Chapter 1

Introduction

This chapter serves as introduction to determine why this study has been done, and what has previously been done in this field. The introduction consists of a brief overview of the study, the purpose of the study, and the scope of the study.

1.1 Introduction

The human energy system is a vast and complex part of the human physiology. Different approaches have been used to study this system, but due to its complexity it is usually broken down into smaller parts. The blood glucose (BG) subsystem has been researched extensively for several decades and was therefore chosen as the main focus of this study.

Several diseases have been linked to the BG subsystem, namely, diabetes, cardiovascular diseases, proliferative and nonproliferative (cancerous and noncancerous) diseases, and so forth. In the search of finding cures and treatment protocols for these diseases, biotechnology is the cutting-edge technology available. Simulation models describing the human physiology and pathogenesis of the diseases have been developed to help this cause. These models lower the risks and costs associated with clinical trials, as real patients do not have to be used. Time also plays a major role in clinical trials and if virtual patients are used, this obstacle is eliminated.

Several simulation models of the BG subsystem currently exist, with the most salient being Bergman's minimal model from 1979 (Bergman *et al.*, 1979). Many of the existing models are based on Bergman's minimal model with a few modifications to describe the system in more detail (Kovács *et al.*, 2010; Liu and Tang, 2008; Makroglou *et al.*, 2006).

However, none of these models uses an integrated approach to incorporate all the external and internal influences on the system. These influences include food intake, exercise, psychological stress, alcohol consumption and the regulation hormones (RH) and counterregulation hormones (CRH). In this study an integrated model is presented with the focus on diabetes. The simulation model will be for educational use by non-diabetic people as well as by people with diabetes, care providers, and medical professionals to learn what effect different influences will have on their BG and how to effectively manage their BG.

1.2 Purpose of the study

The purpose of this study is to research and describe existing educational simulation models of the BG subsystem that focus on diabetes. It will then be determined if any of these models incorporate all the external and internal influences on the system. A newly developed simulation model that incorporates all the external and internal influences on the BG

subsystem will then be presented. The new model will be compared to the existing models to determine if it represents an improvement. This simulation model will be used for educational purposes to educate non-diabetic people, diabetes patients and care providers. It must be experimentally validated to determine its educational value and also to obtain the public's opinion. The evaluation will be questionnaire-based and healthy, nondiabetic people will assess the model.

1.3 Scope of the study

In this study existing educational simulation models of the BG subsystem will be discussed and evaluated in terms of the influences on the subsystem these models take into consideration. A new simulation model will then be discussed and evaluated in a similar manner for comparison to the existing models. The validation of the mathematical model has been done in two previous papers (Mathews and Pelzer, 2009; Pelzer *et al.*, 2011) and will therefore not be discussed here. The new simulation model will then be experimentally validated to assess its educational value; user-friendliness and understanding of the simulation; and basic BG and diabetes knowledge transferred to the user. The evaluation was questionnaire-based. Healthy, nondiabetic people evaluated the model. Two different age groups – 13 years old (grade 7 learners) and 17 years old (grade 11 learners) – completed the evaluation. The results were statistically analysed.

1.4 References

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