered and incorporated into the analysis. The numerical representation of the gut dynamics is presented in Fig. 4(a).

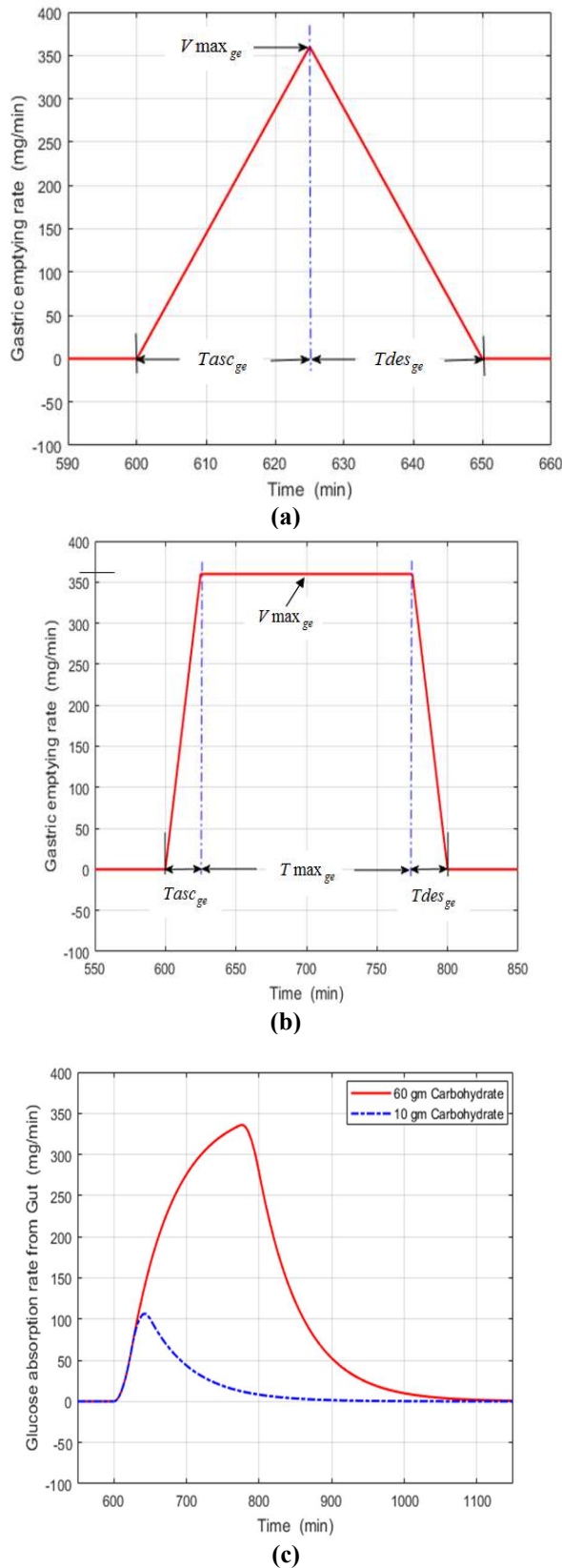


Figure 3 (a) Response of $G_{empt}(t)$ under 10gm Ch absorption ($Ch < Ch_{crit}$); (b) Response of $G_{empt}(t)$ under 60gm Ch absorption ($Ch > Ch_{crit}$); (c) Gut response under 10gm and 60gm Ch absorption.

3) Glucose dynamics across the liver, CNS, cells, and RBCs

The liver is primarily responsible for the regulation and handling of carbohydrates and is characterized as NHGB. Its glucose behaviour is governed by two key limiting components, namely effective active insulin $I_{ea}(t)$ and AG. These variables are determined through a look-up table method, using data compiled from previous studies (Lehmann et al., (1992, 1998)). The computational framework of the liver model is depicted in Fig. 4(a). Cellular glucose utilization depends on both effective active insulin $I_{ea}(t)$ and the plasma insulin concentration $I_p(t)$. The rate of glucose metabolism within cells is determined using a look-up table, with supporting data derived from the literature. The mathematical representation of cellular dynamics is illustrated in Fig. 4(a). RBC and the brain function independently of insulin. The carbohydrate consumption rates of the CNS and RBC are approximately 72mg/hr/kg and 12.96mg/hr/kg, respectively. Their corresponding numerical models are presented in Fig. 4(a).

4) Kidney Glucose Outlines

A fraction of glucose in the VB is eliminated through the kidneys when the BG level surpasses a specific threshold. This process is referred to as renal glucose excretion $G_{ren}(t)$. The rate of $G_{ren}(t)$ is governed by both the glucose concentration $G(t)$ and CCR , as formulated in Eq. (11). When $G(t)$ falls below or equals the RTG , the excretion rate becomes zero, as indicated in Eq. (12). Based on the study, CCR and RTG are assumed to be 1dl/min and 160mg/dl, respectively. The functional behaviour of the kidney can therefore be described as follows:

$$G_{ren}(t) = CCR(G(t) - RTG) \quad \text{if } G(t) > RTG \quad (11)$$

$$G_{ren}(t) = 0 \quad \text{if } G(t) \leq RTG \quad (12)$$

An illustration of the kidney's analytical organization is shown in Fig. 4(a). It is expressed in relation to the terminology used in mathematics as indicated by the Eqs. (11) and (12).

5) BG concentration within the VB

The dynamic expression governing the BG level within the VB is given as follows:

$$\frac{dG(t)}{dt} = \frac{[G_{in}(t) + G_{liver}(t) - G_{out}(t) - G_{ren}(t)]}{V_G} \quad (13)$$

In this study, the glucose distribution volume in the VB (V_G) is assumed to be 175 dl. The patient's anatomical representation is illustrated in Fig. 4(a), and its mathematical description is provided through Eqs. (1)–(13).

6) Micro-Insulin Dispenser

Reference (Cochin, 1997) proposes a specialized MID adopted in this study. The device operates based on a variable pumping rate mechanism to deliver insulin accurately. Its key components include a storage capsule,

C. Linearized TIDM Patient Model

The block diagram illustrates the control system for a T1D patient, showing the interaction between various physiological and control subsystems. The main components and their interconnections are as follows:

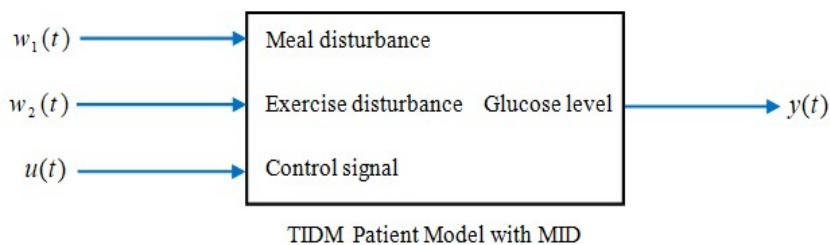
- Micro insulin dispenser:** Receives a control signal $u(t)$ (Insulin dose in mU/min) and outputs an insulin dose $u[1]$ (mU/min). It also receives a feedback signal from the glucose level.
- Gastric emptying sub-system:** Receives chyme intake (gm) and outputs chyme rate (mg/min), which is then converted to glucose rate (mg/min) by the G_{gut} block.
- Kidney sub-system (Gren rate):** Receives plasma glucose level G and outputs glucose rate (mg/min) to the G_{out} block.
- CNS sub-system:** Receives plasma glucose level G and outputs a control signal $u[2]$ (mU/min) to the micro insulin dispenser.
- RBC (Red Blood Cells):** Receives plasma glucose level G and outputs a control signal $u[3]$ (mU/min) to the micro insulin dispenser.
- Look-Up Tables (NHGB):** These tables provide non-linear gain blocks based on plasma glucose level G and other variables like AG (arterial glucose) and RTG (retardation time).
- Mathematical Models:** The system includes several first-order linear dynamics blocks, gain blocks (e.g., $1/60$, $1/2.5 \times 70$), and integrators ($1/s$).
- Key Variables and Signals:**
 - G : Plasma glucose level (mg/dl)
 - AG : Arterial glucose (mg/dl)
 - RTG : Retardation time (mg/dl)
 - $u[1]$: Insulin dose (mU/min)
 - $u[2]$: Control signal (mU/min)
 - $u[3]$: Control signal (mU/min)
 - G_{gut} : Glucose rate (mg/min)
 - G_{out} : Glucose rate (mg/min)
 - G_{ren} : Glucose rate (mg/min)
 - G_{uptake} : Glucose uptake (mg/min)

The diagram shows a complex feedback loop where the micro insulin dispenser adjusts the insulin dose based on the plasma glucose level, which is influenced by the gastric emptying, kidney, and CNS subsystems. The RBC subsystem also provides a feedback signal to the micro insulin dispenser.

(a)

(b)

Figure 4 (a) Simulation model of the MID integrated with the patient system; (b) Structural representation of the simulated MID.



The state-space formulation of the patient model, incorporating process disturbances, is expressed as follows (Sutradhar et al. 2004):

$$\left. \begin{aligned} \frac{dx_m(t)}{dt} &= A_m x_m(t) + B_m u(t) + B_d w(t) \\ y(t) &= C_m x_m(t) + D_m u(t) + v(t) \end{aligned} \right\} \quad (14)$$

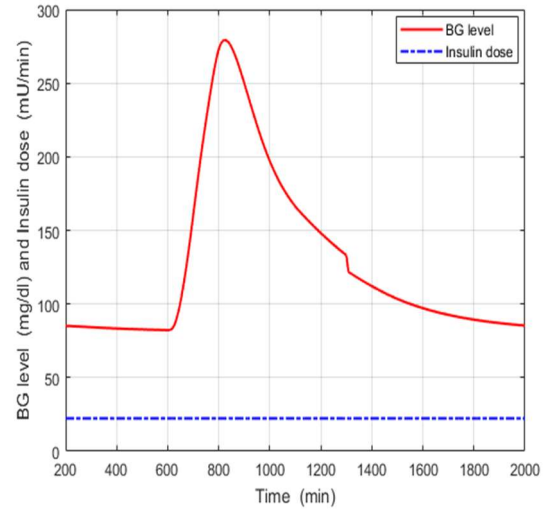
where $w(t)$ shows disruption in the system and it contains two main parts such as meal intake $w_1(t)$ with exercising $w_2(t)$. That $x_m(t)$, $v(t)$, as well as $y(t)$ denote state variables, Sensor noise, as well as that patient model's measured output, accordingly, whereas A_m , B_m , C_m , D_m and B_d deploys the patient model state-space matrices. The patient dynamics are linearized about a chosen reference operating point to obtain the corresponding state-space matrices. The detailed derivation of this process is well documented in the literature (Nanda et al., 2021). In this study, the baseline operating conditions are defined by a BG level of 81 mg/dl and a basal insulin infusion rate of 22.3 mU/min. Using MATLAB's *linmod* function, the state-space matrices of the SIMULINK-based T1DM patient model are computed, as illustrated in Fig. 4(a). This linearization around the operating condition facilitates the design of robust controllers capable of maintaining BG levels within the normo-glycemic range.

D. Study of Dynamic Behavior of the Patient

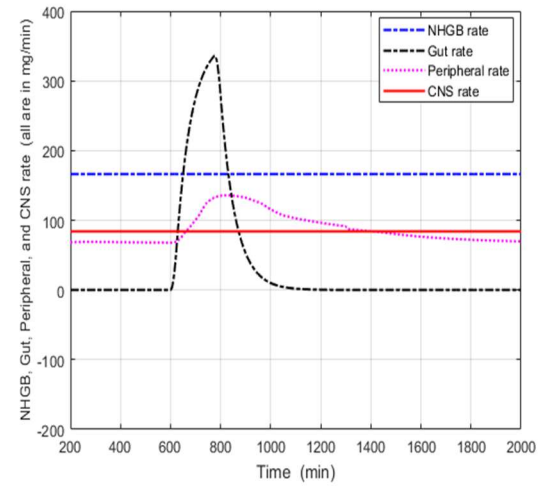
This section presents the variation of several physiological variables associated with BG metabolic processes in the time domain. The analysis considers a scenario in which a 60.00 g meal is consumed at 600 minutes, followed by physical exercise at 1300 minutes under a Gaussian disturbance environment. In this study, a constant insulin infusion, referred to as the basal insulin dosage, is applied throughout the simulation. Fig. 6(a) illustrates the insulin delivery profile along with the corresponding transient fluctuations in plasma glucose concentration within the VB. Fig. 6(b) depicts irregularities in key metabolic rates, including Net Hepatic Glucose Balance (NHGB), gut absorption rate, renal glucose excretion, and glucose uptake by body cells and the Central Nervous System (CNS).

Among various organs, the liver and peripheral tissues play a dominant role in insulin-dependent glucose regulation. In the absence of sufficient insulin, these tissues exhibit reduced glucose uptake, leading to a significant rise in BG levels beyond the recommended threshold of 144 mg/dl, thereby resulting in hyperglycemia. When BG levels exceed the Renal Threshold Glucose (RTG), the kidneys begin to excrete excess glucose from the VB. This relationship between BG levels and renal excretion is illustrated in Fig. 6(c).

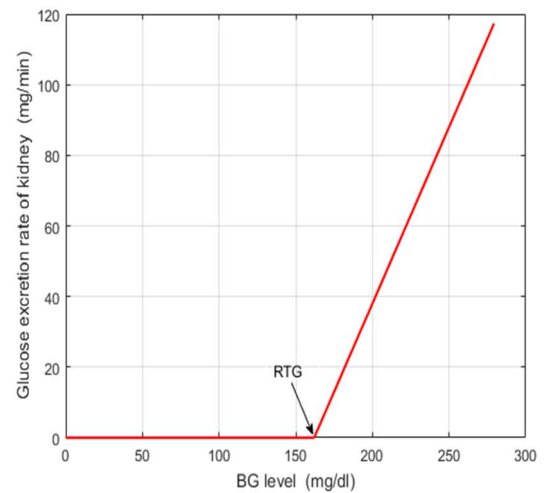
The observations from Fig. 6(a–c) indicate that the patient model exhibits pronounced transient responses, with BG levels consistently exceeding normal limits. These results highlight the system's susceptibility to disturbances, uncertainties, and instability. Therefore, it becomes essential to develop an advanced AP employing a robust and adaptive control strategy to effectively manage these challenges.



(a)



(b)



(c)

Figure 6 Characteristic responses of patient model: (a) variation of BG with insulin action; (b) glucose production and utilization rates across GI organs; (c) renal glucose excretion dynamics with respect to BG levels.

III. MATERIALS AND METHOD

The addition subsections provide a clear description of the AO-TADF control strategy. Using this controller, the patient system is evaluated in terms of stability, accuracy, and robustness. Key performance indices like settling time t_s (min), steady-state error e_{ss} (%), peak overshoot O_{Peak} , and peak undershoot U_{Peak} are analyzed to thoroughly assess the effectiveness of the AO-TADF controller.

A. AO-TADF Controlling Device

Fig. 1 illustrates the configuration of the patient integrated with the AO-TADF controller. The mathematical expression of the proposed controller is given as follows (Nanda et al., 2021; Cochin et al., 1997; Biswal et al., 2022; Kar et al., 2024; Panigrahi et al., 2023):

$$TF = K_t s^n + K_d \left(\frac{N_1 s^\alpha}{N_1 + s^\alpha} \right) + K_a \left(\frac{N_2 s^\gamma}{N_2 + s^\gamma} \right) \left(\frac{N_3 s^\beta}{N_3 + s^\beta} \right) \quad (15)$$

where, the control parameters are represented by tilt gain (K_t), acceleration gain (K_a), derivative gain (K_d), coefficient of tilt (n), pre-filter gains (N_1, N_2, N_3), coefficient of accelerations (γ and β) and coefficient of derivative (α). The proposed AO-TADF controller is an extension of classical PID-type structures, developed to better address the nonlinear and time-varying dynamics of the glucose–insulin system. The tilt term (K_t) represents a modified proportional action, where the coefficient n introduces a nonlinear (or fractional-like) scaling of the error signal. This allows improved sensitivity for large deviations (e.g., *hyper-glycemia*) while maintaining smooth control near the set point. The acceleration term (K_a) corresponds to a higher-order dynamic component, effectively representing the rate of change of the derivative of the error. This term enhances the controller's ability to anticipate rapid glucose variations, such as those occurring after meal disturbances, thereby improving transient response.

The derivative term (K_d) retains its conventional role of predicting error trends and providing damping to reduce overshoot. Together, these three terms form a multi-order control structure that improves robustness, responsiveness, and stability compared to conventional PID/PDF controllers, particularly in handling delays, uncertainties, and nonlinearities in physiological systems. The pre-filters (N_1, N_2, N_3) in the TADF controller serve as first-order low-pass filters applied to the tilt, acceleration, and derivative components of the control signal. Their primary function is to attenuate high-frequency noise arising from glucose sensors and measurement uncertainties, which is critical in physiological systems. In addition, the filters help to prevent derivative kick caused by sudden changes in the reference signal and reduce amplification of noise typically associated with derivative actions. By smoothing the intermediate signals, the pre-filters ensure a more stable and continuous control output, leading to improved robustness and

reliability. Overall, they enhance the practical implement ability of the controller by balancing responsiveness with noise suppression in real-time glucose regulation. The evaluation of control parameters are based on the response of proposed model for the lowest value of the fitness function ($ITAE$). Eq. (16) highlights the mathematical equivalence of $ITAE$. The lowest value of fitness function is responsible for better performance such as less overshoot, undershoot, and better monitor glucose levels of proposed model.

$$ITAE = \int_0^{\infty} |e(t)| dt \quad (16)$$

The AO technique is employed to determine and optimize control parameters for effective monitoring and regulation of BG levels.

B. Optimization Technique

The AO algorithm is inspired by the hunting strategies of Aquila. In AO, a solution population X is generated based on the number of candidates N and problem dimensions D , forming an $N \times D$ matrix. The selection of each candidate is defined by Eq. (17).

$$X_{ij} = rand \times (UB_j - LB_j) + LB_j \quad (17)$$

where, X_{ij} , $rand$, UB_j and LB_j represents candidate of i^{th} row and j^{th} column in X , an arbitrary value between 0 and 1, Upper limit of j^{th} dimension, lower limit of j^{th} dimension accordingly. Here, i changes from 1 to N , j changes from 1 to D . Aquila's hunting strategy can be categorized into four main phases:

- High soaring and vertical dives, indicating wide-area exploration.
- Short gliding along contours, representing targeted search.
- Low-altitude flight with gradual descent, corresponding to extended exploitation.
- Ground movement and prey capture, reflecting intensive local exploitation.

In the high soaring and vertical diving phase, Aquila scans a large region to locate potential prey, which is mathematically interpreted as the expanded exploration stage. This phase is denoted symbolically, and its mathematical representation is provided in Eq. (18).

$$X_1(t+1) = X_{best}(t) \times \left(1 - \frac{t}{T} \right) + (X_M(t) - X_{best}(t) \times rand) \quad (18)$$

where, The maximum number of iterations is shown by T , momentary iteration is illustrated by t , the upcoming iteration's resolution is depicted by $X_1(t+1)$, the best option or the location of the prey is exhibited by $X_{best}(t)$ and $X_M(t)$ represents the mean location of the current iteration. In its second phase, the aquila begins hounding and cornering its prey. This stage is referred to as narrowed exploration or contour flying with brief glide. A mathematical expression of the narrowed attempt stage is used to symbolize the narrowed exploration by the Eq. (19).

$$X_2(t+1) = X_{best}(t) \times L(D) + X_R(t) + (y - x) \times rand \quad (19)$$

Whereas, the second mode's answer for the subsequent iteration is $X_2(t+1)$, $L(D)$ indicates the levy propagation for D , $X_R(t)$ signifies the arbitrarily chosen solution for i^{th} iteration, y and x clarifies the Aquila's spiral descent shape. In the third phase, the AO algorithm shifts to the exploitation stage. During this stage, Aquila performs low-altitude flight, operating either in an extended exploitation

mode or a brief descent phase. It closely observes the movement and behavior of its prey to refine its search. This stage is represented symbolically as X_3 .

Table 1 Optimized control parameters of the AO-TADF controller.

K_t	K_d	K_a	n	N_1	N_2	N_3	α	β	γ
0.31	8	0.05	0.36	13.8	9.7	2.4	0.63	0.31	0.15

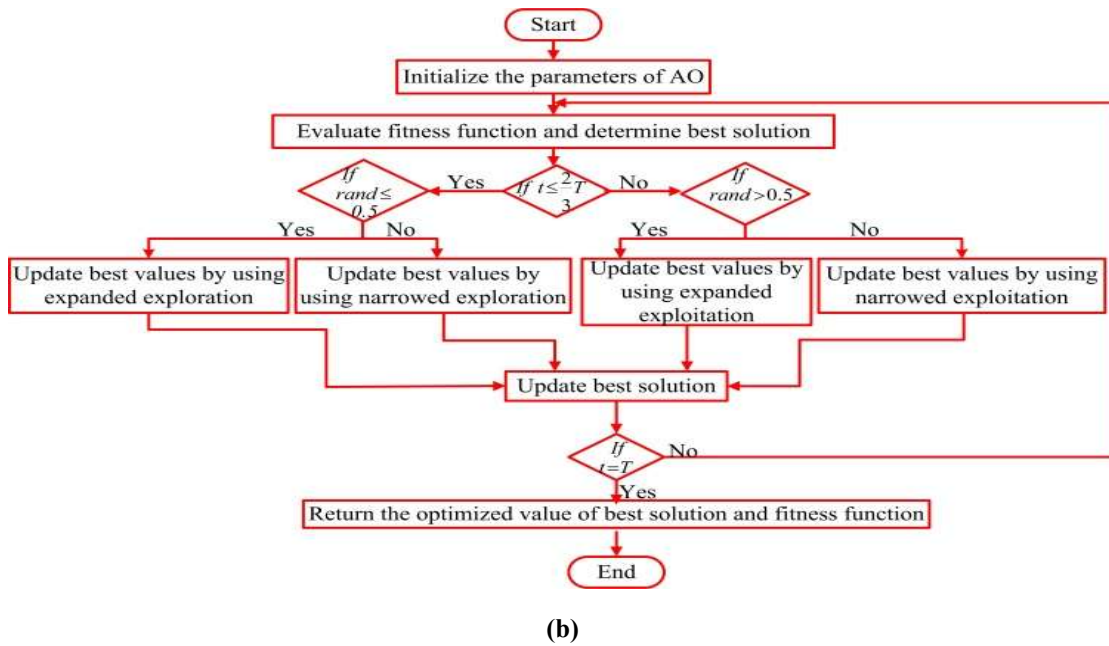
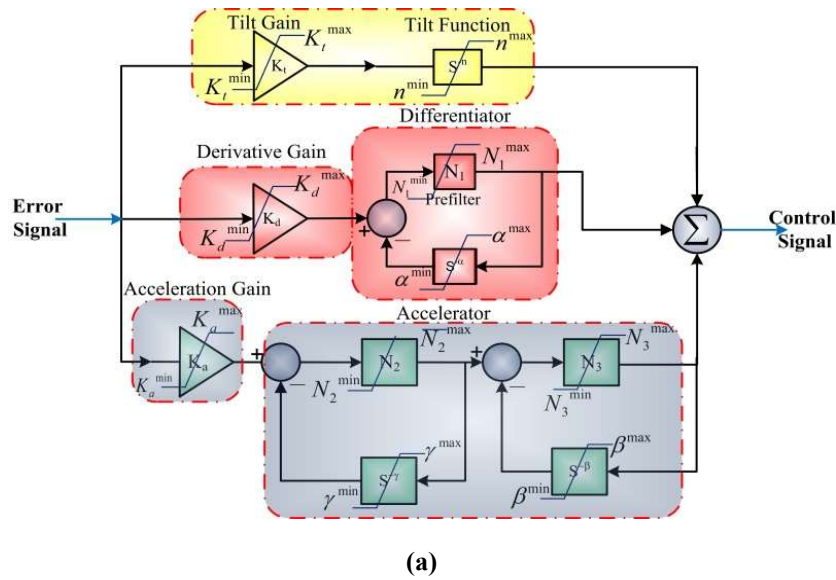


Figure 7 (a) Block diagram representation of the AO-TADF controller; (b) Flowchart illustrating the operational procedure and steps of the AO algorithm.

The mathematical formulation describing the expanded exploration phase is presented in Eq. (20), which defines its operational behaviour within the optimization process.

$$X_3(t+1) = \alpha \times X_{best}(t) - \alpha \times X_R(t) - rand + \delta \times rand \times (UB - LB) + \delta \times LB \quad (20)$$

In contrast, the solution for the next iteration in the second mode is given as $X_3(t+1)$, The α and δ depicts the parameters for the exploitation stage adjustment. Aquila mimics its landing in the final mode while bringing down its prey. This stage represents a phase of slow movement and initial prey capture, indicating a constrained or partial exploitation process, and is symbolically expressed as X_4 . Mathematical expression for the expanded exploration phase depicted by Eq. (21).

$$X_4(t+1) = QF \times X_{best}(t) - G_1 \times X(t) \times rand - G_2 \times L(D) + rand \times G_1 \quad (21)$$

where, the second mode's provided solution for the subsequent iteration is $X_4(t+1)$, QF represents with the quality function to achieve equilibrium between search approaches, $X(t)$ represents the outcome at i^{th} iteration, G_1 and G_2 illustrates Aquila's motion during the hunting technique. The parameters of the optimization method are set such that the optimum control parameter values are promptly attained. Until to the halt point (maximum iteration), the optimization process continues to be carried out once again. The values of the optimal control parameters are chosen in accordance with the lowest value of $ITAE$. The optimal controller gains are chosen on the basis of the AOA as mentioned in Table 1. Further TADF's overall architecture is depicted in Fig. 7(a), which has been developed on the basis of Eq. 15. The Fig. 7(a) has been significantly improved to present a clear and detailed block diagram of the proposed AO-TADF controller along with explicit signal flow explanations. The diagram now begins with the reference BG signal, which is compared with the measured BG obtained from the sensor to generate the error signal $e(t)$.

This error signal is then passed through three distinct control paths: tilt, acceleration, and derivative components. Each path is equipped with dedicated pre-filters (N_1, N_2, N_3), which play a crucial role in attenuating measurement noise and smoothing high-frequency fluctuations before further processing. The filtered signals are then scaled by their respective gains (K_t, K_a, K_d) and shaped using coefficients (α, β, γ, n) to achieve the desired dynamic response. These three processed signals are summed to produce the final control action. The resulting control signal $u(t)$ is applied to the MID, which administers the appropriate insulin dosage to the patient model. The updated BG level is continuously sensed and fed back to the comparator, thereby closing the loop. This structured signal flow ensures improved stability, robustness, and precise glucose regulation under varying physiological conditions. An intricate flow

chart that illustrates the AO technique's operation is shown in Fig. 7(b).

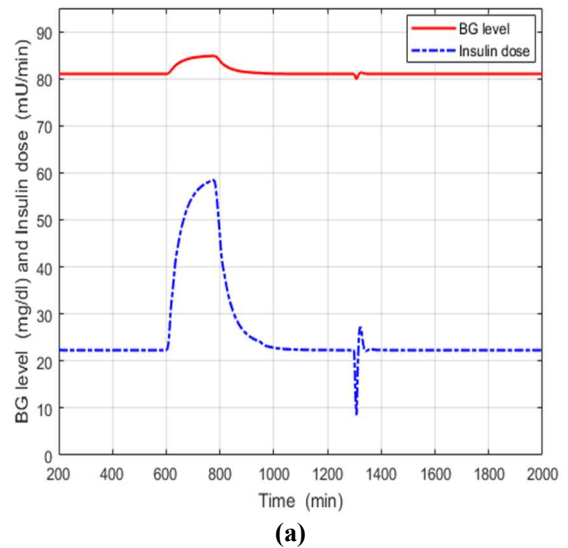
IV. RESULTS AND DISCUSSION

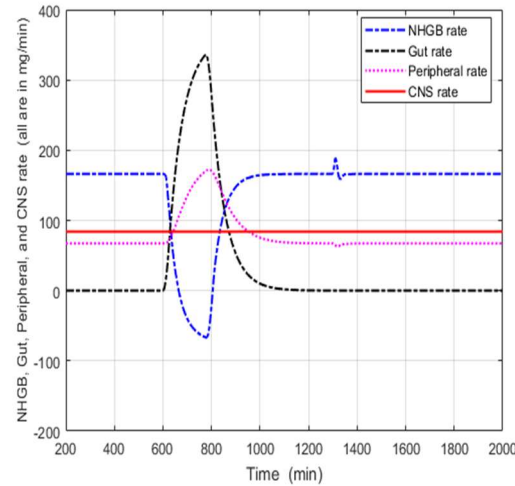
The effectiveness of the patient model using AO-TADF, in terms of accuracy, robustness, and stability, is thoroughly discussed in Sections A, B, and C, respectively. A comprehensive comparative analysis is then presented in Section D.

A. Dynamics of the Patient under AO-TADF Control

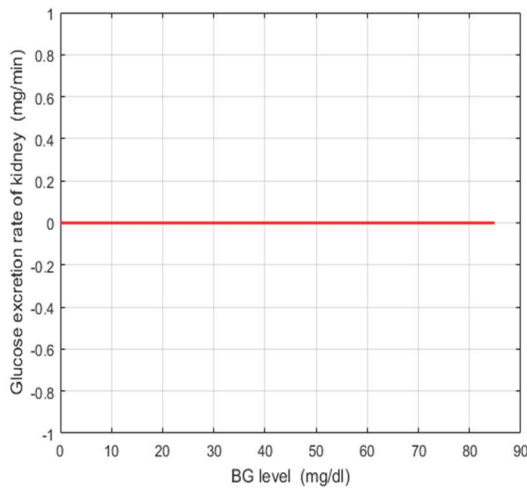
The AO-TADF-based controller integrated into the patient model evaluates all relevant factors that introduce disturbances and uncertainties, such as irregular physical activity, random glucose intake, and sensor-actuator noise. In this analysis, a 60 g meal is administered at 600 minutes, followed by exercise at 1300 minutes, to derive the characteristic relationship between insulin delivery and BG levels, as illustrated in Fig. 8(a). Additionally, other physiological profiles including CNS, NHGB, peripheral uptake, gut absorption, and glucose excretion rates are analyzed under identical conditions and presented in Fig. 8(b).

The influence of the controller on the patient model is further demonstrated in Fig. 8(a-c). Results indicate that insulin-sensitive organs, particularly the liver and peripheral tissues, exhibit enhanced glucose utilization compared to the uncontrolled case, as also observed in Fig. 6(a-c). This improved regulation effectively reduces the BG level to approximately 83.3 mg/dl, thereby preventing hyperglycemia. As the BG level falls below the RTG, no additional glucose excretion by the kidneys is observed, consistent with Fig. 8(c). Overall, the implementation of the AO-TADF-based artificial pancreas leads to improved performance across all patient physiological profiles.





(b)

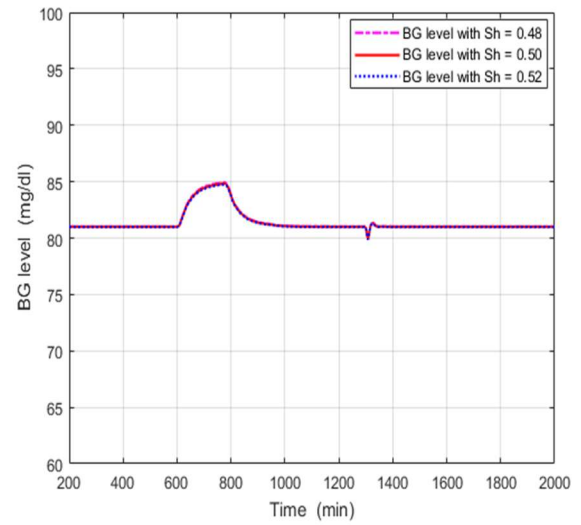


(c)

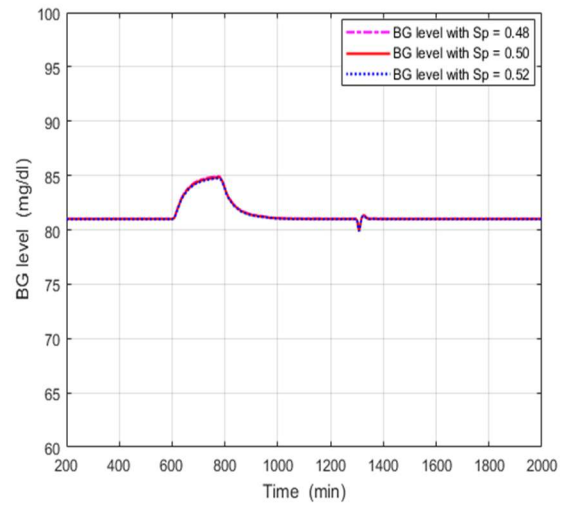
Figure 8 Characteristic responses of the closed-loop patient model: (a) relationship between insulin infusion and BG level; (b) glucose production and utilization rates across GI organs; (c) variation of BG level with renal glucose excretion rate.

B. Robustness Features of the AO-TADF Controller

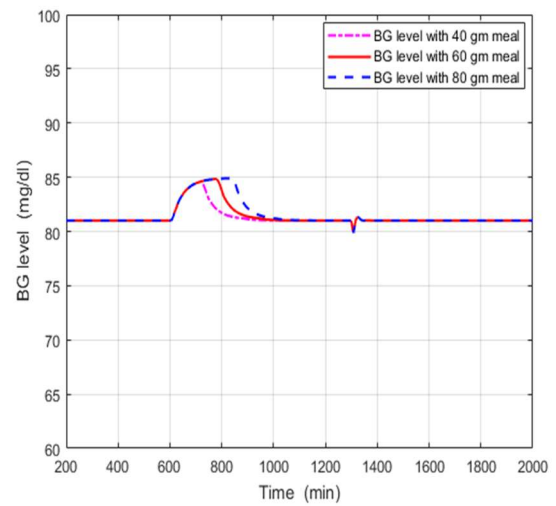
The proposed controller demonstrates strong resilience against diverse meal disturbances and two different types of parameter uncertainties within the model. Fig. 9(a) and 9(b) illustrate the temporal variation of plasma glucose levels with respect to hepatic and peripheral insulin sensitivities, respectively. In addition, Fig. 9(c) presents the changes in BG levels of the T1DM patient model under varying meal disturbance conditions. The simulation outcomes reveal that, although the closed-loop system exhibits minor variations in transient responses, the BG level consistently stabilizes around 81 mg/dl, corresponding to normo-glycemia, with a relatively shorter settling time. This consistent performance under uncertain and disturbed conditions indicates that the AO-TADF controller is highly robust and effectively maintains system stability.



(a)



(b)



(c)

Figure 9 Closed-loop patient responses illustrating the relationship between physiological parameters and meal intake: (a) variation of BG level with hepatic insulin

sensitivity (S_h); (b) variation of BG level with peripheral insulin sensitivity (S_p); (c) correlation between BG level and meal consumption.

C. Patient Model Stability

The Bode responses of both the open-loop and closed-loop systems are presented in Fig. 10(a, b) to verify that the stability criteria are satisfied. As can be seen from the results, the closed model [Fig. 10(b)] possesses more perfection and continuous state stability than the open loop model [Fig. 10(a)]. When comparing the bandwidth (BW) of the opened and closed patient models, the closed model has larger bandwidth. The model, when used in conjunction with the suggested controller, produces glucose level of 81 mg/dl and clearly shows quicker and more stable dynamics. As a result, it validates that the controller and the patient's anatomy work together to provide more stability.

D. Analysis with Comparison

The comparative results presented in the table 2 highlight the performance of different control strategies, namely PID, Fuzzy, Sliding Mode (SM), LQG, H_∞ , and the proposed AO-TADF controller, under identical disturbance conditions of 60 gm carbohydrate intake. All controllers are evaluated using consistent criteria, including insulin dosage, settling

time, peak deviation, steady-state error, and statistical measures such as mean and standard deviation of BG levels.

Among the conventional approaches, PID and Fuzzy controllers exhibit relatively higher insulin requirements and longer settling times, indicating slower system response and reduced efficiency in maintaining *normo-glycemia*. The SM and LQG controllers show improved dynamic behaviour; however, LQG demonstrates a comparatively larger peak deviation, suggesting less robustness to disturbances. The H_∞ controller achieves better stability and reduced variability than earlier methods, with moderate improvements in steady-state error and response time.

In contrast, the proposed AO-TADF controller outperforms all existing techniques across nearly all performance indices. It requires the lowest insulin infusion rate, achieves the fastest settling time, and minimizes both peak deviation and steady-state error. Additionally, it demonstrates superior robustness against noise and disturbances, as evidenced by the lowest mean BG level and smallest standard deviation. These results confirm that the AO-TADF controller provides enhanced stability, precision, and reliability in regulating blood glucose levels within the desired *normo-glycemic* range.

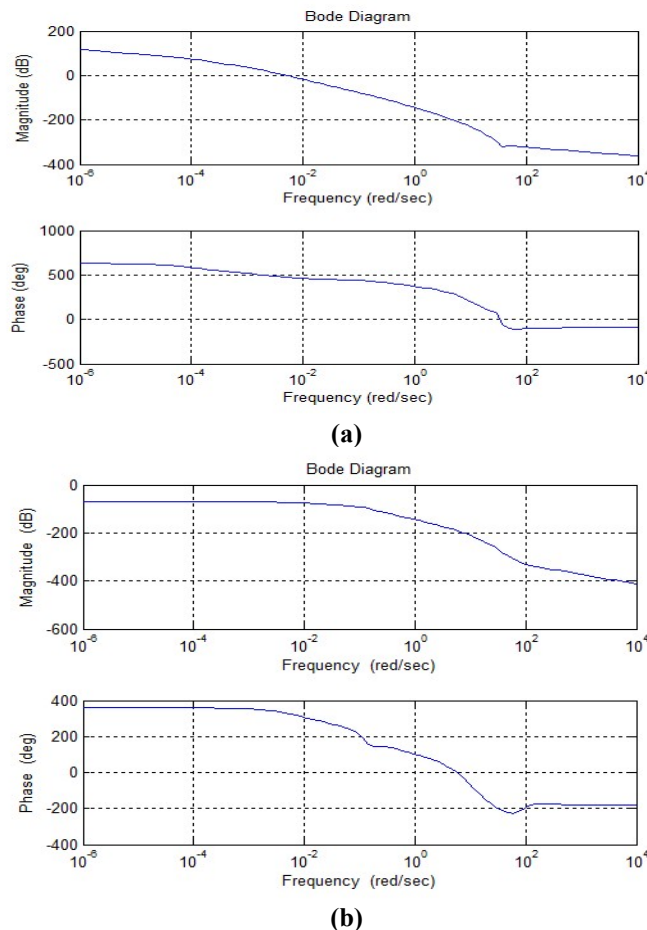


Figure 10 (a) Bode plots representing the patient model dynamics; (b) Bode responses of the patient model under AO-TADF control.

Table 2 A comparative assessment employing various advanced control strategies.

Control Techniques	PID (Sutradhar et al., 2002)	Fuzzy (Panigrahi et al., 2020)	SM (Rout, et al., 2018)	LQG (Patra et al., 2020)	H_∞ (Chee et al., 2005)	AO-TADF (Suggested)
Carbohydrate intake disturbance (gm).	60	60	60	60	60	60
Insulin infusion rate (mU/min).	58.9	58.8	58.7	58.6	58.5	58.2
t_s (min)	285	285	280	270	265	230
O_{Peak} (mg/dl)	5.2	5.3	4.1	8.5	4.5	3.9
U_{Peak} (mg/dl)	3.1	2.1	1.5	1.8	1.2	1.0
Noise (%)	10	10	5	5	5	1
e_{ss} (%)	0	0	0	0	0	0
Mean of BG level (mg/dl)	88	87	86	85	84	82
Standard Deviation	2.5	2.3	2.1	1.8	1.7	1.5

V. CONCLUSION

The proposed AO-TADF control strategy developed in this study effectively assists patients with T1DM in maintaining BG levels within the normo-glycemic range. A detailed mathematical formulation, along with a comprehensive SIMULINK-based model, has been designed for a patient model incorporating meal intake disturbances, enabling systematic testing and performance evaluation. The simulations were conducted under various uncertainties and disturbances arising from dietary intake and physical activity, all referenced to a standard physiological operating condition. The study considers a nominal insulin infusion rate of 22.3mU/min, with the operating point derived from a typical human physiological profile corresponding to a plasma glucose concentration of 81 mg/dl. Under these baseline conditions, the system's response was analysed against multiple perturbations associated with meal consumption and insulin dynamics. The results demonstrate that the proposed AO-TADF controller outperforms conventional control techniques by offering improved precision, enhanced stability, and stronger robustness in regulating BG levels. Furthermore, the findings indicate the feasibility of implementing this control approach in a real-time environment using an integrated embedded system, potentially combined with auxiliary devices such as continuous glucose sensors and infusion pumps for effective BG management in T1DM patients. However, it is important to note that the controller's performance is highly dependent on the appropriate tuning of its parameters. The gain selection process plays a critical role, and at certain operating conditions, the system may exhibit sensitivity, making it potentially vulnerable if not properly optimized. Future work

will focus on validating the proposed AO-TADF controller using clinically accepted platforms such as the UVA/Padova simulator or real patient data. Additionally, comprehensive benchmarking with other metaheuristic algorithms and Monte Carlo-based robustness analysis will be conducted. Further improvements will also target enhanced controller adaptability and real-time implementation for practical artificial pancreas systems.

AUTHOR CONTRIBUTIONS

A. K. Patra: Conceptualization, Investigation, Writing – review & editing, Writing – original draft, Resources, Data curation, Formal analysis. **S. R. Dutta:** Visualization, Validation, Software, Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Resources. **A. Nanda:** Validation, Software, Conceptualization, Supervision, Resources. **R. Agrawal:** Writing – review & editing, Supervision, Resources. **D. K. Subudhi:** Data curation, Formal analysis, Resources. **L. M. Satapathy:** Visualization, Validation, Software, Investigation, Resources.

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APPENDIX A

Nomenclature	Description	Nomenclature	Description
$I_p(t)$	Concentration of insulin present in the plasma.	$G(t)$	BG level
$I_{abs}(t)$	Rate of insulin uptake into the bloodstream	Ch	Carbohydrate
V_I	Insulin distribution volume expressed in deciliters per kilogram	Ch_{crit}	Critical value of carbohydrate
$I_a(t)$	Concentration of active insulin	$G_{gut}(t)$	Intestinal (gut) glucose absorption
K_1	Rate of delay in insulin action for I_p	$G_{in}(t)$	Rate of glucose entry into the VB from the gut (mg/min)
K_2	Rate at which insulin action is delayed for I_a	K_{gabs}	Rate constant for glucose absorption from the gut
$I_{ea}(t)$	Effective insulin activity	$Tasc_{ge}$	Rising time from 0 to $V \max_{ge}$
$I_{ep}(t)$	Plasma insulin effectiveness	$Tdes_{ge}$	Falling time from $V \max_{ge}$ to 0
S_p	Peripheral insulin sensitivity coefficient	I_{basal}	Basal value of plasma insulin
S_h	Hepatic insulin sensitivity coefficient	$G_{ren}(t)$	Glucose excretion through the kidneys
$G_{out}(t)$	Overall glucose utilization rate across all GM organs	K_t	Tilt gain
$G_{liver}(t)$	Net hepatic glucose balance rate for the liver	K_i	Integral gain
V_G	Glucose distribution volume within the VB	K_d	Derivative gain
n	Tilt coefficient	N_1, N_2, N_3	Pre-filter gains

APPENDIX B

Abbreviation	Description	Abbreviation	Description
WHO	World Health Organization	VB	Venous blood
MID	Micro Insulin Dispenser	RTG	Renal Threshold Glucose
LQG	Linear Quadratic Gaussian	GI	Glucose-Insulin
SM	Sliding Mode	GES	Gastric Emptying Subsystem
GA-PIC	Genetic Algorithm-Proportional Integral Controller	CNS	Central Nerves System
MP	Model Predictive	RBC	Red Blood Cell
MPC/LP	Model Predictive Controller with Laguerre Function	NHGB	Net Hepatic Glucose Balance
TAD	Tilt Acceleration Derivative	PID	Proportional Integral Derivative
ITAE	Integral Time Absolute Error	CCR	Creatinine Clearance Rate

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