

Personalized Glucose Prediction Model for Patients With Type I Diabetes

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Abstract. This paper represents an attempt to use machine learning techniques to personalize glucose predictions for patients with type I diabetes (T1D). The study aims at proposing a personalized model, capable to provide real-time blood glucose estimations, taking into consideration patient's health preconditions. The proposed model represents a neural network based on the use of Self-Organized Maps (SOM). It was elaborated using data from 5 patients with T1D, collected with help of a specially created for these purposes support system and pre-trained using a clinical dataset. The study lasted for 3 months.

Keywords: prediction model; diabetes self-management; neural networks; self-organized maps.

I. INTRODUCTION

Type I diabetes is a chronic autoimmune disease characterized by deregulation of glucose metabolism. This metabolic disorder is caused by the autoimmune destruction of insulin-producing beta cells of the pancreas resulting in the absence of insulin secretion. The lack of insulin provokes elevated blood glucose levels (hyperglycemia) leading to spillage of glucose into urine. The excess glucose circulating through the body in the blood stream over time, leads to damage of blood vessels (angiopathy), resulting in serious long-term complications, such as kidney failure, blindness, amputations and heart problems. According to the diabetes control and complications trial [1], the aforementioned complications can be reduced by intensive glycemic control, which involves regular glucose measurements and exogenous insulin administration [2].

First attempts of creating diabetes glucose models were done 60 years ago [3]. Most of modern models are predicting glucose levels based on carbohydrate and insulin dose inputs [4,5]. Unfortunately, these researches haven't created personalized models based on the obtained results. A recent research has demonstrated personalized models based on a virtual data simulator [6]. However, the research was only based on a virtual dataset. The aim of this study is to present an attempt to provide patients with a personalized glucose prediction model using Self-Organized Maps (SOM), based on their own health pre-conditions.

II. METHODS AND MATERIALS

A. A. Dataset

In this research is used a clinical dataset, obtained from Washington University, St. Louis, MO. The dataset is from AIM '94. Data set contains pre- and post-meal blood glucose measurements and insulin doses [7]. This clinical data was used to pre-train the Glucose Kinetics ANN module.

The patient-specific model was elaborated using data from 5 patients with DT1 using a specially created for this purposes

support system. The focus group consisted of 3 male and 2 female individuals, data was collected for a 3 month period. Description of the support system for data collecting can be found in Fig. 1.

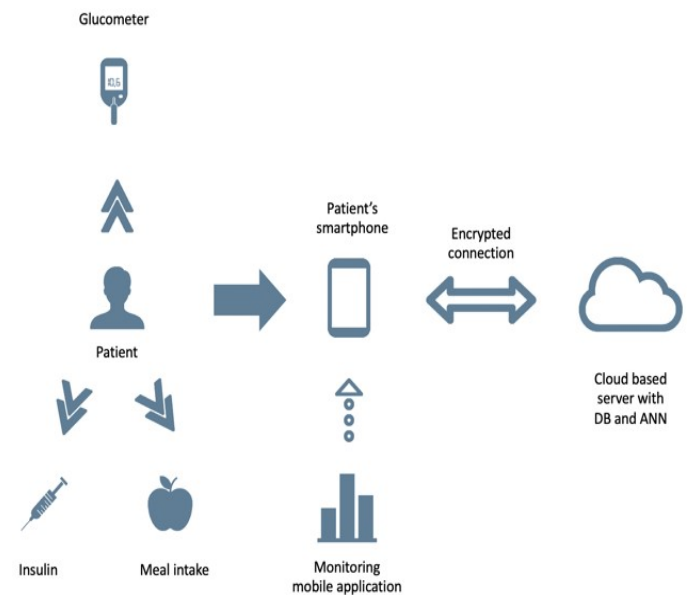


Figure 1. Support system for data collecting from patients

Support system consisted of:

- A monitoring mobile application to collect data on insulin doses, carbohydrates intake and blood glucose levels
- Cloud-based server with ANNs to process the data

B. B. Modelling Methodology

The proposed patient-specific model is a MISO (multi-input-single-output) model that takes as input parameters:

- Insulin (basal and bolus)
- Meal data (carb intake grams)

And returns a model, containing personalized glucose predictions.

It's based on a combination of 2 mathematical Compartmental Models (CM):

- Hovorka Glucose absorption from the Gut model [4]
- The Berger plasma insulin dynamics model [8]

And one neural network, that wraps glucose kinetics and returns glucose prediction. It's a multilayer perception neural network (MLP NN) that incorporates a Self-Organizing Map (SOM) to capture glucose metabolic behavior.

An overview of the modelling system can be found in Fig. 2.

In general, SOMs are mainly used for data clustering and visualization of high dimensional data. However, SOMs can also be trained to learn input-output mappings and used for function approximation. In this paper is used a SOM, adopted from [9]. The SOM consists of a two-dimensional grid of neurons. Every input vector is associated with a neuron in the grid which is called the winner neuron. Every neuron is associated with a weight vector which has the same dimensions as the input vector. During the training stage the weights of the neurons in the neighbor of the winner neuron are updated. The learning rate as well as the scope of the neighbor are decreased as the time periods go by. After the training stage, areas with similar input vectors are created and these vectors are represented by a neuron.

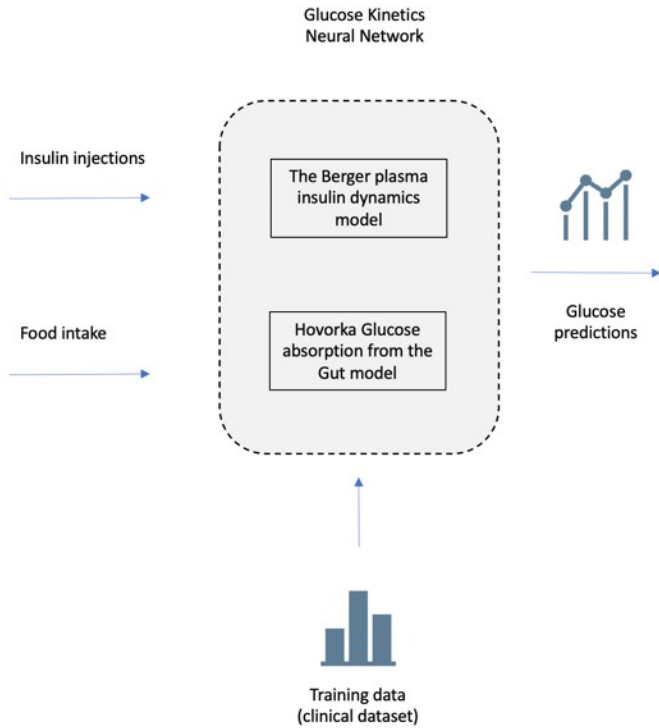


Figure 2. Outline of Glucose Kinetics Neural Network

The two-dimensional grid of N neurons is created and every neuron i is associated with a weight vector w_{in} and a weight value w_{out} . The input vector (v_{in}) has the form:

$$v_{in}(t) = [G(t - s_x + 1), \dots, G(t), I(t + 1), \Psi(t + 1)] \quad (1)$$

where G represents the glucose, I is the rate of appearance of insulin in plasma, Ψ represents the appearance rate of glucose in plasma and s is the number of steps that determine the x time window to be considered for the past glucose measurements. A value $v_{out}(t)$ corresponds to each input vector, which is the next glucose value:

$$v_{out}(t) = G(t + 1) \quad (2)$$

The vector w_{in} has the same dimensions as the input vector. During training, the winning neuron is determined by calculating the euclidean distance between the input vector and the weight vectors w_{in} of each neuron. The neuron with the lowest euclidean distance is the winner ($n_w(t)$):

$$n_w(t) = \arg \min \{ \|v_{in}(t) - w_{in}(t)\| \} \quad (3)$$

At every iteration the weights w_{in} and w_{out} are changed according to the rule:

$$\Delta w_{in}(t) = \theta(t) \cdot \varphi(n_w, i, t) \cdot [v_{in}(t) - w_{in}(t)] \quad (4)$$

$$\Delta w_{out}(t) = \theta(t) \cdot \varphi(n_w, i, t) \cdot [v_{out}(t) - w_{out}(t)] \quad (5)$$

where $\theta(t)$ is the learning rate in the time range from θ_0 to θ_r ,

$$\theta(t) = \theta_0 \cdot \left(\frac{\theta_r}{\theta_0} \right)^{\frac{t}{T}} \quad (6)$$

where T is the number of total time periods. Besides that, $\varphi(n_w, i, t)$ is the neighborhood function in a Gaussian form of:

$$\varphi(n_w, i, t) = e^{\left(\frac{-\|l_i(t) - l_{n_w}(t)\|^2}{2\theta(t)^2} \right)} \quad (7)$$

where $l_i(t)$ and $l_{n_w}(t)$ are the locations of neurons i and n_w respectively.

The reference group was proposed to introduce in the mobile application amount of the carbohydrate intakes, meal data included breakfast, lunch and other meals, taken till dinner. Each meal data reading was in the form of time and the amount of taken carbohydrates.

Collected insulin data consisted of basal insulin and bolus insulin doses together with the corresponding injection time. Measured data from patients was used in the following way: 60% for training, 40% for evaluation.

III. RESULTS

An example of correlation between resulting glucose predictions and glucose measurements are presented in Fig. 3.

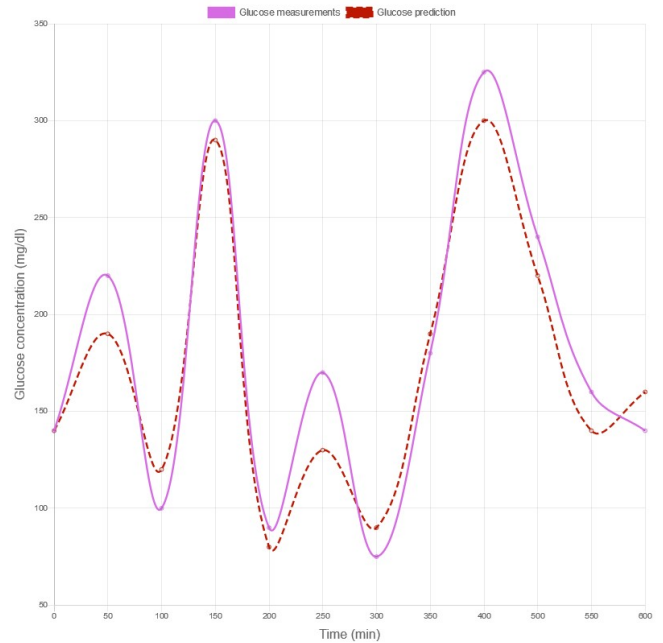


Figure 3. Representative example of glucose predictions (dashed line) and glucose measurements (solid line). 10 hours evaluation

The error grid analysis is presented in Fig. 4. The proposed model was evaluated considering time periods of 30 min and 60 min. The obtained results show best predictions

of normal glycemia values, 2.68% and 3.42% of erroneous readings respectively. A little bit worse results, especially for the 60 min time slot show hyperglycemia predictions, 9.29% of erroneous readings. Most erroneous readings are observed in the range of hypoglycemia, 15.52% and 24.78% respectively

	Hypoglycemia (%)		Normal Glycemia (%)		Hyperglycemia (%)	
	30 min	60 min	30 min	60 min	30 min	60 min
Accurate readings	84.48	75.22	97.32	96.58	92.86	90.71
Erroneous readings	15.52	24.78	2.68	3.42	3.14	9.29

Figure 4. Glucose predictions error grid

IV. CONCLUSIONS

This study represents an attempt to use machine learning techniques to personalize glucose predictions for patients with T1D. The resulting neural network module represents a combination of 2 Compartmental Models and a Self-Organized Map. It was pre-trained using a virtual dataset and evaluated for a 3-months period on the medical records, obtained from a group of 5 patients with T1D. The obtained results show the model’s ability to capture patients’ glucose kinetics and give personalized glucose level predictions.

Future work consists of enhancement and extension of the existing model by introducing more input parameters related to patients’ health conditions

Besides that, to get better results, this study requires a focus-group of more patients and a longer period of trial testing

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