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# A mathematical model for glucose and insulin dynamics with direct connection to the $\beta$-cell cycle

The term diabetes mellitus describes a group of metabolic diseases with persisting hyperglycemia as the main symptom. Interest is increasingly focused on the understanding and treatment of the disease because of its rising prevalence and the variety of severe complications. Recent experimental results indicate the relevance of the $\beta$-cell cycle for the development of diabetes mellitus.
We investigate the dynamics of the interplay of glucose, insulin and the $\beta$-cell cycle with a mathematical model of ordinary differential equations. The basis of the system is built by three different models. To analyze the dynamics of insulin the work of Grodsky [1] introducing a packet hypothesis for insulin storage has been modified. This has been connected with the dynamics of glucose (Topp et al. [2]) and a model for the $\beta$-cell cycle based on Daukste et al. [3]. The advantage of the system consists in its explicit incorporation of the $\beta$-cell cycle with insulin directly enhancing the replication rate of the cells.
In the presentation, the model and its development will be introduced as well as its capability of accounting for metabolic failures in the progression to diabetes.

## References

[1] Grodsky, G.M., *A threshold distribution hypothesis for packet storage of insulin and its mathematical modeling*, The Journal of Clinical Investigation **51** (1972), 2047-2059
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[3] Daukste, L., Basse, B., Bagueley, B.C., Wall, D.J.N., *Using a stem cell and progeny model to illustrate the relationship between cell cycle times of* in vivo *human tumour cell tissue populations,* in vitro *primary cultures and the cell lines derived from them*, Journal of Theoretical Biology (2009), 1-9