Patient-specific Glucose Metabolism Models for Model Predictive Control of T1DM Glycemia

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2012

Link to publication

Citation for published version (APA):
### Background

In recent years, model predictive control (MPC) has been shown to outperform other control approaches to automatic glycemia regulation in diabetic subjects, becoming the most popular choice for the control algorithm [2]. In such a strategy, a model capable of representing the effect of insulin and carbohydrates on blood glucose evolution which is patient-specific, physiologically relevant, parsimonious, yet able to accurately forecast blood glucose is a crucial component affecting significantly the system performances.

### Objective

Estimate data-driven individualized glucose-insulin interaction models suitable for exploitation in a MPC framework.

### Data

8 T1DM subjects (5 M/3 F; age = 45.25±13.53 years, disease duration = 22.37±11.81 years, BMI = 23.88±3.25, Hba1c = 8.27±0.90%) underwent a 3-days visit at the CIC in Montpellier, France, within the European research project DIAdvisor™ [4]. Patients were served standardized meals for breakfast, lunch and dinner (carbohydrate content: 42, 70, 70 [g], respectively) and decided insulin needs based on their personal HemoCue™ Glucose Analyzer outcomes. Blood samples were collected by nurses to measure plasma glucose and plasma insulin concentrations: every hour during day, every 2 hours during night, 30 min before breakfast, 10, 20, 30, 60, 90, 120, 150, 180, 240, 300 min after breakfast and every 15 minutes after lunch and dinner for 2 hours, for a total of 37 blood samples per day.

### Results

All the estimated models were stable, the peak response of BG to 1 [IU] fast-acting insulin being 12.45±10.85 [mg/dL] while that to 10 [g] carbohydrates being 10.56±7.44 [mg/dL]. Cross validation via model-based predictions achieved prediction error standard deviation 14.19±8.45 [mg/dL] and 26.22±15.04 [mg/dL] on 30- and 60-minutes-ahead prediction, respectively.

### Discussion

Population mean values reported in the literature [1] were used for the parameters appearing in the meal model and in the insulin kinetics model, disregarding the inter-personal variability. Moreover, age of the subject, disease duration, BMI, insulin sensitivity, β-cells responsivity and probably many more unknown factors related to both the quantitative and qualitative responses to inputs were not considered. Last, the modeling process was possible due to the very rich and unique database.

### Conclusions

The investigation provided sound and valid individual-specific low-complexity models suitable for application in model-based blood glucose control.

### Acknowledgments

This research was supported by the European project DIAdvisor™ [4].

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**References**


