Lifestyle Log Based Blood Glucose Level Prediction for Outpatient Care

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***Abstract*—Treatment of diabetes mellitus is a crucial problem in modern health care. Surveys show that the currently used methods to estimate the required amount of insulin are quite inefficient in practice as they are based on experience and conjecture. This paper offers a new method to predict the glucose level of people with diabetes. The proposed approach combines two efficient models found in literature. The mixture of the methods tracks the blood sugar level considering nutrition, applied insulin and initial glucose level. According to our tests, the model gives satisfactory results with real patients both in inpatient and outpatient care.**

Keywords—Glucose-level tracking; eHealth; Glucose-Insulin system; Glucose absorption; Diabetes mellitus

I. INTRODUCTION

Diabetes mellitus is a metabolic disease that affects the whole society. It is a typical disease of the modern culture caused by obesity, the lack of physical activity and the changing of culinary culture. At the moment, this problem hits 3% of the population [1], but this number is increasing. The current predictions report that the number of people with diabetes can reach 5% within 2 decades [1]. This underlines the importance of diabetic lifestyle support.

The official classification separates diabetes mellitus into different types, according to clinical age [2]:

- Type 1 diabetes results from the lack of insulin production. The failure of insulin output is caused by an autoimmune destruction of beta-cells in the pancreas, which usually leads to absolute insulin deficiency. Patients diagnosed with Type 1 diabetes have to follow a strict diet and apply subcutaneous insulin by injection or insulin pump.
- Type 2 is the major form of diabetes as it accounts for 90% to 95% of all diagnosed patients (in the USA [3]). It is an insulin resistant stage caused by failure in insulin secretion. The treatment of these patients varies from lifestyle changing through diet and oral medications to subcutaneous insulin necessity.
- The third category contains special types, including gestational diabetes and other types caused by medications, infections, or other illnesses.

The remainder of the paper is structured as follows: Section II presents some related works and summarizes the prospects of this field. Section III contains the description of the proposed glucose level prediction system. Section IV includes the result of several tests with the model. Section V contains the discussion of the results. Finally, Section VI concludes the paper and outlines future work.

II. MOTIVATIONS AND LITERATURE OVERVIEW

The basic motivation of our efforts is to provide diabetics with a tool that they can use in everyday life to predict their blood glucose level. We focus on outpatients treated with insulin injection no matter having type 1, type 2 or other types of diabetes. These patients inject themselves with insulin considering meal, physical activity, sports and also the weather change. The main index to verify the patients' state is HbA_{1c} (Glycated hemoglobin). According to the recent surveys, these values are far from ideal in the case of several patients [4]. The gap does not seem big, but it can lead to serious complications; moreover a big variation of glucose levels endangers the life of a person with diabetes.

The whole metabolism can be divided into two parts, as Figure 1 shows. The first one is glucose absorption from meals and the second one is the glucose controlling system including insulin evolution.

From the aspect of glucose uptake, the absorption from intestine is the main factor, but the stomach has a significant role in the procedure as well. In connection with glucose control, there are many factors to take into account such as glucose uptake, inner insulin production, insulin input, etc. These factors are discussed later in this paper.

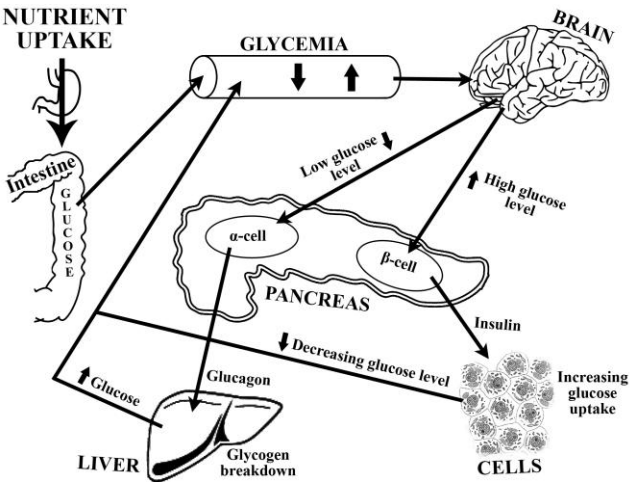

Figure 1. The process of metabolism

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TABLE I. THE PARAMETERS OF THE MODEL OF GLUCOSE ABSORPTION

Model of glucose absorption

Prefixes

Δ	The actual time interval ($\Delta t = t_{i+1} - t_i$).
m	The component is added by meal.
s	The component is in the stomach.
e	The component is ejected from the stomach into the intestine.
i	The component is in the intestine.
f	The component leaves the intestine as faeces.
a	The component is absorbed through the intestinal wall.

Variables

$Prot(t_i)$	The amount of proteins at time step t_i.
$Lip(t_i)$	The amount of lipids at time step t_i.
$Fibr(t_i)$	The amount of fibres at time step t_i.
$Monosac(t_i)$	The amount of monosaccharides at time step t_i.
$Starch_{GI}(t_i)$	The amount of starch with the given GI at time step t_i.
$Mass(t_i)$	The amount of nutriment at time step t_i.
$Water(t_i)$	The amount of water at time step t_i.
GER	Gastric emptying rate. [kJ/min]
$sVol$	Stomach volume. [ml]
τ_{wall}	Average time before the food obtains contact with the intestinal wall. [min]
τ_{GI}	The time of the starch breakdown process with glycemic index GI. [min]

Parameters

BM	The body mass in kg.
GI	The glycemic index of the food.

Constants

$CHOavail$	Bioavailability. The optimized value is 0.76.
$sVol_0$	Exponential constant for stomach emptying. The value is 225 ml.
SER	The specific emptying rate. The optimized value is 0.161.
IAR	Maximal intestinal absorption rate, the estimated value is 2.0 g/min.
$EnergyDens_j$	The energy density is 17kJ/g for proteins, starch and monosaccharide, 0kJ/g for fibres and 39 kJ/g for lipids.
τ_{fibr}	Exponential time constant for excretion set to 180 min.
$\tau_{wall\,0}$	Set to 1000 min.
τ_{100}	Time constant for starch breakdown with GI 100. The optimized value is 28.0 min.
α	Parameter relating τ_{GI} to the glycemic index. The optimized value is 0.0125.

TABLE II. THE PARAMETERS OF THE GLUCOSE CONTROL MODEL

Glucose control model

Variables

$G(t)$	Plasma glycemia. [mM = mmol/l]
$I(t)$	Insulinemia. [pM = pmol/l]
$S_1(t)$	The insulin mass in the accessible subcutaneous depot. [pmol/$kgBW$]
$S_2(t)$	The insulin mass in the non-accessible subcutaneous depot. [pmol/$kgBW$]
$f(G)$	Pancreas Insulin Delivery Rate.

Parameters

K_{xgi}	Rate of glucose uptake by insulin-dependent tissues per pM. [1/(min ∗ pM)]
T_{gh}	Net balance between hepatic glucose output and insulin-independent zero-order glucose uptake (by brain). [mmol/(min ∗ $kgBW$)]
V_G	Distribution volume for glucose. [L/$kgBW$]
K_{xi}	Apparent first-order disappearance rate for insulin. [1/min]
T_{iGmax}	The maximal rate of second-phase insulin release. [pmol/(min ∗ $kgBW$)]
V_i	Distribution volume for insulin. [L/$kgBW$]
τ_g	The delay with which the pancreas varies secondary insulin release in response to varying plasma glucose concentrations. [min]
$t_{max\,,I}$	Time-to-maximum insulin absorption. [min]
$u(t)$	Subcutaneous insulin delivery rate. [pM/min]
$kgBW$	The weight of the patient. [kg]
G^*	The glycemia at which the insulin release is half of its maximal rate. [mM]
γ	The progressivity with which the pancreas reacts to circulating glucose concentrations.

Our model uses a combination of two existing models for nutriment absorption and glucose control.

There are methods for measuring glucose absorption [5] from meals such as the Diabetes Advisory System - DIAS [6]. Lots of models build upon this system though its base is only a simple one-compartment model. In order to create a more precise algorithm, other methods use glycemic indices (GI) [7] allowing mixed meals input such as the two-compartment model from Arleth et al. [8]. These methods provide a simulation closer to reality.

Beside glucose absorption, the evolution of insulin is the other main factor in tracking glycemia. There are even more methods in this field [9] starting with the so called minimal model which is still used in practice since it is a relatively simple method based on ordinary differential equations [10]. Several methods are the approximations of this model, e.g., [11]. The minimal model has low number of parameters, hence a limited predictive power. This problem is solved in more sophisticated methods, using differential equations. These approaches might use integro-differential equations [12], partial differential equations [13] or delay differential equations [14]. Such solutions as the latter support

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subcutaneous insulin depot, create better representation of the Insulin Delivery Rate (IDR), etc.

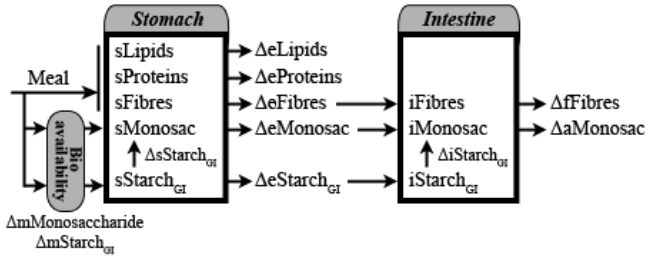

Figure 2. The process of absorption from mixed meals

III. THE PROPOSED METHOD

As mentioned before, our method combines two existing, state-of-the-art models to simulate plasma glycemia by influences of meals and insulin uptake. We chose these models because they have a realistic, comprehensive set of parameters capable of simulating a real-life outpatient as well.

A. Glucose Absorption From Meals

A two-compartment method [8, 15] is used to model the effect of nutrition on blood glucose level. The model proposed by Arleth T. et al. divides the digestion into two segments, as seen in Figure 2. The food first arrives to the stomach compartment followed by emptying into the small intestine and later into the large intestine. The absorption of the monosaccharide happens in the intestinal part; the remaining mass is ejected as faeces.

Simpler methods, like DIAS, operate with carbohydrate as input and take some components (e.g., lipids, proteins, starch) out of consideration. In contrast, our model takes protein, lipid, monosaccharide, fibre and starch as input, each one having its own effect during the absorption. In addition, the method can deal with mixed meals by using GI. Moreover, digestion overlap is handled properly as well.

The whole process is based on mass balance equations [15]. The equations for the stomach compartment are as follows:

$$sProt(t_{i+1}) = sProt(t_i) + \Delta mProt(t_i) - \Delta eProt(t_i) \quad (1)$$

$$sLip(t_{i+1}) = sLip(t_i) + \Delta mLip(t_i) - \Delta eLip(t_i) \quad (2)$$

$$sFibr(t_{i+1}) = sFibr(t_i) + \Delta mFibr(t_i) - \Delta eFibr(t_i) \quad (3)$$

$$\begin{aligned} sMonosac(t_{i+1}) = {} & sMonosac(t_i) + \Delta mMonosac(t_i) \\ & * CHOavail - \Delta eMonosac(t_i) \\ & + \sum_{GI} \Delta sStarch_{GI}(t_i) \end{aligned} \quad (4)$$

$$\begin{aligned} sStarch_{GI}(t_{i+1}) = {} & sStarch_{GI}(t_i) + \Delta mStarch_{GI}(t_i) \\ & * CHOavail - \Delta eStarch_{GI}(t_i) \\ & - \Delta sStarch_{GI}(t_i) \end{aligned} \quad (5)$$

The rate of ejection from stomach to intestine is measured by the gastric emptying rate (GER) [15]:

$$GER = SER * BM * \left(\frac{BM}{70}\right)^{0,425} * (1 - e^{sVol/sVol0}) \quad (6)$$

$$\begin{aligned} sVol = {} & \left(sProt + sLip + sFibr + \sum_{GI} sStarch_{GI}\right) * 3 \\ & + sMonosac * 18 \end{aligned} \quad (7)$$

The ejection from stomach to intestine is calculated considering the energy of the food components ($Energy_j$) using GER. The following equations [15] determine the actual ejections for each state variable ($StateVar_i$):

$$\Delta eStateVar_i = \Delta t * sStateVar_i * \frac{GER}{sTotalEnergy} \quad (8)$$

$$sTotalEnergy = \sum_j sEnergy_j \quad (9)$$

$$sEnergy_j = sStateVar_j * EnergyDens_j \quad (10)$$

The next compartment is the intestine, where proteins and lipids do not play a role anymore. The absorption of monosaccharides (15) happens here.

$$iFibr(t_{i+1}) = iFibr(t_i) + \Delta eFibr(t_i) - \Delta fFibr(t_i) \quad (11)$$

$$\Delta fFibr(t_i) = iFibr(t_i) * (1 - e^{-\Delta t/\tau_{fibr}}) \quad (12)$$

$$\begin{aligned} iMonosac(t_{i+1}) = {} & iMonosac(t_i) + \Delta eMonosac(t_i) \\ & - \Delta aMonosac(t_i) \\ & + \sum_{GI} \Delta iStarch_{GI}(t_i) \end{aligned} \quad (13)$$

$$\begin{aligned} iStarch_{GI}(t_{i+1}) = {} & iStarch_{GI}(t_i) + \Delta eStarch_{GI}(t_i) \\ & - \Delta iStarch_{GI}(t_i) \end{aligned} \quad (14)$$

$$\begin{aligned} \Delta aMonosac(t_i) = \min\Big\{ & iMonosac(t_i) \\ & * \left(1 - e^{-\frac{\Delta t}{\tau_{wall}}}\right), (IAR * \Delta t)\Big\} \end{aligned} \quad (15)$$

$$\tau_{wall} = \tau_{wall\,0} * iFibr/iMass \quad (16)$$

$$\begin{aligned} iMass(t_i) = {} & iMonosac(t_i) + \sum_{GI} \Delta iStarch_{GI}(t_i) \\ & + iWater(t_i) + iFibr \end{aligned} \quad (17)$$

$$iWater(t_i) = iMonosac(t_i) * 37 \quad (18)$$

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The following equations [15] calculate the breakdown of starch into monosaccharides:

$$\Delta sStarch_{GI}(t_i) = sStarch_{GI}(t_i) * (1 - e^{-\Delta t/\tau_{GI}}) \quad (19)$$

$$\Delta iStarch_{GI}(t_i) = iStarch_{GI}(t_i) * (1 - e^{-\Delta t/\tau_{GI}}) \quad (20)$$

$$\tau_{GI} = \tau_{100}[1 + \alpha * (100 - GI)] \quad (21)$$

The definition of the parameters is given in Table I. For further details of the model see [15].

B. Glucose Control System

A sophisticated glucose control system model was chosen using Delay Differential Equations (DDE), proposed by P. Palumbo et al. [14, 16]. This model has several parameters to support both type 1 and type 2 diabetics (see Table II). It is also possible to use insulin pump or subcutaneous insulin injections as input. The method uses two subcutaneous depots (accessible and not-accessible) to simulate subcutaneous insulin absorption. The main equations [16] are:

$$\frac{dG}{dt} = -K_{xgi}\,G(t)I(t) + \frac{T_{GH}}{V_G} \quad (22)$$

$$\frac{dI}{dt} = -K_{xi}\,I(t) + \frac{T_{iGmax}}{V_I} f\big(G(t - \tau_G)\big) + \frac{1}{V_I t_{max\,,I}} S_2(t) \quad (23)$$

$$\frac{dS_2}{dt} = \frac{1}{t_{max\,,I}} S_1(t) - \frac{1}{t_{max\,,I}} S_2(t) \quad (24)$$

$$\frac{dS_I}{dt} = -\frac{1}{t_{max\,,I}} S_1(t) - u(t) \quad (25)$$

The Insulin Delivery Rate (IDR) is modeled by the nonlinear $f(G)$ function [16]:

$$f(G) = -\frac{\left(\frac{G}{G^*}\right)^{\gamma}}{1 + \left(\frac{G}{G^*}\right)^{\gamma}} \quad (26)$$

IV. RESULTS

We implemented the combined model in a prototype and checked the correctness of our implementation by comparing its results to those published for a virtual patient in the original paper [16]. The parameters of the model were taken from the literature [17], from an intravenous glucose tolerance test experiment on an obese patient, slightly changed to simulate Type 2 diabetes mellitus (see Table III). The results showed good correlation to those published.

In the next step, we validated this model on outpatient data. Two persons with diabetes mellitus were examined. The first test involved a woman with Type 2 diabetes, while the second patient was a Type 1 diabetic man (see Table IV). Both patients are treated with subcutaneous insulin injection.

TABLE III. THE PRAMETERS OF THE VIRTUAL PATIENT WITH TYPE 2 DIABETES MELLITUS

Parameter	Value
K_{xgi}	3.11*10^{-5}
T_{gh}	0.003
V_G	0.187
K_{xi}	1.211*10^{-2}
V_i	0.25
T_{iGmax}	0.236
τ_g	24
$t_{max\,,I}$	55
G^*	9
γ	3.205

TABLE IV. THE PARAMETERS OF THE OUTPATIENTS WITH SUBCUTANEOUS INSULIN TREATMENT

Patient *A* with Type 2 diabetes mellitus		**Patient *B* with Type 1 diabetes mellitus**	
Birth date	1952	Birth date	1993
Gender	female	Gender	male
Height	156 cm	Height	196 cm
Weight	78 kg	Weight	83 kg
Applied insulin	Lispro	Applied insulin	Glulisine
Peak	60	Peak	55
Quantity/Unit	6000 pmol	Quantity/Unit	6000 pmol

The tests on both persons were executed with the same parameters as seen in Table III except T_{iGmax} , which is set to 0.1. The patients used similar types of insulin with the same quantity per unit indicator. On the whole, a reliable comparison can be made between the outcomes.

The first diabetic patient, outpatient A, was treated as inpatient to adjust her inordinate glycemia. Medication, glucose readings and meals were logged during 6 days including 15 meals and 45 glucose level measurements by ordinary blood sugar meter. The available meal log may contain inaccurate values if the patient consumed other meals except those offered as the controlled menu.

In the case of outpatient B, a controlled experiment was conducted during 3 days with 13 meals. The blood sugar level was monitored by a Medtronic Guardian Real-Time Continuous Glucose Monitoring (CGM) System, measuring the actual value every 5 minutes. The food portions were measured properly with scale and the time of meal and insulin input was logged correctly with minimum possibility of false values, using an android-based nutrition logger application [18].

Two different kinds of tests were made with each patient. The first simulation used meal wise records, i.e. the meals were treated as separate tests. Each test was run with zero startup blood insulin level and no running glucose absorption. The second test used a whole day's data with zero startup blood insulin level in the morning. During this test, the absorption of the insulin and the glucose from food could be in progress at the next meal as well.

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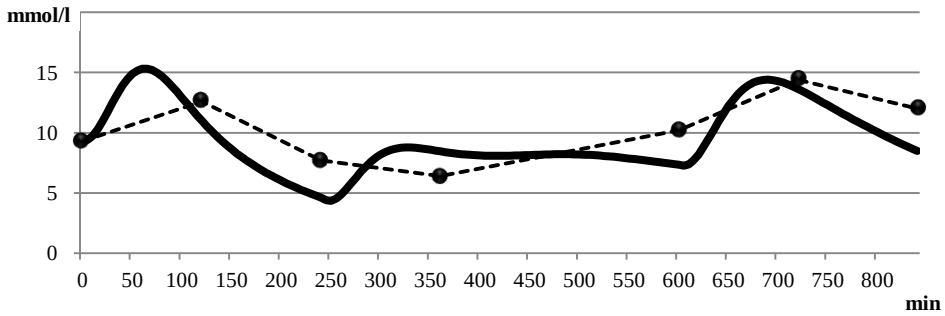

Figure 3. The fifth day of the meal wise test of patient A (solid line – model estimations, dashed line – measured values)

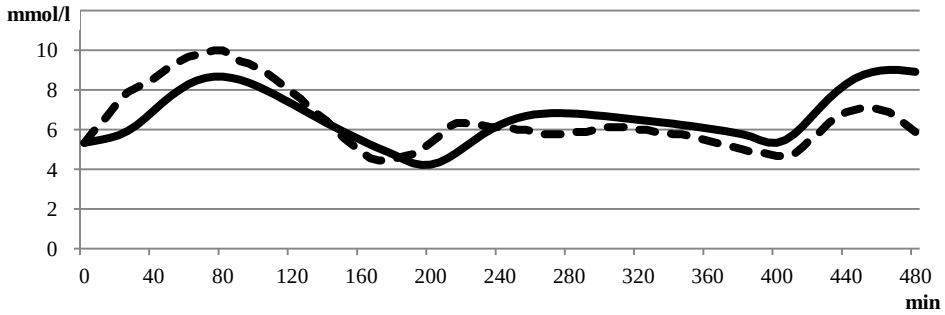

Figure 4. The first eight hours of the whole third day of patient B (solid line – model estimations, dashed line – measured values)

The results (see Table V and Table VI) prove that the whole day test gives better tracking as it takes more factors into account. The model copes with insulin absorption and digestion overlap which means around 5% improvement regarding the rate of deflection. The average deflection decreased with 0.45 mmol/l.

Comparing the two patients (see Figure 3 and Figure 4), there is more than 1 mmol/l decrease in average deflection if the experiment is properly logged. There is also more than 10% increase in the significant fields of rate of deflection (<3mmol/l).

TABLE V. COMPARISON BETWEEN THE TWO TESTS WITH PATIENT A

Patient A – 15 meals		**Meal wise**	**Whole day**
Average deflection		4,0 mmol/l	3,55 mmol/l
Rate of deflection	< 3 mmol/l	50 %	57 %
	< 5 mmol/l	68 %	79 %
	< 8 mmol/l	93 %	93 %

TABLE VI. COMPARISON BETWEEN THE TWO TESTS WITH PATIENT B

Patient B – 13 meals		**Meal wise**	**Whole day**
Average deflection		2,99 mmol/l	2,35 mmol/l
Rate of deflection	< 3 mmol/l	65 %	69 %
	< 5 mmol/l	76 %	81 %
	< 8 mmol/l	94 %	100 %

V. DISCUSSION OF RESULTS

The large differences between the prediction and the measured values that we experienced for patient A were most probably due to the poor quality of the dietary log. Also, it is harder to assess the performance of the model using only point-wise measurement data. For patient B, the error was fairly low (below 4 mmol/l) in the first hours (Fig. 4) and since we can re-start the model after the meals, the error calculated for the whole day in Table VI is over-pessimistic. However, the 4 mmol/l error is still fairly large, so relying solely on the model we could not exclude emergency situations (i.e. hypoglycemia) in a real life application. For better results, more efficient parameter training is needed.

The meal wise test with patient A also shows (see Figure 3) that using an ordinary blood sugar meter can lead to considerable errors in blood sugar level estimation. The patient measured a high value at 120 minutes, but the model shows even higher values between the two real-life measurements (0 min and 120 min). In this situation the gap is small, but there could be bigger differences as well. The frequent presence of these situations can lead to higher HbA_{1c} values.

Long-term model based predictions are in general less unreliable as the deviations accumulate. However, the proposed approach gives satisfactory results for short time prediction, which is the main demand to estimate the required amount of insulin in outpatient care.

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VI. CONCLUSION AND FUTURE WORK

The paper presented a combined model for the short time prediction of the blood glucose level, based on the dietary log of type I and type II diabetic patients. The results are satisfactory even without any model training.

Further research is needed for

- training the model to support personal variations in model parameters
- extending the model to use also other physiological data available like physical activity and stress.

Our aim is to decrease the average error under 1 mmol/l, which is a sufficient margin of error considering that the currently used real measurements have similar margin of error. The model is currently being further evaluated in a clinical study involving 20 rehabilitation patients, as an add-on module to the Lavinia lifestyle mirror [18].

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