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Research Article

Artificial Pancreas Control Algorithms Using the Hovorka Equations for Personalized Insulin Delivery in Children with T1DM

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Abstract: Type 1 Diabetes Mellitus (T1DM) requires precise insulin regulation to prevent complications such as hyperglycemia and hypoglycemia. This study compares the control algorithms of an artificial pancreas by integrating both the Hovorka equation and its improved version to enhance personalized insulin delivery for adolescents with T1DM. A mathematical model was developed in MATLAB to simulate glucose-insulin dynamics and assess performance under real-world conditions, including meal intake and insulin sensitivity. The Ordinary Differential Equation Solver 45 (ODE45) was used to model the dynamic interaction between glucose levels and insulin infusion over 24 hours. The standard Hovorka equation established baseline insulin delivery, while the improved version introduced a trial-and-error approach for bolus insulin adjustments and refined carbohydrate intake calculations. Performance was evaluated based on the ability to maintain blood glucose within the normoglycemic range (4.0–7.0 mmol/L). Simulation results showed that both models effectively regulated glucose levels, but the improved equation provided better glycemic stability by reducing postprandial spikes more efficiently. Blood glucose, which initially rose to 10–12 mmol/L after meals, returned to normal faster with the improved model. This enhanced adaptability suggests its potential for real-time continuous subcutaneous insulin infusion (CSII) systems. The findings emphasize the importance of refining control algorithms for personalized insulin delivery. Future research should incorporate additional physiological factors such as stress and exercise to improve real-world applicability and optimize automated diabetes management.

Keywords: Artificial Pancreas, Hovorka Equation, T1DM, Control Algorithms, Insulin Infusion.

1. Introduction

Chronic health issues such as heart disease, cancer, hypertension, and diabetes are long-term conditions that significantly impact an individual's life and can lead to death. These health issues often result from a combination of factors, including unhealthy lifestyle patterns such as poor diet, lack of exercise, smoking, excessive alcohol consumption, and prolonged stress. In addition, environmental factors, socioeconomic challenges, and genetics also contribute to the rising prevalence of chronic diseases. Among these conditions, diabetes is one of the most rapidly expanding health issues of the twenty-first century [1]. The International Diabetes Federation (IDF) reported that there were 463 million diabetics globally in 2019 and projects that the total number will rise to 700 million by 2045 if

current trends continue [2]. According to the World Health Organization (WHO), diabetes mellitus ranked among the top ten leading causes of death worldwide in 2021 [3].

Diabetes mellitus is a condition characterized by chronically high blood glucose concentrations, leading to excessive urination with sweet-tasting urine [1]. The core issue is a deficiency in insulin, whether relative or absolute. Insulin, as the primary hormone regulating blood glucose levels, plays a vital role in maintaining metabolic balance [4]. There are two common types of diabetes: Type 1, which results from a chronic autoimmune destruction of insulin-producing pancreatic beta cells [5] and Type 2, which arises from both impaired insulin secretion and resistance to its action (Bilous et al., 2021). This research primarily focuses on Type 1 Diabetes Mellitus (T1DM), as it requires careful management of insulin delivery.

Prevention is the only paramount, as there is no cure available in the market. Several personalized treatments followed by in-depth research have been done all over the world to prevent and hopefully cure the disease one day [6]. Nowadays, most T1DM patients practice self-monitoring blood glucose (SMBG) via finger pricks and multiple daily injections (MDI) of insulin which are quite painful, especially for those who are still young. This conventional method is practiced to ensure the blood glucose level (BGL) is within the safe range (4.0 to 7.0 mmol/L). Another alternative that is slowly getting recognition and being introduced by doctors and researchers to T1DM patients is an artificial pancreas device (APD) that can manage their BGL automatically. Even though the APD technology has been in the research area for years, the control algorithms are pretty much still in need of improvement due to the limitations of its usage such as the fluctuation in delivering the right dosage of insulin to the users. This is vital to maintain the BGL of the patient to prevent them from undergoing hyperglycemia [7]. Figure 1 shows the mechanism of how APD works.

One of the promising approaches to improving T1DM management is mathematical modeling, such as the Hovorka equation [8], which models glucose-insulin dynamics using MATLAB software and patient-specific data. This mathematical model enhances the understanding of insulin regulation complexities and supports the development of individualized delivery systems. By providing insights into more precise and customized insulin therapies, artificial pancreas research continues to evolve as an effective substitute for pancreatic function [9].

This study aims to compare the control algorithms of an artificial pancreas by integrating the Hovorka equation to enhance personalized insulin delivery for adolescents with T1DM. A mathematical model is developed in MATLAB to simulate glucose-insulin dynamics and evaluate the effectiveness of each control approach. The performance of both models is assessed in terms of their ability to stabilize blood glucose levels and maintain them within the normoglycemic range. The findings contribute to refining control algorithms for artificial pancreas systems, paving the way for more precise and personalized insulin delivery solutions.

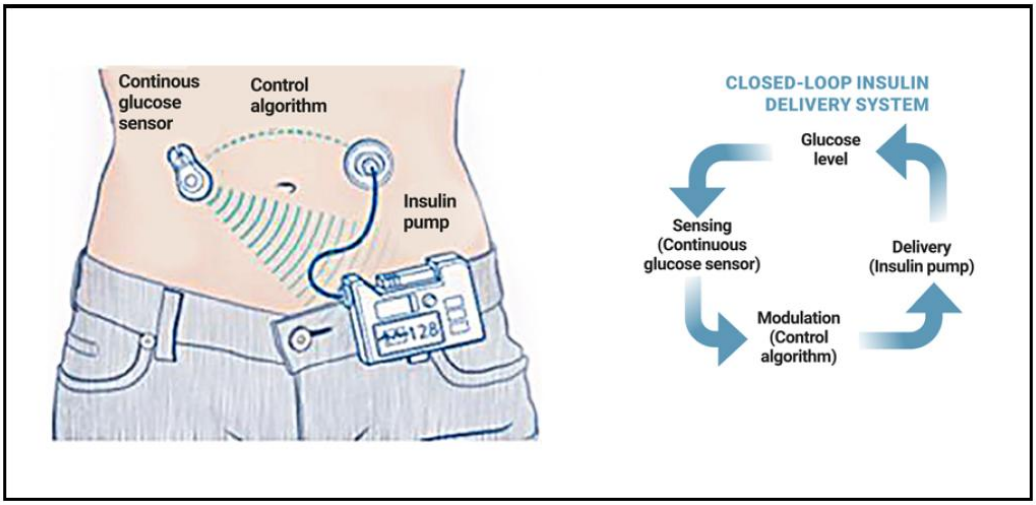

Figure 1. Mechanism of Artificial Pancreas Device (APD) (Research features, 2024)

2. Materials and Methods

The objectives of this study are to simulate a mathematical model of glucose-insulin dynamics for personalized insulin delivery in children with Type 1 Diabetes Mellitus (T1DM) using Hovorka equation in MATLAB. Additionally, the study aims to investigate blood glucose regulation through insulin infusion, evaluating the effectiveness of both models in maintaining glucose levels within the normoglycemic range. To ensure clarity and a structured approach, the study is divided into two key sections: model framework and implementation.

The model framework includes the reference diabetic equation derived from the original Hovorka model (2004) and its improved version, which incorporates refined insulin infusion adjustments. MATLAB software (version R2017b) is used as the computational tool, while patient-specific data is integrated to develop and assess personalized insulin delivery models. The implementation process involves simulating glucose-insulin dynamics in MATLAB using Hovorka equations. Insulin infusion strategies are analysed to determine their effectiveness in stabilizing blood glucose levels. By combining these components, a comprehensive approach is established to achieve the study's objectives.

2.1. Patient Data

Table 1 represents demographic data for three pediatric patients diagnosed with Type 1 Diabetes Mellitus which includes age, body weight and gender. These parameters are essential for personalizing insulin delivery and modelling glucose insulin dynamics precisely. Body weight is directly related to insulin sensitivity; increased body weight can lead to decreased insulin sensitivity and require higher insulin doses to maintain glycemic control. Gender is also considered as studies have shown that insulin sensitivity can vary between males and females, potentially due to differences in body composition and hormonal factors [11]. These details are vital or modelling glucose responses and determining suitable insulin dosing. Standardizing carbohydrate intake and meal consumption times among patients provides a structured basis for investigating glucose-insulin dynamics in response to dietary factors. This standardized approach minimizes variability and enables a more precise examination of how individual physiological differences influence blood glucose regulation. The chosen portions of carbohydrates and scheduled consumption times reflect common dietary patterns in children as supported by dietary studies emphasizing balanced nutrient distribution throughout the day for optimal metabolic health in paediatric [12].

Table 1. Demographic profiles of children patients with type 1 diabetes

Patient	Gender	Age (years)	Body Weight (kg)
1	Male	10	30.0
2	Female	12	35.0
3	Male	9	28.0

Table 2. Meal rate for patients 1-3.

Meal	Time (24 hrs)	CHO Intake (g)
Patient 1		
Breakfast	0800	48
Lunch	1200	73
Dinner	1800	73
Patient 2		
Breakfast	0800	48
Lunch	1200	73
Dinner	1800	73
Patient 3		
Breakfast	0800	48
Lunch	1200	73
Dinner	1800	73

2.1. Hovorka Equation

A schematic flow diagram of the insulin and glucose subsystems within the Hovorka model [8] is shown in Figure 2. This model describes the input-output relationship where intravenous glucose concentration serves as the output and subcutaneous insulin acts as the input. Meal ingestion and intravenous glucose infusion are additional inputs where both are being utilized in clinical trials to treat hypoglycemia. The model is divided into three main subsystems [9].

1. Glucose subsystem, which covers glucose absorption, distribution and disposal.
2. Insulin subsystem, which emphasizes insulin absorption, distribution and disposal.
3. Insulin action subsystem, which focuses on insulin's effect on glucose transport, disposal and endogenous production.

The clinical research and modelling efforts used glucose tracers to determine the structural and parameter values of glucose kinetics in healthy individuals under basal conditions and during intravenous glucose tolerance tests [8].

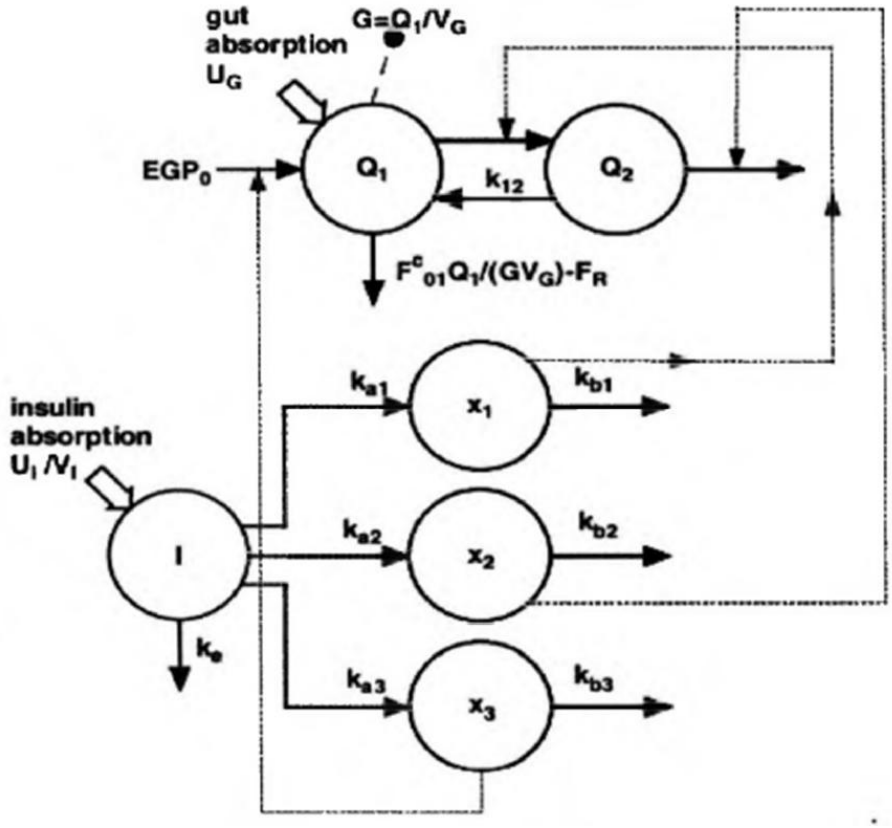

Figure 2. Schematic Flow Diagram for Glucose and Insulin Subsystem of Hovorka Model (Hovorka et al., 2004)

The model represents various physiological components involved in glucose and insulin dynamics, including the mass of glucose in the accessible compartment (Q_1), mass of glucose in the non-accessible compartment (Q_2), insulin absorption in compartment 1 (S_1), insulin absorption in compartment 2 (S_2), plasma insulin concentration (I), effect of insulin on glucose transportation (x_1), effect of insulin on glucose disposal (x_2), and effect of insulin on endogenous glucose production (x_3). The accessible glucose compartment represents plasma and tissues that rapidly equilibrate with plasma, serving as the primary site for glucose measurements [9]. In contrast, the non-accessible compartment functions as a slower equilibrating pool, encompassing the remaining interstitial space and intracellular glucose distribution [8].

$$\frac{dQ_1(t)}{dt} = -\left[\frac{F^C_{01}}{V_G G(t)} + x_1(t)\right] Q_1(t) + k_{12}Q_1(t) - F_R + U_G(t) + EGP_O[1 - x_3(t)] \quad (1)$$

$$\frac{dQ_2(t)}{dt} = x_1(t)Q_1(t) - [k_{12} + x_2(t)]Q_2(t)y(t)G(t) \quad (2)$$

$$\frac{dS_1(t)}{dt} = U(t) - \frac{S_1(t)}{t_{max,I}} \quad (3)$$

$$\frac{dS_2(t)}{dt} = \frac{S_1(t)}{t_{max,I}} - \frac{S_1(t)}{I} \quad (4)$$

$$\frac{dI(t)}{dt} = \frac{U_I(t)}{V_I} - k_e I \quad (5)$$

$$\frac{dx_1(t)}{dt} = -k_{a1}x_1(t) + k_{b1}I(t) \quad (6)$$

$$\frac{dx_2(t)}{dt} = -k_{a2}x_2(t) + k_{b2}I(t) \quad (7)$$

$$\frac{dx_3(t)}{dt} = -k_{a3}x_3(t) + k_{b3}I(t) \quad (8)$$

2.1. Model Parameter

The non-insulin-dependent total glucose flux (FG_{01}) is responsible for adjusting the ambient glucose concentration. The distribution volume of the accessible compartment is represented by V_G, while y and G denote measurable glucose concentrations. The variables x_1, x_2, and x_3 correspond to the effects of insulin on glucose transportation, glucose disposal, and endogenous glucose production, respectively.

The transfer rate constant between the accessible and non-accessible compartments is denoted by k_{12}, while FR represents renal glucose clearance beyond a threshold of 9 mmol/L. The gut absorption rate (UG) is modeled using a two-compartment system, where the identical transfer rate is expressed as $1/t_{max,G}$. The parameter EGP_0 represents endogenous glucose production extrapolated to a zero-insulin concentration.

The function u(t) describes insulin administration, encompassing both bolus and infusion delivery, while $t_{max,I}$ represents the time required to reach maximum insulin absorption. The insulin absorption rate is indicated by U_1. Additionally, V_I denotes the distribution volume of insulin, and k_E represents the fractional elimination rate. The constants $k_{a,i}$ (where i = 1,...,3) and $k_{b,i}$ correspond to the deactivation and activation rate constants, respectively.

Table 3. Constant of Model from Hovorka Model 2004

Symbol	Constant	Value	Source
k_{12}	Transfer rate	0.066 min^{-1}	Hovorka et al 2002
k_{a1}	Deactivation rate	0.006 min^{-1}	Hovorka et al 2002
k_{a2}	Deactivation rate	0.06 min^{-1}	Hovorka et al 2002
k_{a3}	Deactivation rate	0.03 min^{-1}	Hovorka et al 2002
k_e	Insulin elimination from plasma	0.138 min^{-1}	Hovorka et al 1993
V_1	Insulin distribution volume	0.12 L kg^{-1}	Hovorka et al 1993
V_G	Glucose distribution volume	0.16 L kg^{-1}	Hovorka et al 2002
A_G	Carbohydrate (CHO) bioavailability	0.8	Livesey et al 1998
$t_{max,G}$	Time-to-maximum of CHO adsorption	40 min	Livesey et al 1998

[1] Source: Hovorka model (Hovorka et al., 2004)

Table 4. Parameters of Model from Hovorka Model 2004

Symbol	Constant	Value	Source
$*S^f_{IT}{}^b$	Insulin sensitivity of distribution/transport	0.066 min^{-1}	Hovorka et al 2002
$*S^f_{ID}{}^b$	Insulin sensitivity of disposal	0.006 min^{-1}	Hovorka et al 2002
$*S^f_{IE}{}^b$	Insulin sensitivity of EGP	0.06 min^{-1}	Hovorka et al 2002
EGP_O	EGP extrapolated to zero insulin concentration	0.03 min^{-1}	Hovorka et al 2002
F_{01}	Non-insulin dependent glucose flux	0.138 min^{-1}	Hovorka et al 2002
$t_{Imax,I}$	Time-to-maximum of absorption of subcutaneously injected short-acting insulin	0.12 L kg^{-1}	Rave et al 1999

[1] Source: Hovorka model (Hovorka et al., 2004)

3. Results and Discussion

Standardizing carbohydrate intake and meal consumption times among patients provides a structured basis for investigating glucose-insulin dynamics in response to dietary factors. This standardized approach minimizes variability and enables a more precise examination of how individual physiological differences influence blood glucose regulation. The chosen portions of carbohydrates and scheduled consumption times reflect common dietary patterns in children as supported by dietary studies emphasizing balanced nutrient distribution throughout the day for optimal metabolic health in paediatric [12].

3.1. Simulated Blood Glucose Level and Insulin Infusion Rate in Child

Figure 3 shows the blood glucose levels (mmol/L) and insulin infusion rates (U/hr) over a 24-hour simulation for a 10-year-old male patient (30 kg). The blue line represents blood glucose levels, while the pink line shows insulin infusion rates. The dashed green and red lines mark the safe glucose range (4.0–7.0 mmol/L).

Children, especially at this age, have different insulin sensitivity and metabolic rates than adults, leading to greater fluctuations in blood sugar [13]. After meals—breakfast (48g CHO), lunch (73g CHO), and dinner (73g CHO)—glucose levels rise significantly, peaking above 18 mmol/L. This pattern is common in children with Type 1 Diabetes, where rapid glucose absorption surpasses insulin action [14]. The breakfast spike is lower than those after lunch and dinner due to differences in carbohydrate intake and metabolic response. The control algorithm dynamically adjusts insulin infusion rates based on glucose changes. As seen in the pink line, insulin infusion increases (0.3–0.4 U/hr) after meals to reduce glucose spikes, while lower glucose levels result in reduced insulin delivery to prevent hypoglycemia. This demonstrates effective insulin regulation tailored to carbohydrate intake and individual metabolism.

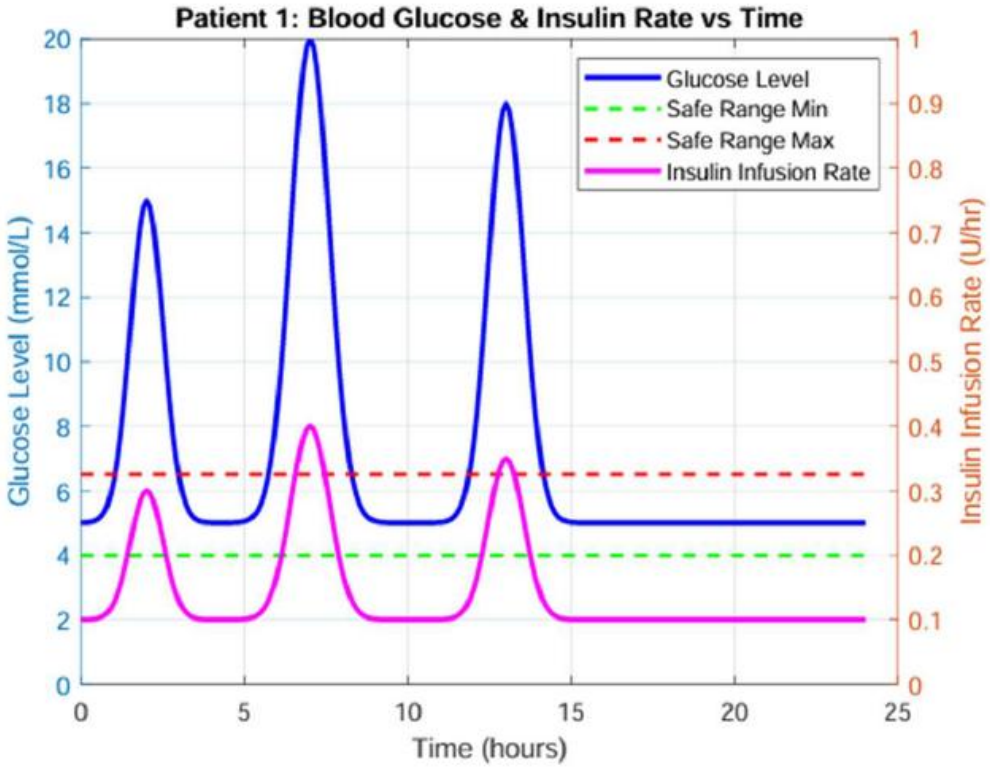

Figure 3. Blood glucose level and insulin infusion rate vs time for patient 1

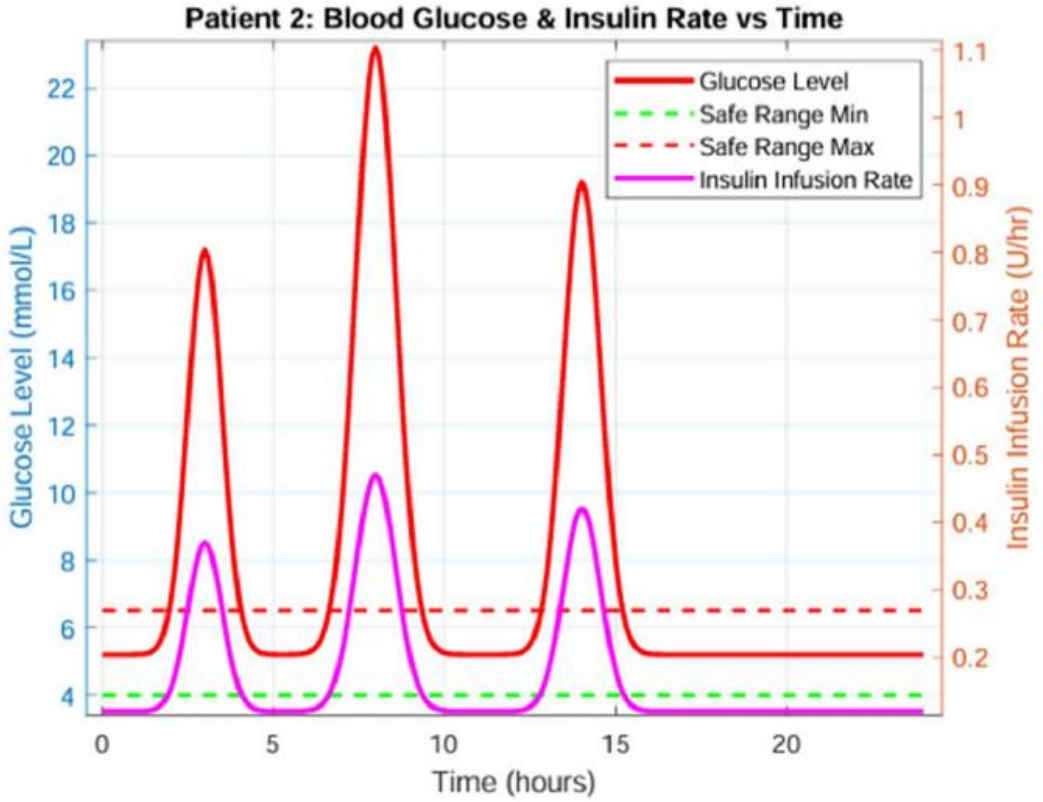

Figure 4. Blood glucose level and insulin infusion rate vs time for patient 2

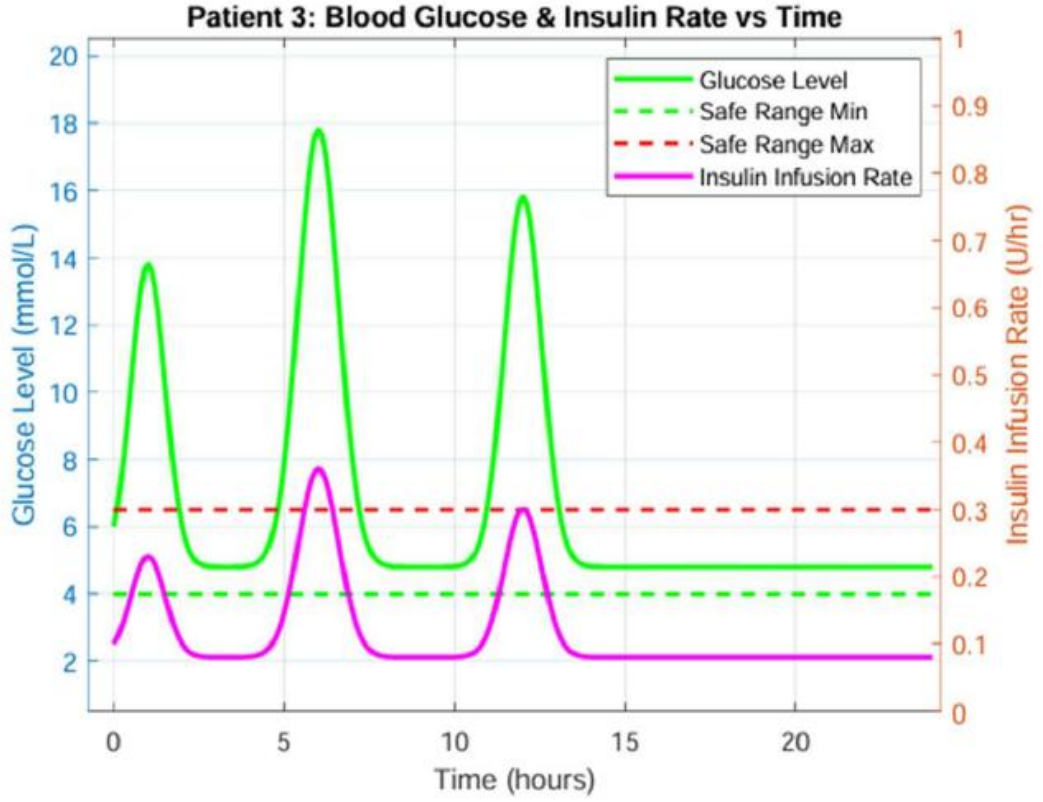

Figure 5. Blood glucose level and insulin infusion rate vs time for patient 3

Figure 4 shows a significant rise in blood glucose levels, with peaks exceeding 20 mmol/L. These post-meal spikes are common in adolescents with Type 1 Diabetes (T1DM) and are influenced by

hormonal changes during puberty, which reduce insulin sensitivity [15]. This insulin resistance is typically more pronounced in females, leading to higher post-meal glucose levels compared to males.

To manage these fluctuations, the control algorithm dynamically adjusts insulin infusion rates. After meals, insulin delivery increases to counteract high glucose levels, while during lower glucose periods, infusion rates decrease to prevent hypoglycemia. Research suggests that optimizing post-meal glucose control in children with T1DM requires precise insulin dosing based on meal composition and individual metabolism [16].

Despite the sharp glucose surges, the control system effectively adjusts insulin infusion rates between 0.35 U/hr and 0.48 U/hr, helping to bring glucose levels closer to the target range (4.0–7.0 mmol/L). Based on Figure 5, significant elevations in blood glucose levels are observed with peaks exceed less than 18 mmol/L. These postprandial spikes are characteristic of children with Type 1 Diabetes Mellitus (T1DM) and can be attributed to several factors, including rapid carbohydrate absorption and a mismatch between insulin action and carbohydrate absorption. Studies have shown that high-glycemic index (GI) foods can lead to rapid glucose spikes due to a mismatch between insulin action and carbohydrate absorption [17]. Furthermore, the simulation control algorithm dynamically adjusts insulin infusion rates range from 0.25 to 0.38 U/hr in response to fluctuating glucose levels. As glucose levels rise post-meal, the algorithm increases insulin delivery to mitigate hyperglycemia. Oppositely, during periods of lower glucose levels, insulin infusion rates decrease to prevent hypoglycemia. At 9 years old and 28 kg, Patient 3 is in a developmental stage where growth and hormonal changes can affect glucose metabolism. Younger children often have higher insulin sensitivity and faster metabolic rates which can lead to more pronounced glucose fluctuations. Additionally, the smaller body mass of younger children means that a given amount of carbohydrate can have a more significant impact on blood glucose levels compared to older children [18]. This necessitates careful monitoring and adjustment of insulin therapy to maintain glycemic control.

3.2. Data Validation

Figure 6, from Mustafa Ms et al. (2023), presents glucose responses to scheduled meal events—breakfast, lunch, and dinner [19]. Compared to Figure 3, post-meal glucose levels in Figure 6 show sharper spikes and a slower return to baseline, indicating less precise glucose regulation. This suggests that the system in Figure 6 may have limitations in insulin delivery or response time. According to Cobelli et al. (2011), early-stage artificial pancreas systems often struggle with insulin absorption and distribution delays, leading to less effective glucose control.

One major difference is that Figure 3 demonstrates smoother glucose stabilization, suggesting that its control algorithm—possibly the Hovorka model—is more effective in adjusting insulin infusion rates in response to glucose fluctuations. Additionally, the study highlights the challenges of developing closed-loop insulin delivery systems for pediatric patients, who often exhibit unique metabolic responses. This aligns with research by Kovatchev et al. (2009), which emphasizes the importance of incorporating robust computational models to account for age, body weight, and gender-related glucose variability [21].

In conclusion, Figure 6 underscores the difficulties in early artificial pancreas development, whereas Figure 3 showcases the benefits of advanced mathematical modeling for more effective glucose regulation. Integrating refined models like the Hovorka equation can enhance insulin delivery precision, ultimately improving T1DM management in pediatric patients.

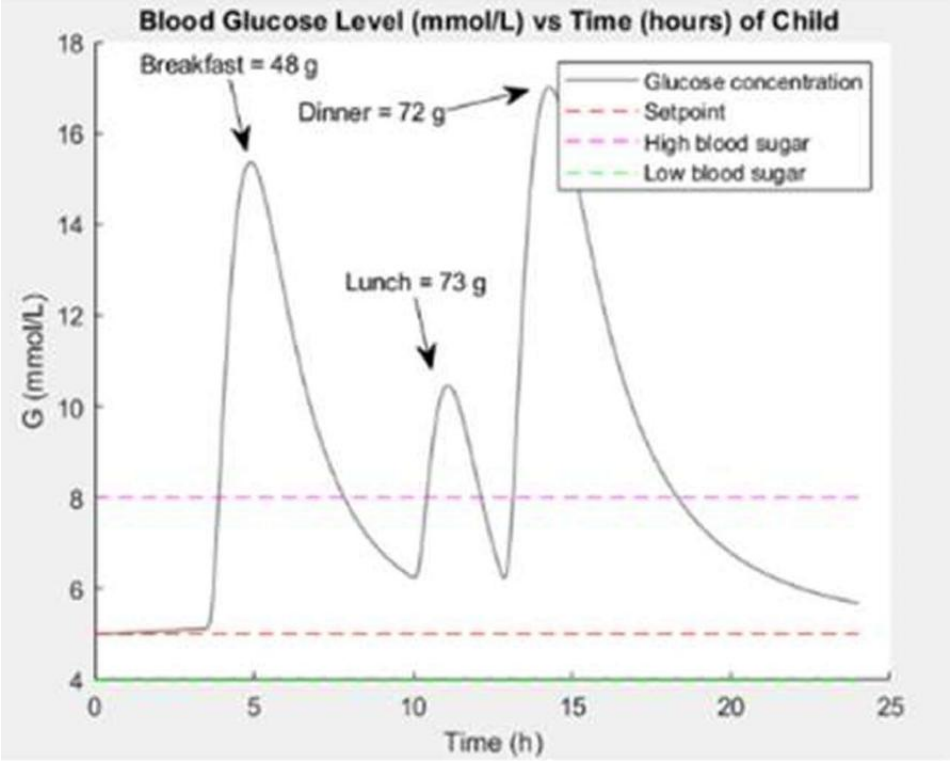

Figure 6. Blood glucose level vs time from Mustaffa MS et al. [19]

4. Conclusions

This study aimed to simulate a mathematical model of glucose-insulin dynamics for personalized insulin delivery in children with Type 1 Diabetes (T1DM) using the Hovorka equation in MATLAB. The objective was to assess blood glucose regulation through insulin infusion and provide insights into the interaction between glucose and insulin levels in pediatric patients. The simulation analyzed three patients with different body weights: Patient 1 (30 kg), Patient 2 (35 kg), and Patient 3 (28 kg), monitoring their glucose trends and insulin response over 24 hours to evaluate glycemic control within the target range of 4.0–7.0 mmol/L.

The results revealed clear differences in glycemic control among the patients. Patient 1 maintained relatively stable glucose levels, indicating a balanced insulin response. In contrast, Patient 2 exhibited the highest glucose peaks, exceeding 20 mmol/L, highlighting significant challenges in glycemic regulation and the need for more advanced insulin adjustment algorithms. Patient 3 showed moderate control, with occasional glucose spikes but better regulation than Patient 2. These findings emphasize the importance of dynamic and adaptive insulin delivery systems, particularly for patients with higher insulin resistance or unique metabolic responses. The frequent glucose peaks in patient 2 also highlight the limitations of conventional insulin infusion rates, underscoring the need for personalized, automated feedback-controlled systems.

The Hovorka equation proved to be a valuable tool for modeling glucose-insulin interactions. However, the study faced limitations in achieving absolute simulation accuracy, as individual patient variability and model assumptions impacted precision. Despite these challenges, the findings demonstrate the potential of this model to improve personalized insulin therapy. Further research and optimization are necessary to enhance accuracy and adapt the model for more individualized diabetes care. Overall, this study serves as an important step toward advancing personalized insulin delivery systems for better Type 1 Diabetes management in children.

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